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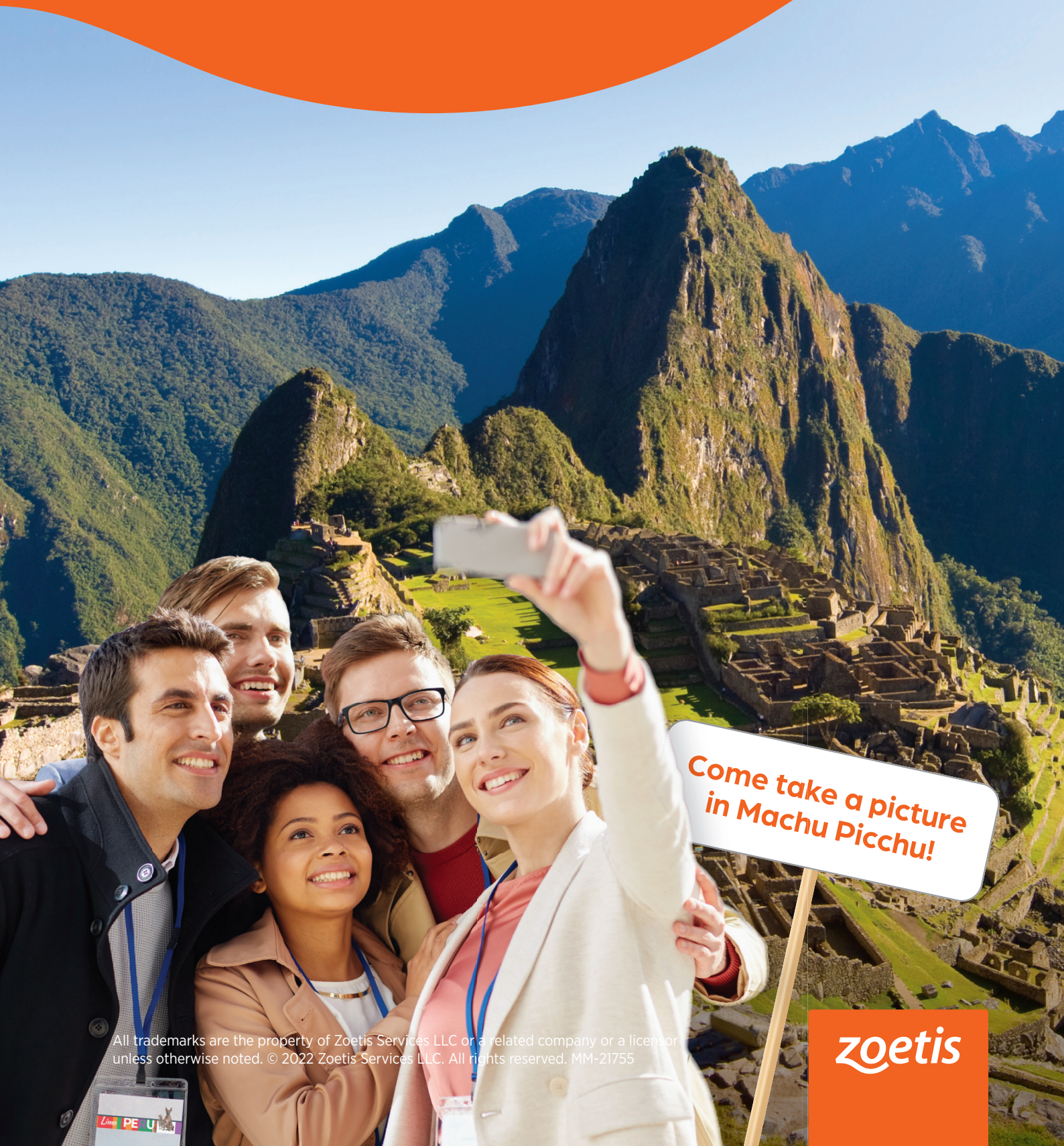

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OTITIS IN CATS: STEP BY STEP

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ABSTRACT BODY:

Otitis in cats

Otitis externa is a multifactorial and aetiologically complex disorder of the ear canal in domestic animals, including the cat. Diagnosis is usually by means of direct otoscopic examination and cytology of otic exudates. Studies report prevalence in pet cats to be between 2% and 10%.

Except for the Scottish Fold, domestic cats have upright pinnae, allowing for air circulation. This makes them less vulnerable to environmental factors that predispose to otitis, such as humidity and the effects of bathing or swimming. Despite the conformational difference in the Scottish Fold, an increase in otitis has not been reported in this breed.

Feline ear have a moderate amount of dark brown ceruminous debris present in the canal, this is more common in breed as Persian or Himalayas. If no other clinical signs are present, this is not suggestive of otitis externa. Cerumen may also be clear, white or yellow.

Otodectes cynotis (ear mite) is a psoroptid mite that accounts for 50–80% of cases of otitis externa in cats. It is highly contagious via direct contact with infested animals. *Otodectes cynotis* can also infest hosts including dogs, foxes, ferrets and occasionally humans. Chronic otitis and ceruminous gland hyperplasia secondary to ear mite infestation can be predisposing factors for the development of ceruminous gland tumours in dogs and cats.

Feline ear cytology can be challenging to interpret because cat´s cerumen can absorb stain, resulting in a stain artifact resembling bacterial cocci. Evaluating for pairs of cocci may help avoid misinterpretation.

Malassezia species, most commonly *Malassezia pachydermatis*, can be found on aural cytology and culture in normal cats. The prevalence of *Malassezia* species on otic cytology reported in the literature ranges from 23% to 83%. In normal cats, the median density of *Malassezia* species on cytology ranges from 0.2 to 0.5 organisms per HPF.

Rod-shaped bacteria are rarely seen in healthy cats, so their presence suggests otic pathology. In one study, *Bacillus* species and *Pantoea* species accounted for 17% and 4% of bacterial isolates from the ears of feral cats, respectively.

Keratinocytes and nucleated keratinocytes are a normal finding in feline otic cytology and were not increased in a group of cats with otitis externa. Low numbers of melanin clumps and saprophytes can also be seen in normal cats.However, inflammatory cells are not a normal finding in the feline ear, and their presence suggests disease.

Among cats diagnosed with allergies at a veterinary teaching hospital, 17% had bacterial otitis externa, and 6% had *Malassezia* otitis externa. Otitis externa is most common in cats with feline atopic skin syndrome.

Allergic skin disease should be considered in cats where infection recurs after cytologic cure and where other causes have been excluded. Cats with otitis externa owing to allergies are likely to experience pruritus in other body regions, rather than having only otic disease. Control of otitis externa in a patient with allergic skin disease requires the underlying allergy to be addressed.

Feline middle ear is divided in to two chambers (dorsolateral and larger ventrolateral chamber) by a thin bony plate. There is only minimal communication between the two chambers (there is a small hole at the caudal end of the bony plate which allows for communication). The epithelial lining of the middle ear contains more goblet cells than in the dog (fluid produced is more mucoid). The eustachian tube, oval and round windows are as for the dog. Sympatetic nerves course a bit more ventrally on the medial wall of the tympanic cavity than in the dog (more susceptible to trauma). The facial nerve passes through the middle ear in a bony channel (less likely to be affected by the presence of an otitis media, as compared to the dog).

Inflammation within the middle ear may be infectious (bacterial, fungal) or noninfectious (foreign body, neoplasia, polyps, trauma, cholesteatoma).

Otitis media is most commonly an extension of an otitis externa, through a perforated tympanum. Extension of infection up the Eustachian tube does occur but is rare in the dog. It is more common in the cat, likely because cats have a higher incidence of posterior pharyngeal inflammatory disease due to viral infections. With ascending infections, the tympanum is usually intact. In the middle ear of both dogs and cats, mucoid secretions are normally produced that drain through the Eustachian tube into the posterior pharynx. The middle ear of both dogs and cats have a normal bacterial flora. Obstruction of the Eustachian tube

due to posterior pharyngeal inflammatory disease or neoplasia may result in the accumulation of secretions within the middle ear. Otherwise normal microflora of the middle ear may then produce infection. These fluid accumulations may also be sterile. This pathogenesis for otitis media is rare.

It is not uncommon to see concurrent otitis interna along with otitis media in both the dog and cat. In these instances, inflammation/infection accesses the inner ear through the round and oval windows (covered by only by a thin layer of epithelium).

Polyps are most noted in young cats (variably reported as 18 months or 6.0 years; range of 2.5 months to 18 years). Although most are unilateral, polyps can be bilateral. Most polyps appear to grow from the epithelial lining of the epitympanic cavity/tympanic cavity, then extend either through the tympanum into the horizontal canal or down through the auditory canal in to the posterior pharynx. Although uncommon, it is possible to have polyps grow in both directions (about 10% of cases). Histologically, polyps consist of a core of loosely arranged fibrovascular tissue with variable numbers and types of inflammatory cells throughout (lymphoplasmacytic; lymphoid aggregates/follicles; pleocellular inflammation). This core is covered by a stratified squamous to ciliated columnar epithelial layer. The epithelial layer thickens with chronicity, producing almost a capsular effect.

Clinical signs associated with aural polyps include signs of otitis externa (head shaking, scratching ears, otic exudate), and/or the development of neurologic signs (Horner's syndrome, head tilt, nystagmus, ataxia). Growth into the posterior pharynx is suggested by nasal discharge, sneezing, stertorous respiration, dyspnea, and dysphagia.

The etiology of polyps remains unknown. Attempts have failed to document the consistent presence of viruses in polyp tissue.

AURAL POLYPS:

Current therapies of choice include removal by traction/avulsion or removal by ventral bulla osteotomy. Traction alone results in an overall cure in about 60% of cases. A significant reduction in the incidence of recurrence following traction has been noted in cats treated with oral prednisolone following traction removal. The authors therapy of choice for aural polyps is to remove as much of the polyp as possible from the ear via traction/avulsion. Deeper tissue removal (to just inside the opening to the middle ear) is facilitated by utilizing biopsy forceps directed through a video otoscope. Cytologic examination dictates the choice of systemic and topical antibiotic/anti-fungal therapy.

Post avulsion, the ear is flushed with dilute vinegar and water (1:2 to 1:3), once every other day and treated with a "safer" topical otic preparation (see above in these Proceeding), Post avulsion, the cat is treated with oral prednisolone, beginning at 2-3 mg/kg/day for 2 weeks, then 1 – 1.5 mg/kg/day for 2 weeks, then 0.5 – 0.75mg/kg/day for 2 weeks, then 0.5-0.75 mg/kg once every other day for 2 weeks (6-8 weeks of therapy). This therapeutic attack has resulted in regression of remaining inflammatory tissue within the middle ear and healing of the tympanum (as documented by video otoscopy). It is rare to see recurrences of aural polyps using this protocol. Refractory/recurrent cases are managed by ventral bulla osteotomy.

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BREEDING MANAGEMENT AND BREEDING HEALTH

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ABSTRACT BODY:

Dogs are non-seasonal breeders and ovulate spontaneously.

The onset of puberty in the bitch corresponds with the first oestrus and will occur anytime between 3.5 and 24 months (average: 10-12 months). In general, small breeds experience their first oestrus earlier and large breeds later. For some breeds it can be physiologic to not show heat up to the age of 24 or even 30 months of age, e.g. Afghan hound, Greyhound, Whippet, Saluki.

Interoestrous Interval (IEI)

On average the IEI in bitches is 7 months (normal range 5-12 months). Only in the Basenji is the IEI 1 year. The mean IEI does not (or very little) differ between pregnant vs. non-pregnant cycles.

Although new research data challenges this, it has been widely accepted that a minimum of 4 months from the onset of prooestrus is necessary to allow for remodeling of the uterus. Bitches may show highly consistent or quite variable IEI. Dogs housed together or litter mates may show similar IEI.

Tools to determine stage of oestrous cycle

Test for "breeding reflexes":

Stimulation of the perineal area will result in **"flagging"** (lateral deviation of the tail) and **"winking"** (upward tipping of the vulva). Lumbar pressure will result in **"lordosis"** (standing firmly and arching her back); note vaginal discharge

VAGINOSCOPY:

Vaginoscopy is best performed using a vaginal speculum made from plexiglass and a strong light source. Some KY jelly on the speculum can be used to ease vaginal insertion. Along with being useful for monitoring the oestrous cycle, it is also essential for diagnosing several genital disorders.

During oestrus monitoring, the vaginal wall and its mucosal folds (plicae) are assessed.

CYTOLOGY:

Exfoliative vaginal cytology is a valuable clinical method to monitor the oestrous cycle as changes in cytology closely follow endocrinological events.

The major cell types present are:

- non cornified cells
  - parabasal cells: small, round – oval, "fried egg" appearance
  - intermediate cells: larger than parabasal cells, higher cytoplasm : nuclear ratio
- cornified cells
  - superficial cells: pyknotic nuclei, angular shape to the cytoplasm
  - anuclear squamous cells: anuclear, largest of vaginal epithelial cells, resemble "cornflakes"

Cells that can be present: erythrocytes, bacteria (especially early in prooestrus) **BUT inflammatory cells (especially bacteria phagocytosing cells) should not be seen in oestrus (-> warrants further work up).**

SERUM HORMONE LEVELS:

Determine serum concentrations for progesterone and/or luteinizing hormone (LH); LH test is expensive and peak is easy to miss (has to be performed daily), therefore, progesterone is measured to indirectly determine the time of the LH surge, serum progesterone only needs to be measured every other day.

BEWARE! Whole blood should not be chilled prior to centrifugation. Ideally, keep it at room temperature and centrifuge as soon as possible.

Serum can be stored at – 20 degrees Celsius.

PLEASE NOTE:

Lower case "d" refers to LH surge, upper case "D" refers to dioestrus

(therefore, d 0 = D -8), e.g.

d 0: day of the LH surge (based on serum progesterone levels)

d 1-7: days of oestrus in relation to LH surge

D 1: first day of dioestrus (based on cytology)

Breeding Management

Prior to breeding a general health test should be performed. Screening for certain genetic diseases should be recommended in some breeds (e.g. for hip/elbow dysplasia) and breeding should be strongly discouraged in affected bitches.

Vulvar conformation should be evaluated in all bitches. Although some conditions may improve under hormonal influence, others may prevent a normal breeding or whelping. A digital vaginal exam (in middle to large breed bitches) may reveal vaginal tissue bands,

tumors, and foreign bodies.

A vaginoscopy should be performed if the bitch has a history of being non-receptive during oestrus or is showing signs suggestive of an inflammation of the reproductive tract (vulvar discharge, excessive licking, scooting, abnormal odor etc.).

If available, a rigid endoscope can be used to evaluate the vagina even in small dogs.

Serology: in most countries all breeding animals (male and female) are tested for the presence of *Brucella canis* (*B. canis*) antibodies.

Titers for canine herpesvirus can be performed. Negative titers require isolation of the bitch from potentially positive dogs especially in the last 3 weeks of pregnancy and the first 3 weeks post whelping.

Culture: Although vaginal cultures are often requested by breeders, they are rarely indicated in the "normal" bitch. Bacteria can be found in almost any bitch at any stage of the cycle in the vagina.

Vaginal cytology seems to be a much better way of assessing genital infection.. Leukocytes in an oestrus smear are abnormal and antimicrobial treatment should be initiated based on culture and sensitivity results. In most cases, the breeding management will progress as planned as the embryos will only enter the uterus at the beginning of dioestrus leaving ample time to treat the infection.

How to manage the breeding?

At the first sign of prooestrus (vulvar swelling/ bleeding), take the first vaginal smear for cytology and a serum sample for progesterone determination.

Keep doing this every other day until the serum progesterone indicates it is the time of the LH surge (doubling of serum progesterone level, most commonly ~ 1.5 – 2.5 ng/ml). Stop cytology at the first day of dioestrus (D1).

When to breed depends on the method of breeding:

NATURAL MATING:

let the dogs breed every other day from d 3 of oestrus until the first day of dioestrus

(D1). Alternatively, let them breed on d 3 and 5 or d 4 and 6, if the stud dog is in high demand or other restraints limit the frequency.

Dog semen can survive up to 10 days (average 6 days) in the female reproductive tract. Therefore, a breeding between two days before the LH surge (d -2) until d 6 of oestrus can result in high conception rates. Multiple males can sire the same litter (superfecundation)!

Artificial insemination:

- fresh and chilled semen: inseminate either on day 3 and 5 or day 4 and 6.
- frozen semen: depending on how many doses are available;
  - if only one dose: d 5
  - if two doses: d 4 and 5 or on d 5 and 6.

Frozen semen has a very limited life span (only up to 24 hours). Therefore, it should be deposited into the uterus the closest to day 5.

d 0: LH surge
d 2-3: ovulation
d 4-6: oocytes mature and ready to fertilize

Oocytes remain viable for 2 to 3 days.That means that on d 6 some oocytes can begin to show signs of degeneration. An additional serum sample for progesterone measurement should be taken at the time of D 1, in order to determine that the progesterone has continued to rise and therefore, ovulation has occurred. Determining the LH surge and D 1 of dioestrus are helpful tools to determine the day of whelping.

Whelping will occur:
57 ± 1 day post D 1 of dioestrus
65 ± 1 day post LH surge (d0)

	Prooestrus	Oestrus
Test for "breeding reflexes"	Reflexes reduced in the beginning, progressively increasing Discharge: light-coloured serosanguineous in the beginning, darker in the end	pronounced flagging, lordosis, "standing for the male", reduced vaginal discharge
Vaginoscopy	Vaginal folds swollen, moist, hyperaemic, gradually starting to shrink	folds becoming increasingly shrunken/ angular (crenulated), pale in color, dry
Cytology	keratinized superficial cells (SC) increasing during pro-oestrus, > 90% marks beginning of oestrus, numerous red blood cells	keratinized SC index stays > 90% through oestrus, drops to < 50% at beginning of dioestrus
Blood tests:	LH: only increases for short period of time at the beginning of oestrus Progesterone: < 0.2 ng/ml in anoestrus and early in oestrus, slowly increases to ~ 1 ng/ml during oestrus and rises sharply to ~2 ng/ml at the time of the LH surge	LH surge marks the beginning of oestrus. Progesterone is around 2 ng/ml at time of LH surge and can be measured to infer LH surge; after that it increases less reliably (4-10 ng/ml at time of ovulation)

003 / #25

REVISANDO A LOS CLÁSICOS: OBSTRUCCIÓN URETRAL FELINA.

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ABSTRACT BODY:

OBSTRUCCION URETRAL FELINA: REVISITANDO A LOS CLÁSICOS

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La obstrucción uretral felina (OU) es una patología frecuente en gatos machos, dolorosa y sujeta a complicaciones que pueden poner en riesgo la vida del paciente si no se interviene rápidamente. Afecta mayoritariamente a gatos machos que sufren enfermedad del tracto urinario felino (FLUTD en inglés).

Según previos estudios, la causa más frecuente de FLUTD es la cistitis idiopática felina (CIF) (55-69% de los casos) seguida por urolitiasis (12-22%). Infecciones urinarias bacterianas representan el 1.5-20% de los casos, habiendo diferencias en la incidencia según sexo (más frecuentes en hembras) y edad (muy rara en machos jóvenes). Causas menos frecuentes de FLUTD serian neoplasia (0.3-3.6%) o neuropatías (0.2-3%).

Aunque la patofisiologia de la CIF no está del todo clara, se cree que es debida a complejas interconexiones entre la vejiga de la orina, el sistema neuroendocrino y factores ambientales (el estrés puede desencadenar los episodios). Algunos autores definen la CIF como una “ansiopatía”.

El 15-67 % de los gatos machos con CIF y el 20-66 % de los que presentan urolitiasis pueden desarrollar obstrucción uretral (OU) (tapones uretrales, cristaluria, cálculos). Los desequilibrios electrolíticos asociados a la obstrucción pueden comprometer seriamente la función cardiaca (arritmias) y la obstrucción produce un aumento de presión en la pelvis renal que compromete la perfusión renal, reduce la tasa de filtración glomerular y puede conducir al fallo renal agudo.

Estos pacientes deben ser tratados inmediatamente con analgésicos, liberando la obstrucción uretral y administrando fluidoterapia intravenosa para corregir el trastorno electrolítico. El pronóstico a corto plazo es bueno si la obstrucción se resuelve tan pronto como sea posible, con una supervivencia de 91-95%. No obstante, si la obstrucción no se trata puede ser letal en 3-6 días. En el caso de los cálculos vesicales, la cirugía o disolución médica (solo si son de estruvita) deben ser recomendadas.

El pronóstico a largo plazo es algo peor, con ratios de recurrencia publicados en torno al 15-36%, generalmente durante la primera semana. Suelen recidivar los gatos de mayor edad y que ingieren menos agua.

A pesar de estas estadísticas, el coste del tratamiento standard con hospitalización es considerable y algunos propietarios pueden tener que optar por la eutanasia. No obstante, algunos gatos que se presentan mas estables pueden mejorar tras la desobstrucción sin ser hospitalizados. Esta opción debe ofrecerse siempre antes de eutanasiar.

Desde el punto de vista del veterinario de emergencias, los tratamientos necesarios en estos pacientes son analgesia, desobstrucción y fluidoterapia (y corrección de la hiperkalemia si está presente). La evidencia de otros tratamientos comúnmente empleados es controvertida y se requieren de más estudios para entender mejor la patofisiología del proceso y la evidencia de algunos de los tratamientos empleados.

Aunque se ha descrito y demostrado que la cistocentesis descompresiva es un procedimiento seguro cuando se realiza bajo sedación, un estudio reciente no demostró ninguna ventaja en la reducción del tiempo de sondaje o en la normalización de las anormalidades electrolíticas o en acortar el tiempo de hospitalización.

No está claro por cuanto tiempo debemos mantener la sonda uretral, aunque es razonable mantenerla hasta resolver hematuria y azotemia (aproximadamente 24-48h). Parece que el uso de catéteres más finos (3.5 F) es mejor tolerado y asociado a menos complicaciones.

Uno de los tratamientos más comúnmente usados es prazosin con el objetivo de controlar espasmo uretral. La evidencia de este tratamiento no está del todo clara (el espasmo uretral no está bien definido en gatos y la medicación solo afecta a la musculatura lisa uretral presente en la parte más craneal de la uretra, que no es donde se presentan la mayoría de las obstrucciones).

Un estudio retrospectivo de pequeño tamaño en 2013 demostró una menor tasa de recidivas en los pacientes tratados con prazosin en comparación a los tratados con fenoxibenzamida o sin tratamiento. En 2017 se publicó un estudio prospectivo en el que no

se encontraron diferencias significativas entre el grupo tratado con prazosin (0.25 mg/gato bid) y el placebo en la tasa de recidivas. Un estudio reciente (2022) demuestra que su uso puede incluso predisponer a recidivas. Los autores identifican limitaciones en el estudio (ser retrospectivo y tener menos casos en el grupo de animales no tratados) pero aun así concluyen no recomendando el uso de prazosin.

La práctica de lavar la vejiga con suero fisiológico en casos de severa hematuria también ha demostrado no tener impacto clínico en la prevención de recidivas, aunque en dicho estudio el protocolo de tratamiento no estaba estandarizado y el número de casos era insuficiente para demostrar diferencias estadísticas significativas.

El uso de otras terapias como antiinflamatorios (esteroideos y no esteroideos), antibióticos, infusiones intravesicales de glucosamina/pentosan no han probado ser de utilidad en reducir las recurrencias y en el caso de los antibióticos, solo deberían usarse si se demuestra infección bacteriana.

El aumento del agua ingerida demostró reducir la tasa de recidivas a largo plazo. El uso de dietas urinarias húmedas también es de ayuda en el manejo de estos pacientes, ya sea por su composición o por el hecho de aumentar la proporción de agua en la dieta.

La uretrostomia perineal puede ser una alternativa en pacientes que recidivan constantemente pero no debe ser la opcion inicial de tratamiento ya que no está exenta de complicaciones y puede no ser de ayuda en todos los casos.

Tenemos claro cómo tratar estos pacientes en la sala de emergencias cuando llegan obstruidos, pero son necesarios más estudios prospectivos para acabar de entender por completo esta patología (CIF) y la forma de tratamiento óptimo a largo plazo para reducir la tasa de recidivas.

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004 / #26

COMO DIFERENCIAR EPISODIOS SIMILARES A ATAQUES EPILÉPTICOS

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ABSTRACT BODY:

El primer paso frente a un paciente que se presenta por sospecha de ataques epilépticos, es intentar confirmar que los episodios que sufre el animal realmente lo son. Esto puede resultar un reto para el veterinario ya que, por un lado, un ataque epiléptico puede manifestarse de formas muy diversas y, por otro lado, hay episodios que clínicamente pueden parecerlo y no lo son.

La confirmación de que un evento episódico corresponde a un ataque epiléptico es mediante electroencefalografía. Sin embargo, no suele ser una técnica disponible para el clínico. Por tanto, a la hora de valorar estos episodios, será imprescindible lo siguiente: 1) fijarse en diferentes características observables típicas de un ataque; 2) conocer la clasificación semiológica actual (tipos de ataques descritos según su manifestación clínica) y 3) reconocer episodios que no lo son, aunque clínicamente puedan parecerlo.

Características generales observables de un ataque epiléptico:

- Inicio agudo
- Sin factor desencadenante
- Duración corta (segundos- minutos)
- Autolimitante
- No interrumpible
- Alteración de la conciencia
- Presencia de fase pre y/o post ictal
- Presencia de signos autonómicos

Clasificación semiológica actual

- Ataques epilépticos generalizados
- Tónico-clónicos
- Tónico
- Clónico
- Mioclónico
- Atónico
- Ataques epilépticos focales
- Motores
- Autonómicos
- Comportamentales
- *simples o complejos según si hay o no alteración de la conciencia.

Eventos paroxísticos que pueden parecer ataques epilépticos y no lo son:

Como se ha mencionado previamente, existen muchos eventos paroxísticos que no son ataques epilépticos, pero es fácil confundirlos como tal debido a su manifestación clínica similar. Es vital reconocerlos, ya que tanto el protocolo diagnóstico como el tratamiento serán diferentes. En estos pacientes, es imprescindible una exhaustiva anamnesis. Si desafortunadamente no vemos el episodio, solo dispondremos de la información que nos dará el propietario; por tanto, cualquier dato es de mucha utilidad, y deben intentar ser lo más objetivos posibles. Es muy útil la grabación de estos episodios.

Algunos ejemplos de estos episodios son los siguientes. Se describen solamente las características más significativas de cada uno de ellos:

- Sincope: Consiste en un colapso agudo, flácido y de corta duración con pérdida de la conciencia, secundario a un problema cardiaco. La duración es de pocos segundos y ocurre después o asociado al ejercicio, excitación o estrés.
- Movimientos involuntarios de la cabeza (*head bobbing*): Consisten en movimientos paroxísticos rítmicos de la cabeza en un animal consciente. Estos movimientos pueden ser verticales o horizontales, y con una frecuencia y duración muy variable (desde uno esporádico a 20 por día). Se pueden interrumpir fácilmente. Ni está clara la fisiopatología, pero se sospecha que es un tipo de desorden del movimiento asociado a distonía. Es más frecuente en dóberman, bulldog y Bóxer jóvenes.
- Movimientos involuntarios- Discinesias: Son un grupo heterogéneo de síndromes clínicos caracterizados por episodios episódicos y recurrentes de movimientos involuntarios que afectan a una o varias extremidades. El animal está consiente, pero le alteran la postura y la march. Tienen una frecuencia muy variable, y que pueden durar hasta 90 minutos. Son autolimitantes. En ocasiones están inducidos por estrés. La mayoría de ellos tienen unas características específicas de raza.
- Mioclonías no epilépticas: Son movimientos rítmicos e involuntarios de un grupo de músculos que producen el movimiento de la parte afectada. El animal está totalmente consciente. Pueden ser

episódicos o constantes, y pueden persistir durante el sueño. Son secundarios a cambios patológicos en los núcleos de NMI, secundarios a una infección previa de moquillo.

- Temblores generalizados. Consisten en movimientos involuntarios de baja amplitud, pero alta frecuencia, generalmente continuos, y que empeoran con la excitación. El animal está consciente. Puede estar caudado por diferentes etiologías como tóxicos, problemas cerebelares o hipomielinización central en cachorros.
- Narcolepsia: Es un síndrome caracterizado por hipersomnolencia y episodios de cataplejía. Se trata de una alteración neurológica que afecta a los mecanismos reguladores del ciclo vigilia- sueño. Se ha descrito en diferentes razas de perros, y puede tener origen congénito (dóberman, labrador y teckel) o adquirido. Clínicamente se ve como un colapso súbito, flácido (perdida del tono muscular) de diferente intensidad y frecuencia, asociado a un factor desencadenante, que suele ser la comida. Normalmente de corta duración.
- Trastornos de la fase REM del sueño: Son cuadros clínicos que consisten en los movimientos y las conductas repetitivas fisiológicas durante la fase REM del sueño, pero que por su intensidad o frecuencia se consideran patológicas.
- Alteraciones del comportamiento: Pueden manifestarse como torneo, persecución de la cola, lamidos compulsivos,… secundarios a alteraciones de la conducta. Pueden ser constantes o episódicos, de duración variable, y desencadenado por situaciones de ansiedad o frustración. En general, se pueden interrumpir.

STEP-BY-STEP LOCAL ANESTHETIC BLOCKS FOR ANY PRACTICE

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ABSTRACT BODY:

Local anesthetics provide profound pain relief, which has a major medical impact on the patient's health and healing. The use of local/regional blocks improves anesthetic safety by allowing the patient to be maintained at a lower dose of inhalant, thus decreasing inhalant-mediated cardiovascular and respiratory depression. Local anesthetic blockade decreases pain, both intra- and post-operatively. In humans, local blocks have been shown to decrease the likelihood of chronic pain development from the acute pain source (eg, incisional pain). The use of local anesthetics decreases the need to administer drugs like opioids that can cause vomiting.

A few of my favorite blocks: Step by Step

For all of the blocks listed below, the dosages are: bupivacaine or ropivacaine (2-4 mg/kg dog, 1-2 mg/kg cat) OR lidocaine (4-6 mg/kg dog, 2-4 mg/kg cat). Many of the locations create a volume limitation (eg, most oral blocks) and in this instance, the approximate volume of injection will be listed. However, the full dose of local anesthetic should be calculated so that it is not exceeded if other blocks are added. A comment will be added for blocks in which NOCITA is appropriate. The label dose for the dog (5.3 mg/kg) is generally adequate in both cats (label dose 10.6 mg/kg) and dogs. As with the other drugs, the dose is volume limited for many blocks.

Intraperitoneal Lavage for ovariohysterectomy (OHE), Abdominal Surgeries, and Cesarean Sections

- Draw up full dose of bupivacaine, ropivacaine or lidocaine.
  - This dose is IN ADDITION to the small volume of local anesthetic used for an incisional block AND the very small volume of lidocaine used for arytenoid desensitization prior to intubation in cats. The dosages studied are higher than the dosages recommended here so there is obviously a robust safety margin.
  - **If necessary**, dilute the drug with saline – the total volume needs to be 0.4-0.6 ml/kg to 'lavage' or 'bathe' the entire abdominal cavity.

This is generally not necessary with standard local anesthetic concentrations.

- After opening the linea (preferred for efficacy) OR after completing the OHE (usually more practical so that the drug isn't absorbed by sponges used during the procedure), insert the syringe (no needle!) through the incision into the abdomen and 'bathe' the peritoneal cavity with the mixture by instilling – literally squirting - it right into the abdomen. Complete the OHE and close the incision as usual.
  - Publications are available in both dogs and cats for the efficacy of this block for OHE. In humans, the block is also used for abdominal exploratory surgeries and cesarean sections. These uses are also common in veterinary medicine, but there are no research studies. Excellent resource: Steagall PVM, Benito J, Monteiro B, et al. Intraperitoneal and incisional analgesia in small animals: simple, cost-effective techniques. J Small Ani Pract 2020;61(1): 19-23.

Testicular Block

This block should be done in both dogs AND CATS

- Draw up full dose of lidocaine, bupivacaine or ropivacaine.
- NOTE on choice: Lidocaine has a shorter duration of action but is the only local anesthetic that can be administered IV and this is a highly vascular site so some people feel more comfortable with lidocaine.
- The low end of the dose is generally adequate since the volume will be somewhat limited by space in the tissue.
- Clip or pluck hair (if necessary - NOT REQUIRED) and do an initial GENTLE scrub of surgical site (the goal is to avoid causing scrotal irritation).
- Insert the needle fully into the body of the testicle, usually with the needle tip pointing towards the body of the spermatic cord, but this isn't critical.
- ASPIRATE
- Inject ½ of the drug into the body of each testicle until you feel 'pressure' or until the calculated dose has been injected. The volume that can be injected into the testicle is generally about 0.2-2.0 ml per testicle depending on patient size.

The drug migrates up the spermatic cord so the area of crushing (PAINFUL!) is blocked.

- For an incision directly over the testicle (cats), continue infiltrating as the needle exits the testicular body to block the skin and subcutaneous tissue. For

an incision in another location (dogs), inject local anesthetic in skin and subcutaneous tissue at the incision site.

This small amount of drug is in addition to the amount calculated for the testicles.

Sacrococcygeal Block for un-obstructing blocked cats, any other perineal surgery, anal gland removal, tail amputation, assisted delivery of pups/kittens, etc…). NOT JUST FOR CATS!!!

- Draw up 0.1-0.2 ml/kg lidocaine, bupivacaine or ropivacaine.
  - Bupivacaine and ropivacaine have longer duration of action.
  - 0.1 mg/kg morphine will prolong duration. Draw up the 0.1 mg/kg then use the local anesthetic to obtain a total volume of 0.2 ml/kg (Pratt et al. JVECC 2020;30(2):170-178). In this study, cats required rescue analgesia at an average of 8 hours (bupivacaine alone), 10 hours (bupivacaine-morphine) or 2 hours (no epidural).
- Place the cat in sternal recumbency with the rear-legs stretched out caudally.
- Locate the sacrococcygeal space by palpating at the base of the tail while 'pumping' the tail up and down. The sacrococcygeal space can be palpated between the rigid sacrum and the movable coccygeal vertebrae.
- Shave the hair and scrub this space.
- Place a finger on midline just in front of or behind the sacrococcygeal space. This will serve as your guide as you place the needle.
- Insert a 25-G 1-inch needle through the skin at about a 30-degree angle off vertical and right over the sacrococcygeal space.
- If the needle encounters bone, back out slightly and redirect either a few degrees steeper (closer to perpendicular) or a few degrees flatter (towards a 45-degree angle from vertical).
  - Make small moves, you are using the needle as a probe to gently find the space.
- When in the correct spot, the needle will advance deeper with no resistance. A 'pop' might be felt as the needle passes through the ligamentum flavum and into the space.
- Aspirate. Inject the drug(s) over roughly 5-10 seconds. Slow injection allows a more even spread.
- Withdraw the needle and observe for tail and anus relaxation. The tail and anus can be lightly pinched while observing for a response from the patient. The penis will be easy to extrude.
- If no relaxation in 5 minutes, repeat the process ensuring that the needle actually slides off the bone into a space – a subcutaneous injection will not be effective. In a large study, time to do the block was an average of 2 minutes, but two attempts were not uncommon (Pratt et al. JVECC 2020;30(2):170-178). So try again!
- More details: Coccygeal epidural with local anesthetic for catheterization and painmanagement in the treatment of feline urethral obstruction. O'Hearn M, Wright B. JVECC 2011;21(1) 2011:50–52.

Caudal Maxillary Block to desensitize the caudal molars for dental/surgical procedures

The caudal maxillary approach is often preferred over the infraorbital approach because the field of desensitization is much larger. This block will desensitize all ipsilateral tissue from the caudal molars rostrally and from the skin to midline. Use this approach if working on the caudal molars or doing surgeries on the nares, nasal passages, sinuses, soft palate or any other structures of the maxilla. In cats and brachycephalic dogs, the distance from the infraorbital foramen to the pterygopalatine fossa (where the maxillary nerve and its branches enter the skull) is very short and caudal diffusion of drug injected at the infraorbital foramen may be adequate for blocking the caudal structures of the skull.

- **Extraoral approach 1** (extraoral, from zygomatic arch): Insert the needle percutaneously along the ventral border of the zygomatic arch approximately 0.5 cm caudal to the lateral canthus of the eye. The needle is kept horizontal and directed medially and slightly cranially (in an angle that would draw an imaginary line with the premolars on the opposite side of the head) until it hits bone. At this site, the maxillary nerve enters the pterygopalatine fossa.
- **Extraoral approach 2** (extraoral, from bony orbit): Approach the pterygopalatine fossa from the bony orbit. The needle is placed at the midpoint of the ventral rim of the bony orbit and inserted straight down between the globe and the bone.
- **Intraoral approach**: Open the mouth as wide as possible. From inside the mouth, insert a short needle no more than 2-4 mm (to avoid being close to the globe) inside the mouth just caudal and medial to the last molar.
- **Infraorbital canal catheterization:** New one! I haven't tried it. Literally a catheter is placed through the infraorbital canal to a distance that would reach the caudal aspect of the canal. Open access: Evaluation of a modified infraorbital approach for

a maxillary nerve block for rhinoscopy with nasal biopsy of dogs. Fizzano KM, Claude AK, Kuo LH, et al. Am J Vet Res. 2017 Sep;78(9):1025-1035.

For all 4 techniques, aspirate and inject. The volume that can be injected is approximately 0.1 to 1.0 ml, depending on the patient's size.

Mandibular nerve block for dentistry and mandibular surgery/trauma pain

*Abstract truncated due to space

006 / #23

A DAY IN THE LIFE OF A NUTRITIONIST IN GENERAL PRACTICE

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ABSTRACT BODY:

A DAY IN THE LIFE OF A NUTRITIONIST IN GENERAL PRACTICE

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In this lecture, I intend to share how I approach nutrition in my clinical cases in general practice. Obviously, each day is different and while some other presentations focus only on ideal cases where perfect management has resulted in optimal outcomes, I will try to provide a novel and honest perspective from someone with specialist level training working within the constraints of varying owner motivations, knowledge and financial capabilities. My belief is that we need to show considerable compassion, patience and flexibility with many owners in order to achieve the best-possible outcome for each of the animals entrusted to our care. Our acceptance that ideal outcomes are not always possible also alleviates some of the stress we carry as practitioners to achieve functional or at least easily defensible outcomes. The WSAVA Global Nutrition Committee has wonderful resources that provide guides to processes, and a toolkit of information that can assist with both personal development and guiding owners towards choices that support the health and wellbeing of their pets1. By building a collaborative, non-judgemental, open, trusting relationship with our clients, we can improve our ability to optimise the care of their pets by working towards incorporation of these guidelines. While this may require irrepressible patience and an acceptance that we will fail with some cases, by maintaining a commitment to ongoing personal development, we will truly optimise the care we can provide in our clinical setting.

Clinical cases that may be discussed include those where nutritional assessment and intervention may need to be delayed or even ignored in preference to obtaining emergency care or pursuing a diagnosis where euthanasia may the owner's chosen option. We will also cover cases where raw-meat-based diets, grain-free diets or other less-common options are chosen by owners along with nutritional approaches for novel species. My approach to cases with owners that are sometimes portrayed as difficult or ignorant will be offered.

In addition to discussing WSAVA resources and clinical cases, recent publications that discuss client communication around nutrition2,3, articles that elucidate impacts of various feeding methods4,5,6,7 and recent nutrition-based continuing education will be incorporated so that attendees with hopefully acquire resources that may guide ongoing learning opportunities.

Finally, the importance of being compassionate with oneself will be examined. While we often start our careers looking to minimize uncertainty and vulnerability, by maintaining a growth mindset and gaining experience over time we can guide our own personal development. By developing and implementing healthy coping strategies we can ease some of the ongoing stressors that we encounter with our work. Sharing how we prioritize this process may help with minimizing the scourge of mental health struggles within our field.

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007 / #24

INTEGRATING ORNAMENTAL FISH MEDICINE INTO A SMALL ANIMAL PRACTICE

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ABSTRACT BODY:

On Long Island, in the New York metropolitan area, the demand for advanced veterinary care for ornamental and pet fish has existed for many years and has been growing slowly. The speed of growth has not been faster due to the general lack of knowledge by the pet fish owning segment of the public as to the availability of veterinary services. The advent of modern web-based information is accelerating the pace of this demand. Small animal/exotic veterinary practices are now being consulted more frequently about pet fish care. The addition of these services is feasible and can be integrated profitably. Based on surveying other veterinary practices by this author, it was determined that a) the capitalization of the pet fish practice with no hospital facility most resembles the business model of the relief (per diem) veterinarian b) compared to the relief veterinarian, the salary is superior and comparable to the small animal/exotic specialist c) for the practice owner or principal veterinarian, it was hard to offset the lost productivity as a pet fish vet compared to the regular in-office services, and d) the small animal/exotic employed vet, working part time at the facility, part time as a pet fish vet, was very profitable. In a follow-up survey, 5 ornamental/pet fish veterinarians practicing at least 10 years in this specialty were asked to respond to questions concerning the business aspects of their practice. Based on the type of practice within the specialty, these aspects varied considerably. For the existing small animal/exotic practice, the introduction of this specialty requires very minimal new equipment purchases and most standard practice tools, drugs and lab services are utilized. The primary difference, based on the very high (>90%) call volume for koi consultations, is the need to practice onsite rather than via office visit. This often translates to more travel time than actual consultation, with the need to adequately capitalize this time. Fee structures vary considerably, but all surveyed practitioners calculate this travel time into their fees.

The intangible benefits of ornamental/pet fish practice for the small animal exotic practitioner are the opportunity to leave the office and enjoy practicing outdoors, especially during the warmer months. Additionally, many clinical cases offer challenging diagnostic and treatment dilemmas to stimulate the professional mind.

A description of the various services provided by the pet fish veterinarian will be covered in this presentation.

ECOGRAFIA Y RADIOGRAFÍA COMO COMPLEMENTO EN EL DIAGNÓSTICO DE LAS EFUSIONES PLEURALES

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ECOGRAFIA Y RADIOGRAFÍA COMO COMPLEMENTO EN EL DIAGNÓSTICO DE LAS EFUSIONES PLEURALES

INTRODUCCIÓN

La pleura es una membrana serosa de origen mesodérmico que recubre el parénquima pulmonar, el mediastino, el diafragma y la superficie interna de la pared torácica. Anatómicamente se subdivide en pleura visceral y pleura parietal según la estructura a la que recubre.

De forma natural, hay una pequeña cantidad de líquido entre ambas hojas pleurales, aproximadamente entre 0.1 y 0.3 ml/kg, que tiene como función facilitar el deslizamiento de los pulmones dentro del tórax durante los movimientos respiratorios y mantener independientes ambas membranas pleurales.

La efusión pleural es una afección relativamente frecuente en perros y gatos; la cual es una acumulación anormal de líquidos en la cavidad pleural y constituye una manifestación clínica común a numerosos procesos primarios y secundarios.

El fluido pleural puede acumularse de forma patológica como consecuencia del aumento en su formación o disminución de su resorción (1,2,3). La cantidad de líquido presente en el espacio pleural aumentará en el caso de presentarse alguna de las siguientes alteraciones: aumento de presión hidrostática capilar (ej: fallo cardiaco congestivo), disminución de la presión oncótica (ej: hipoalbuminemia), alteración del drenaje linfático (ej: neoplasia), aumento de permeabilidad capilar (ej: inflamación, infección, neoplasia) (4).

La cantidad de líquido determinará la magnitud de la sintomatología clínica, en ocaciones las cantidades de líquido anormal son poco significativas y representan un hallazgo incidental durante los exámenes clínicos y de imágenes; mientras que, en ocaciones, la presencia de abundante cantidad de líquido es la principal responsable de la sintomatología clínica del paciente, siendo una prioridad la estabilización del mismo y el diagnóstico oportuno para la correcta elección del tratamiento.

De las técnicas de imagen, la radiografía lateral como dorso ventral de tórax y la ecografía torácica son las técnicas más accesibles y sencillas por encima de la tomografía computerizada (TM) (5).

La radiografía y ecografía son diagnósticos por imágenes complementarios, brindando cada una de ellas información rápida y precisa de la cantidad y ubicación del líquido en la cavidad torácica; permitiendo así la toma de desciciones frente al abordaje del paciente para su tratamiento y pronóstico.

En especial, la ecografía es útil durante el drenaje y caracterización del líquido, asi como el seguimiento del paciente.

Abordaremos, en el desarrollo de la charla, casos clínicos en los cuales la principal presentación clínica fue disnea severa, con disminución de moderada a severa en la saturación de oxigeno; frente a lo cual se realizó, además de la estabilización del paciente, el diagnóstico de efusión pleural mediante el uso de radiografía y ecografía como técnicas de diagnóstico por imágenes; además determinaremos las ventajas del uso de cada una de las técnicas para el diagnóstico y seguimiento de la efusión pleural como signo principal de diferentes patologías.

RADIOGRAFÍA

La valoración radiográfica del tórax en pequeños animales es la prueba diagnóstica realizada con mayor frecuencia ya que oporta gran información de las patologías torácicas, por lo que ha alcanzado una gran importancia en la clínica diaria de pequeños animales. Igualmente pueden usarse para valorar la respuesta terapéutica o progresión de una enfermedad.

El reconocimiento de las alteraciones radiográficas se basa en el conocimiento correcto de la apariencia radiográfica normal. Cada estructura presenta un tamaño, forma, opacidad, márgenes, contorno, número y localización característicos. La clave de la interpretación se basa en evaluar las regiones extratorácicas hasta las intratorácicas (6).

RADIOGRAFÍA EN EFUSIONES PLEURALES

Bajo circunstancias normales la pleura no es visible. La alteración pleural más frecuente es la presencia de efusión pleural. La efusión pleural se identifica mejor en proyección dorso ventral ya que en ésta el líquido se acumula en un menor espacio (7).

En caso de tener que valorar la silueta cardíaca en un paciente con efusión pleural, la proyección indicada es

la ventro dorsal, en ésta lel líquido se acumula en un mayor espacio, y los pulmones aireados flotan hacia el hilio rodeando el corazón, asi se mejora el contraste para la visualización de la silueta cardíaca (8).

Características radiográficas en la efusión pleural:

- Distensión del espacio pleural
- Aumento de densidad
- Retracción de los márgenes pulmonares
- Visualización de las cisuras pleurales (en el caso de derrames pleurales leves, se observan las fisuras interlobulares, como signo temprano de derrame pleural)
- Pobre visualización del corazón y diafragma (efecto silueta)

Otros signos radiográficos de efusión peural son bordes pulmonares redondeados (sobre todo en procesos crónicos), aumento y menor definición de espacio mediastínico y separación de borde pulmonar de vertebras torácicas.

La efusión pleural suele ser bilateral pues la pleura presenta fenestración que comunica ambos hemitórax; se observará efusión unilateral en casos en que ésta se encuentre encapsulada por la formación de absceso, si las fenestraciones están cerradas por procesos neoplásicos o en piotórax (8). La efusión pleural unilateral se observa mejor en proyección dorso ventral ó ventro dorsal.

ECOGRAFÍA

La ecografía pulmonar, como herramienta de imagen, es mucho más sensible que la radiografía pulmonar para el diagnóstico de efusión pleural, ya que permite la visualización de pequeñas cantidades de líquido libre pleural (a partir de 2 ml/kg).

Es por tanto parte indispensable del diagnóstico cardiopulmonar.

ECOGRAFÍA EN EFUSIONES PLEURALES

En efusión pleural de moderada a severa, se puede observar además la presencia de atelectasia pulmonar debido a la presión del líquido sobre el parénquima pulmonar. La imagen del pulmón atelectásico flotando en el líquido pleural libre se denomina signo de medusa o lengua (4).

La ecográfica también ayuda de manera indirecta a tipificar el líquido encontrado en la cavidad pleural observando su ecogenicidad y densidad. También puede brindarnos en muchas ocaciones el diagnóstico de la patología que origina el derrame pleural.

Además es una herramienta útil para la toma de muestras y drenaje del líquido de manera sencilla y con menor riesgo de dañó en órganos torácicos.

Tipificación del líquido pleural

Una vez obtenida la muestra, a través de la punción ecoguiada, es importante la clasificación del mismo. En medicina veterinaria la diferenciación tradicional entre trasudados, trasudados modificados y exudados en función de la cantidad de proteína total y el recuento celular en líquido pleural se considera el paso inicial en el estudio de cualquier efusión pleural, pero al igual que ocurre en medicina humana existen superposición de valores, sobre todo en lo referente a diferenciar un trasudado modificado y un exudado.

¿Qué determino en el líquido pleural?

Una vez obtenido el líquido pleural mediante toracocentesis, se debe proceder del siguiente modo:

- Reservar una muestra para cultivo en la misma jeringa donde se ha extraído o en tubo estéril sin conservantes, esto se debe realizar siempre para casos de líquidos con mal olor, líquidos purulentos, opacos o cremosos.
- Si se va a demorar la llegada de la muestra al laboratorio, se debe enviar también una torunda junto con medio de cultivo, de este modo si se degrada la muestra puede hacerse el cultivo microbiológico.
- El resto de la muestra debe depositarse en tubo de EDTA para realizar: Citología y recuento celular (macrófagos, células mesoteliales), determinación de proteínas totales mediante refractómetro (en el caso de líquidos opacos o cremosos, se realizará en el sobrenadante), lactato deshidrogenado (LDH), triglicéridos (en caso de quilo), pruebas específicas de patologías compatibles

Tabla 1: Parámetros laboratoriales diferenciando trasudados, trasudados modificados y exudados.

Parámetro	Trasudado	Trasudado modificado	Exudado
Proteína total (g/L)	<25	25-50	>30
Gravedad específica	1.018	1.010 – 1.030	>1.080
Células x 109/L	<0,5	0,5 - 5,0	>5,0
Bacterias	No	No	Posible

Fuente: Sturgess K, Villar D.(8).

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FOOD ALLERGY IN THE FELINE PATIENT

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ABSTRACT BODY:

With the understanding of canine atopic dermatitis, our understanding of atopic states in cats has lagged behind. One of the least understood hypersensitivity disorders in either dogs or cats, though, is food allergy. The reported prevalence varies greatly and many studies have not confirmed the diagnosis by dietary challenge. We also lack good knowledge of the pathogenesis of these disorders in dogs and cats, and therefore have difficulty in making the diagnosis efficiently and correctly. Yet a food allergy is likely to be the easiest to control: we simply avoid the offending allergens.

Food allergy is only one type of adverse food reaction, and is characterized by involvement of the immune system in the pathogenesis of disease. Other adverse food reactions include food intolerances, pharmacologic reactions, and toxin-mediated reactions. Food allergies in people are divided into IgE-mediated and non-IgE-(cellular) mediated reactions. The best understood of these latter include eosinophil-mediated diseases of the gastrointestinal tract, but there are other inflammatory diseases of the gut that have been attributed to food hypersensitivity. Even in humans, food allergies are difficult to understand.

The major dermatologic sign of food allergy in cats, as well as dogs, is pruritus. The classic food allergic cat has intense pruritus and evidence of self-trauma around the head and neck, but cats can also have one of the triad of syndromes we often associate with allergic skin disease: hair-pulling/overgrooming, military dermatitis, and eosinophilic granuloma complex, particularly eosinophilic plaque. Our suspicions of food allergy are increased in pets with concurrent dermatologic and gastrointestinal disease, but either skin disease or gastrointestinal disease can occur as the sole organ involved. It is of interest to me that many cats are treated chronically for inflammatory bowel disease; it seems prudent to try to determine whether dietary management could help these patients, in order to avoid the use of chronic steroid therapy.

Diagnosis of food allergy can be difficult. Most veterinary dermatologists agree that the diet history and a diet trial remain the gold standard for diagnosis, but we don't agree on what constitutes the appropriate diet and how long it should be run. Furthermore, diet trials are dependent on the owner's ability to adhere to them, and to recognize and track the changes that occur when potentially allergenic foods are reintroduced into the diet. Some clients are more motivated than others to explore this option. The diagnosis of food allergy can be complicated when the pet has atopic dermatitis. It is often advocated to rule out food allergy first prior to diagnosis and treatment of atopic dermatitis. Yet pets with both disorders may not show sufficient improvement during the food trial for an owner to recognize improvement. It is not helpful therefore to make hard and fast rules about how to approach the diagnosis of these disorders. Each patient needs to be addressed individually.

The first step in diagnosing food allergy is a careful history. In areas with limited pollen seasons, seasonal pruritus rules out the need to pursue food allergy. For the vast majority of cats and dogs, food allergy is a nonseasonal disease. A careful food history is very important, but often hard to elicit. What we need to know is what proteins and carbohydrates the animal has eaten in the past. Most clients don't read the list of ingredients on the label and they may forget about treats, tablescraps, flavored medications, and edible toys. Cats that go outdoors may be eating a variety of things that we will never discover. Many clients believe that a change in diet will precipitate food allergy but that simply is not the case. It is difficult to break gastrointestinal immune tolerance. Fairly long term exposure to the dietary allergen in question is the norm.

The second step in diagnosing food allergy is selecting the appropriate diet for the trial. Choosing a novel protein can be difficult because of our lack of knowledge about cross-reactive proteins in dogs and cats. There is immunologic evidence that IgE from beef-allergic humans can sometimes crossreact with venison and lamb, and that IgE from chicken-allergic humans can crossreact with duck. Recent studies have shown that these diets are contaminated by small amounts of chicken or beef or other dietary ingredients; thus, they are not truly limited ingredient diets and are poor choices for diagnosis, although for some patients they might be suitable for feeding after the diagnosis has been made. The alternative to the novel protein diet is the hydrolyzed diet. There are several extra elements to consider when we are trying to use a diet for cats with suspected food allergy.

Convincing owners to be strict with the food trial can be difficult. Many clients enjoy feeding their cats and dogs treats. Once the diet trial is accepted by the client, the next step is to decide how long to feed. We should see signs of improvement within 6-8 weeks. There may not be complete resolution of the signs but the response should be significant enough for the client to observe. If there is no improvement in that time is less likely that the diagnosis is food allergy.

Re challenge with food is important, because we can determine what foods precipitate itch, we can get our patients back onto a commercial diet and simply avoid the offending protein. This gives the client and the pet more freedom with diet choices and selection of treats. Many clients will prefer not to go through this phase and continue to feed the elimination diet.

Is there a role for skin testing or blood allergy testing in the diagnosis of food allergy? Skin testing has proved uniformly unreliable and none of the veterinary dermatologists will recommend this test. Blood allergy testing is available from several companies. Most veterinary dermatologists have found these tests unhelpful as well.

Many pets with food allergy have other allergies as well. Concurrent flea allergy and food allergy is quite common, and it is not unusual for food allergic pets to have concurrent atopic dermatitis/atopy. Determining the underlying cause of pruritus in cats and dogs, therefore, can be quite complicated and can take a considerable amount of time.

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SELECTIVE BREEDING AND PET HEALTH. WHAT CAN WE DO?

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ABSTRACT BODY:

Domesticated animals are produced through the process of selective breeding. The term domestication can be defined as the process by which humans alter how an animal looks and behaves. In order to benefit humans by selecting for desirable traits when breeding.

Cats were originally thought to be domesticated approximately 3,600 years ago in Northern Africa Skulls of cats identified as mainly being of the species *Felis sylvestris lybica* (the African wildcat) have been found in Egyptian cat burial grounds but more recent evidence suggests that feline domestication likely occurred around 10,00 years ago in the middle east. It is likely that feline domestication occurred sympatrically with cats becoming increasingly tolerant of humans as they benefitted from predating rodents found in grain stores as human agriculture developed. Whilst the domestic cat was classified as *Felis catus* in 1758 by Carolus Linnaeus, it is actually a subspecies of the African wildcat and may be called *Felis sylvestris catus.*

No one knows exactly when or why the first domestic dogs appeared but it is likely that dog domestication occurred between 20,000 and 40,000 years ago years ago. Domestic dogs are originally descended from the Eurasian grey wolf. The fossil record suggests that dog-like animals have been around for more than 30,000 years. And genetic data suggests that dogs appeared around 15,000 years ago. Debate still exists as to whether dogs were domesticated at one or more locations concurrently. With the first domestic dog remains appearing in the early Neolithic period.

Selective breeding can be used to produce healthy animals by selecting against disease traits and genes, or by increasing the fitness of animals coping with specific environmental challenges. In 1959 Dr, Dmitri Belyayev domesticate wild silver foxes by selecting them only for their tameness and responses to humans. Naturally, silver foxes would avoid humans and run away from them. But over 50 generations later, almost all of these foxes seek out human contact. The domestication process also resulted in some unexpected phenotypic changes. The foxes were selected only for tameness, not for any physical characteristics but such as changes in coat colouring, floppy ears, and rolled tails occurred. It is impossible to be absolutely selective when breeding for a specific trait, as changes in behaviour are linked to the same genes that also affect physical changes. For example white plumage hens show significantly higher aggression than red-brown plumage hens (1).

Because of these gene linkages, selecting for a particular behaviour, or physical characteristic can often have inadvertent consequences. For example, merle coat colouration is expressed by the merle gene and inherited in an autosomal, incomplete dominant manner. It has the effect of deleting pigments in hairs, skin, nose and mucous, iris and tapetum lucidum, and stria vascularis of the inner ear. This depigmentation of the stria vascularis causes early death of sensory hair cells resulting in hearing impairments. Dogs with the homozygous merle mutation may demonstrate excessive white coat, pink skin, nose and mucous, light blue irises, and irreversible hearing impairments.

Genetic diversity of domestic dogs was reduced by the domestication process, but more recently, genetic diversity has been severely reduced again by selective breeding for aesthetics (3) which became highly fashionable in the 19th and 20th centuries, a period known as the 'Victorian Explosion'. This resulted in the development of many of the dog breeds we recognise today. Over 400 breeds of dog are recognised, most originating for selective breeding that occurred at this time, though more recently, breed standards have shifted to promote more extreme conformations resulting in an epidemic of health and welfare problems created as animal aesthetics are prioritised over animal welfare. Selective breeding for aesthetics is problematic for both cat and dog health and welfare as it results in a spectrum of conformational and genetic defects and diseases with extreme conformation such as brachycephaly, skin folds, chondrodystrophy (such as that seen in Scottish fold cats), hip dysplasia, elbow dysplasia, anal furunculosis, atopy, genetic disease such as urate abnormality in Dalmatians and mitral valve disease in Cavalier King Charles Spaniels. It is important that veterinarians involved in animal breeding are aware of expert guidance and the ethical standards expected globally such as:

"The WSAVA calls on veterinarians and breeders to ensure that criteria used for the selection of breeding animals include the ability to reproduce naturally and exclude anatomical characteristics that predispose to

hereditary disease, such as extreme conformations including size, skin folds, angulation and extremely short faces (brachycephaly). If a breed demonstrates a disease-predisposing anatomy then selection should be towards a moderate and less extreme anatomy"

https://wsava.org/global-guidelines/hereditary-disease-guidelines/

Whilst many health schemes exist to reduce hereditary and conformational diseases in dogs and cats, insisting on rigid conformational and aesthetic standards may unfortunately limit the impact any of these schemes can have. As a profession should we also consider the positions we take on the status quo of breed standards? Similarly we should also consider the breeds that we may promote and use in marketing our veterinary businesses to ensure that this use of animals aligns with good standards of health and welfare.

Research suggests that breeding strategies incorporating screening schemes can be successful in significantly reducing the prevalence of an inherited disorder and improving the overall health of certain breeds. Screening has significantly reduced the prevalence of patellar luxation in Dutch Kooiker dogs. Conversely a 2 year analysis of the mitral valve disease screening scheme in Cavalier king Charles spaniels in the UK has shown no impact. (3). However a more recent study suggests that the age at which 50% of CKCS female dogs examined by GP veterinary surgeons developed a murmur increased by 0.6 years between 1991-2010, so longer term data analysis may identify greater benefits. Whilst this is promising, we should give consideration as to whether the speed of progress and its impact on animal welfare is sufficient. Dutch Kooiker still have a higher prevalence of patellar luxation than other breeds and CKCS still suffer significant mitral valve diseases as well as a host of other genetic pathologies. How do we set the threshold for acceptable animal welfare? With approximately 400 inherited disorders identified, many of which are complex and polymorphic, it may be that successfully breeding away from susceptible individuals with a breed population is no longer possible due to the prevalence of these disorders within some breeds.

Outcrossing can improve the health of some breeds whilst having little to no phenotypic impact. For example in 1973 a purebred dalmation (all of whom have mutations of SLC2A9 resulting in abnormal uric acid metabolism) was crossed with a pointer, homozygous for the normal gene variant (4). In 2011 a direct descendant of that dog competed at Crufts, but faced a backlash from breeders and clubs concerned about the purity of the breed. Increasing genetic diversity is known to reduce the risk of hereditary disease (5), but whilst artificial concepts of breed 'purity' continue to be prioritised over genetic and conformational health, veterinarians are likely to continue to deal with potentially preventable health problems. The social factors driving the continued selection of extreme conformations are complex, and further research in veterinary and social sciences may be helpful in challenging the cognitive dissonance identified in owners of brachycephalic pets for example (6).

Every day, veterinary professionals deal with the health and welfare impacts of selective breeding decision-making. For some breeds, screening and breed health schemes offer hope of a healthier future, but for others, rigid concepts of breed purity and aesthetics may condemn thousands of animals to a lifetime of suffering. The One-Health implications of managing preventable welfare problems in our pets are well documented (7) and so we should be mindful of the impacts on ourselves our students and our colleagues, whilst making efforts to promote responsible breeding and purchasing decisions in breeders, owners, and the general public.

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TRASTORNOS EN CALCIO Y POTASIO: CUANDO LOS ELECTROLITOS TE LLEVAN A LA SALA DE URGENCIAS

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ABSTRACT BODY:

TRASTORNOS EN CALCIO Y POTASIO: CUANDO LOS ELECTROLITOS TE LLEVAN A LA SALA DE URGENCIAS

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La precisión con la que el organismo mantiene los niveles normales de electrolitos y otras sustancias como la glucosa es asombrosa. Los mecanismos homeostáticos están en permanente vigilia para asegurar el buen funcionamiento de todos los sistemas fisiológicos.

Potasio y Calcio son dos electrolitos presentes en multitud de funciones fisiológicas y sus alteraciones pueden provocar situaciones en las que la vida del paciente corra peligro.

POTASIO (K)

El K es uno de los cationes mayormente localizado a nivel intracelular. Los niveles plasmáticos en perros y gatos suelen estar entre 3.5-5.5 mmol/L.

Una de las principales funciones del potasio es generar el potencial de membrana en reposo que es de vital importancia en el buen funcionamiento del musculo esquelético y cardiaco, tejido nervioso y en los epitelios transportadores.

From Fluids electrolytes and Acid Base disorders in Small Animal medicine.4th edition Di Bartola

La hiperkalemia va a reducir el potencial en reposo haciendo a la célula hiperexcitable.

El principal reservorio de potasio del organismo se encuentra en el musculo esquelético. Las alteraciones en el potasio van a producir cuadros de debilidad muscular y alteraciones en la conductibilidad cardiaca principalmente.

Un estudio retrospectivo reciente analizó los factores de riesgo y la severidad de los signos clínicos y pronóstico de pacientes en la sala de urgencias con alteraciones en el nivel de potasio en 2441 casos atendidos en urgencias[2].

Los niveles de potasio sérico dependen del balance de K que entra y sale del organismo (ingesta oral – perdidas en orina y tracto digestivo) y también de las translocaciones de K entre el espacio intra y extracelular.

Tanto la hipokalemia como la hiperkalemia pueden producir signos de debilidad muscular (principalmente la hipokalemia) y alteraciones en el ritmo cardiaco (bradicardia > taquicardia) en los pacientes.

Los principales factores de riesgo de **hipokalemia** fueron la anorexia prolongada (o fluidoterapia sin suplementación de K), la alcalemia, hiperglucemia, tratamientos con insulina y beta agonistas y las perdidas excesivas renales (fallo renal intrínseco, diuresis post obstructiva, acidosis tubular renal) y digestivas (vómitos/diarreas), los tratamientos con diuréticos (tiazidas y del asa de Henle), la Diabetes cetoacidótica (DKA) y el hiperadrenocorticismo.

El tratamiento de la **hipokalemia** moderada severa (K< 2-3mmol/L) consiste en la administración de KCl o KPO3 intravenosa o gluconato potásico oral. Generalmente se plantea una infusión continua a 0,5 mEq/kg/h durante 4 h y se monitorizan los niveles séricos de K frecuentemente.

Respecto a la **hiperkalemia**, los factores principales fueron excesiva suplementación con K (oral o intravenosa), la acidemia, betabloqueantes, lisis celular por reperfusión tisular o quimioterapia, obstrucción uretral, fallo renal (agudo >crónico), hipoadrenocorticismo, el uroabdomen y la administración de IECAS.

La mortalidad de los pacientes hiperkalémicos fue superior a los normokalémicos.

En la **hiperkalemia**, el tratamiento dependerá de la causa y severidad. La fluidoterapia intravenosa podrá corregir situaciones leves o moderadas.

Ver tabla para corrección de hiperkalemia moderada -severa.

Medicación	Dosis	Acción
Gluconato cálcico 10%	0,5-1,5 ml/ kg en 20 min. Monitorizar ECG	Protección miocardio mientras corregimos hiperK. Solo si no hipercalcemia
Bolo de Dextrosa 50% iv	0,5-1 ml/Kg diluida 1:4	Liberación insulina, movilización K al interior de la célula
Insulina regular	0,25 ui/kg con bolo de dextrosa + CRI dextrosa 2,5-5 %	Movilización K hacia el interior de la célula

Las membranas celulares están hiperexcitables en hiperkalemia y el aumento de los niveles de calcio circulante elevara el umbral de despolarización, estabilizando así las membranas y la función cardiaca.

El bicarbonato también puede usarse para corregir hiperkalemia pero rara vez es necesario y su uso se asocia a un incremento de la mortalidad.

CALCIO (CA)

El calcio interviene en multitud de funciones fisiológicas (contracción muscular, transmisión neuromuscular, metabolismo intracelular, constitución del esqueleto, estabilización de membranas, tono vascular, producción hormonal, coagulación sanguínea y crecimiento y división celular indispensable en curación de heridas). Es también mensajero intracelular.

El 99% del calcio del organismo se encuentra en el esqueleto y solo el 1 % en tejido blando. De ese 1%, el 0,9 % se encuentra intracelular y solo el 0,1% está en el espacio extracelular/plasma.

El calcio del espacio extracelular puede estar unido a proteínas (40%), formando complejos de calcio (fosfato, lactato, bicarbonato cálcico) (5%) y el 55% restante se encuentra en la forma ionizada (iCa) que es la biológicamente activa.

La hipoalbuminemia afectara a la medición de calcio total. Mediremos el calcio ionizado siempre que sea posible. Los mecanismos de homeostasis del calcio mantienen los niveles de iCa en márgenes muy estrechos. Las hormonas involucradas en esta homeostasis son la PTH, la vitamina D y la calcitonina.

Esquema homeostasis Calcio

Tanto la hipocalcemia como la hipercalcemia severa pueden ser letales para el paciente, especialmente si los cambios se producen de manera rápida.

El proceso diagnostico incluye analítica de sangre, orina, radiografías torácicas, ecografía abdominal, citología, analítica hormonal (PTH, rPTH y medición de metabolitos de la vitamina D).

Se habla de **hipercalcemia** cuando los niveles de Ca ionizado son >1.5 mmol/L en perros y 1.4 mmol/L en gatos.

Los pacientes en crisis hipercalcémicas (con niveles de iCa > 1.8 mmol/L) pueden presentar convulsiones, ataxia, coma, fasciculaciones musculares y arritmias cardiacas.

No obstante, cuando la evolución de la hipercalcemia es crónica y los pacientes presentan signos clínicos menos severos (PU/PD en perros, anorexia, constipación, perdida peso).

Las causas más frecuentes de hipercalcemia en perros son cáncer (p.ej: linfoma, adenocarcinoma sacos anales, carcinoma, timoma, mieloma múltiple), fallo renal, hiperparatiroidismo e hipoadrenocorticismo. En el caso de los gatos, hipercalcemia idiopática y fallo renal son causas más frecuentes de hipercalcemia que la neoplasia. El linfoma mediastínico puede presentarse con hipercalcemia.

El acrónimo “HARDIONS” en ingles nos puede ayudar a recordar los principales diagnósticos diferenciales frente a la hipercalcemia. H por hiperparatiroidismo y hipercalcemia humoral neoplásica, A por enfermedad de Addison, R por renal, D por hipervitaminosis D, I por idiopática, O por osteólisis/ enfermedad granulomatosa, N por neoplasia y S por spurious (fallo laboratorial). No obstante, la lista de diagnósticos diferenciales no se limita a estos.

Las consecuencias de la hipercalcemia es la mineralización de tejidos blandos como el corazón y el riñón siendo esta más severa cuando [ca mg/dl] x [P mg/dl] > 60.

El tratamiento de la hipercalcemia consiste en identificar y controlar la causa.

Fármaco	Dosis	Objetivo
Fluidoterapia IV (NaCl 0,9%)	Al menos x2 dosis mantenimiento	Rehidratación, calciuresis
Furosemida	1-2 mg/kg q6-12 h o 0.1-1 mg/kg/h CRI	Calciuresis
Glucocorticoides	Dexametasona: 0,1-0,2 mg/kg Prednisona: 1-2 mg/kg q 12h	reducen la resorción ósea, la absorción intestinal y promueven excreción renal de calcio
Calcitonina	4-6 ui/Kg SC q 8-12h	reducen la actividad osteoclástica. Usado en hipervitaminosis D
Bisfosfonatos	Pamidronato:1-2 mg/kg infusion 4h cada 3-4 sem Alendronato:1-3 mg/kg/sem SC	reducen la actividad osteoclástica. Usado en hipercalcemia idiopática
Calcimiméticos	7,5-30 mg/perro/d	bloquean la síntesis de PTH. Futuro? $$$

La **hipocalcemia** suele ser una causa más frecuente de presentación de urgencias. Si iCa están por debajo de 1 mmol/L y si la evolución de la hipocalcemia ha sido rápida, el paciente puede presentar cambios comportamentales, fasciculaciones músculos faciales, marcha rígida, atáxica, prurito facial, arritmias cardiacas (taquicardia, prolongación segmento QT, bloqueo atrioventricular), hipotensión refractaria y parada respiratoria.

La situación más clásica en emergencias es la hipocalcemia/eclampsia puerperal en hembras post parto.

También perros en el postoperatorio de paratiroidectomia por hiperparatiroidismo primario están en riesgo de presentarse hipocalcémicos. Se suele recomendar monitorización frecuente e incluso suplementación con calcio oral o calcitriol tras la cirugía.

Otros diagnósticos diferenciales son enfermedad renal crónica, hipoparatiroidismo primario, pancreatitis, malabsorción intestinal, enteropatía con pérdida de proteínas (PLE), transfusión con anticoagulantes que contienen citrato, intoxicación con etilenglicol entre otras.

La presencia de hipocalcemia en pacientes sépticos se considera un factor pronóstico negativo.

El tratamiento de hipocalcemia en emergencias consiste en la administración de gluconato cálcico (o cloruro cálcico) siempre intravenosa. La administración subcutánea de sales de calcio puede conducir a necrosis tisular.

Infusión de Gluconato cálcico al 10% (0,5-1,5 ml/kg) en 20 min con constante monitorización de ECG. Los signos clínicos pueden tardar 30-60 min en resolverse una vez que los niveles de calcio están normalizados.

Si la situación no se normaliza con un bolo de calcio, CRI de calcio elemental a dosis de 1-3 mg /kg/h debe considerarse mientras se inicia la suplementación oral con sales de calcio (25-50 mg/kg de calcio elemental) y calcitriol (20-30 ng/kg q 24h dividido en 2 durante 3-4 días y luego 5-15 ng/kg d dividido en 2 tomas como mantenimiento).

En los pacientes con hipoparatiroidismo primario el objetivo es mantener los niveles de calcio justo por debajo del valor normal para evitar los efectos deletéreos de la hipercalciuria sobre el riñón.

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ABSTRACT BODY:

Los ataques epilépticos son una causa muy frecuente de visitas a la clínica diaria.

Un ataque epiléptico es la manifestación clínica de una excesiva actividad eléctrica, hipersincrónica, de un grupo de neuronas de la corteza cerebral (focos epileptogénicos). Aparecen como consecuencia de un desequilibrio entre mecanismos excitadores e inhibidores del encéfalo.

Los ataques epilépticos se pueden clasificar según su semiología (visto anteriormente), según el número de episodios (ataque aislado, cluster o status epiléptico) y según su etiología (causas). A lo largo de la historia, ha habido diferentes clasificaciones etiológicas, siendo la siguiente la más aceptada actualmente:

- Ataques epilépticos reactivos: Reacción transitoria del cerebro frente a una alteración sistémica metabólica, tóxica o nutricional. Por tanto, tienen un origen extracraneal. Lo más frecuente son los tóxicos, la hipoglicemia y la hipocalcemia.
- Epilepsia secundaria o estructural: Son ataques epilépticos recurrentes causados por una lesión estructural identificable al cerebro (encefalopatías vasculares, procesos inflamatorios- infecciosos (meningoencefalitis), traumatismos (TCE), anomalías congénitas, neoplasias o procesos degenerativos). Según la reseña del paciente, la presencia de otros signos neurológicos y el inicio y la progresión de los mismos, una entidad será más o menos probable. Suelen ir acompañados de otros déficits neurológicos.
- Epilepsia idiopática: Es una enfermedad del cerebro consistente en ataques epilépticos recurrentes, sin otros signos neurológicos. No existe ninguna casusa estructural identificable y se denomina epilepsia genética (en los casos donde se ha identificado el gen responsable: lagotto romangolo y pastor belga), de probable origen genético (cuando hay alta prevalencia de raza: >2%) o bien de origen desconocido. Tiene unas características clínicas determinadas:
- Historia de más de 2 ataques, separados 24 horas entre ellos
- Edad de presentación del primer episodio entre 6 meses y 6 años de edad
- Intervalo de más de un mes entre el primer ataque y el segundo
- Examen físico y neurológico interictal normal
- No alteraciones en las pruebas diagnósticas (análisis, RM, LCR)

El protocolo diagnóstico en pacientes con ataques epilépticos confirmados, irá destinado a determinar la causa que los produce. Consiste en una buena reseña y anamnesis, examen físico y neurológico, analíticas sanguíneas completas (para diagnosticar o descartar causas extracraneales), y pruebas de imagen (RM) y análisis de LCR para diagnosticar o descartar los procesos intracraneales.

El objetivo del tratamiento es disminuir la frecuencia, duración y severidad de los ataques, y mejorar la calidad de vida de los pacientes y sus propietarios. Existen diferentes fármacos en el mercado. El fármaco antiepiléptico ideal debe tener una eficacia excelente, un elevado índice terapéutico, pocos efectos secundarios, una farmacocinética que permite dosificarlo en 1 o 2 veces al día, barato, y buena palatabilidad. A pesar de que el fármaco perfecto no existe, si existe el “mejor” para cada paciente. Es decir, hay que individualizarlos en cada caso.

Según las evidencias científicas existentes, las opciones terapéuticas actuales recomendadas para el control a largo plazo son las siguientes:

- Perros: 1. Fenobarbital, 2. Bromuro potásico, 3. Levetiracetam, 4. Zonisamida, 5. Felbamato
- Gatos: 1. Fenobarbital, 2. Levetiracetam 3. Zonisamida.

Es importante recordar que hay fármacos de humana cuyo uso no está recomendado en perros y gatos debido a su toxicidad (fentoina, lamotrigina, primidona,…) o porqué tienen una farmacocinética inapropiada (carbamazepina, fenotina, valproato,..)

Se recomienda empezar el tratamiento en las siguientes situaciones:

- Epilepsia secundaria: se deberá tratar con antiepilépticos y también la causa primaria.
- Epilepsia idiopática en los siguientes casos:
- El periodo interictal es inferior a 6 meses (más de 3 ataques anuales)
- Los signos post ictales son severos y prolongados
- Se ha presentado un episodio de cluster o status epiléptico
- Ha incrementado la frecuencia, duración y/ o severidad de los ataques

Se considera que un paciente es refractario al tratamiento, cuando no se consigue un adecuado control de las crisis con la terapia adecuada y tolerada con dos medicaciones antiepilépticas. Aproximadamente el 20- 30 % de los pacientes con epilepsia se consideran refractarios. Sin embargo, antes de considerar que un paciente es refractario, debemos asegurarnos de que no estamos frente a un caso de pseudoresitencia (mal control de ataques debido a una mala dosis o frecuencia de los fármacos, niveles terapéuticos sanguíneos inadecuados, diagnóstico erróneo, interacciones con otros fármacos..). Se considera que este es la principal causa de mal control de ataques tanto en medicina humana como en veterinaria.

El manejo no farmacológico se recomienda en los pacientes refractarios a las medicaciones antiepilépticas convencionales. Se consideran como tratamiento alternativo para ayudar a estos pacientes, y siempre combinado con las terapias convencionales: nutrición, terapias alternativas (homeopatía, acupuntura), suplementos dietéticos (CBD), manejo comportamental, cirugía.

CAT-SPECIFIC ANESTHESIA & ANALGESIA TIPS

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ABSTRACT BODY:

Cats Facts

- Often nervous or fractious: Increased circulating catecholamines and increased Fear/Anxiety/Stress (FAS) = Increased dose of anesthetic drugs required
- Small body size: May be difficult to dose, to fit to monitoring & anesthetic equipment & to keep warm
- Species-specific drug metabolism: May not metabolize drugs the same as dogs do (eg, NSAIDs): Species-specific response to drugs; may respond differently than dogs do (eg, opioids sometimes)

These differences add to the fact that cats are at higher risk than dogs for anesthesia-related deaths (risk factor of 0.11% vs 0.05%, respectively, in healthy patients and 1.33% vs 1.4%, respectively; Brodbelt 2009).

Preanesthesia

As with other species, patients should be stabilized prior to anesthesia and premedication should be utilized to improve the safety of anesthesia by allowing a decrease in the dosages of induction and maintenance drugs. Anxiolytics are often very helpful to decrease FAS.

Common Previsit Pharmaceuticals

Gabapentin	100-200 mg/cat 2 hours before leaving home +/- night before leaving home
Trazodone	50-100 mg/cat, same timing as gabapentin, can administer with gabapentin
Maropitant	1-2 mg/kg orally 2 hours before leaving home, nausea/vomiting can cause FAS

Preoperative drugs or drug classes and key cat points:

Opioids	Excellent choice for all cats. Moderate to profound analgesia, minimal adverse effects, reversible effects. Many drug and delivery options. Cat specific opioids: Simbadol® (24-hr duration) & Zorbium® (4-day duration, transdermal).
Alpha-2 Agonists	Excellent choice for many cats. Mild to profound sedation with analgesia. Effects are reversible. Contraindicated in some forms of cardiovascular disease but may be beneficial in left ventricular outflow tract obstruction because of decreased heart rate. Causes emesis, prevent with maropitant.
Acepromazine	Often a good choice. Provides 4-8 hours of light to moderate sedation, which is a benefit, especially for many hospitalized cats. No analgesia, effects not reversible.
Benzodiazepines	Minimal adverse effects, minimal sedation in young, active patients. Can be effective, especially when combined with an opioid, in very young/very old/sick cats. Midazolam would be the most likely benzo to be used as a premed since it can be administered IM. AnxiolyticJ.
Ketamine	Often administered IM as part of an anesthetic protocol – so not really a premed but administered concurrently with premeds (usually dexmedetomidine and an opioid).

Alfaxalone	Can be used at a low dose (0.5-2.0 mg/kg) IM for moderate sedation of 20-45 mins in very young/ old or sick cats (in general, not enough sedation for healthy or excited cats). Better sedation when combined with an opioid.

Induction drugs and key cat points

Propofol	Can easily be titrated 'to effect'. Cleared by multiple routes. Causes mild to moderate dose-dependent cardiovascular and respiratory depression. Can be administered with a benzodiazepine or ketamine ('ketafol') to decrease propofol dose. IV only.
Alfaxalone	Can easily be titrated 'to effect'. Causes mild to moderate dose-dependent cardiovascular and respiratory depression. Can be administered with a benzodiazepine to decrease alfaxalone dose. Can be administered IM or IV.
Ketamine	Can be administered IM or IV. Minimal to no respiratory changes, provides mild to moderate increase in heart rate and blood pressure (through stimulation of the sympathetic nervous system). Cleared in part unchanged in the urine. MIGHT contribute to sympathetically-driven arrhythmias? No muscle relaxation, administer with a benzodiazepine or alpha-2 agonist. CRI for analgesia.
Tiletamine/ Zolazepam	Physiologic effects similar to ketamine/benzodiazepine. Very potent, small volumes easy to dose – and to overdose if not careful.
Inhalants	Not the safest choice for induction. Dose is very high – increases risk for anesthesia-related death. Staff are exposed to gases so human health concerns as well as cat health concerns.

Cat facts for induction drugs

- Propofol can be safely used in cats with hepatic lipidosis (Posner LP, et al. JAVMA 2008;232(12):1841-3), even though propofol itself is a lipid.
- The preservative in the 'newer' propofol (Propoflo28) is NOT toxic to cats (Taylor PM, et al. J Feline Med Surg 2012;14(8):516-26).
- Alfaxalone is an excellent option in cats for both sedation and induction. It causes dose-dependent cardiovascular and respiratory depression similar to that of propofol but absorption after IM administration is an advantage in cats and small dogs.
- KETAMINE IS NOT CONTRAINDICATED IN ANY CAT BREED (eg, Savannah or Maine Coons). The presence of cardiac disease, which these breeds may be more prone to, could maybe be a contraindication – but not the breed itself.
- Inhalant induction (mask or chamber induction) is a risk factor for anesthesia-related death and should NOT be the routine method of induction to anesthesia in cats (Brodbelt 2009).

Cat specific information on intubation:

- Intubate carefully (not cat-specific but cats are more difficult to intubate than most dogs).
- Apply a drop of lidocaine on each arytenoid. Cats are more prone than dogs to laryngospasm. The lidocaine reduces the likelihood of laryngospasm. Lidocaine does not cause adverse effects – cetacaine does.
- If still difficult to intubate, administer more induction drug. Don't intubate an awake cat.
- Inserting an endotracheal tube was a risk factor for anesthesia-related death in cats (Brodbelt 2009) – but it isn't the tube that is a risk factor, it is poor intubation that is risky.
- Laryngeal mask airways are an excellent option in cats. They are easy to place, do not damage the airway and come in a variety of sizes.
- Disconnect patients from breathing systems before repositioning them – especially cats. The twisting of the tube in the trachea as the patient is repositioned can cause tracheal damage.
- Don't use a rigid mouth gag for intubation (or for dentistry, or anything else) in cats. These mouth gags cause excessive opening of the mouth which can cause occlusion of the maxillary artery, which is the main source of blood supply to the retina and brain in cats (Martin-Flores M, et al. Vet J 2014;200:60-64). Occlusion of this artery secondary to mouth gag use has been linked to blindness and neurologic dysfunction, some of which led to euthanasia (Stiles J, et al. Vet J 2012: 2012;193(2): 367-73).

Cat facts for the maintenance phase of anesthesia

- Inhalants are commonly used. Injectable drugs are also commonly used in cats for short procedures. These are often administered IM since really small cat veins can make IV injection difficult.
- Analgesia
  - To decrease the dose-dependent impact on cardiovascular and respiratory function, keep the inhalant DOSE LOW! The best way to do that is to use analgesia.

Support and Monitoring Key Cat Points

- Support

Be careful with fluids!

- Administration of fluids was listed as a risk factor for anesthesia-related death (Brodbelt 2009). But it isn't the fluid – it is the amount of fluid - that kill cats.
- Calculate – and administer – the volume of fluids very carefully. Best to draw up the desired amount in a syringe vs trying to deliver a small volume from a fluid bag. Can also use rate-limiting tools like syringe pump or burette.
- Cats are very likely to be hypothermic – be aggressive with warming - & monitor body temperature
- Prevention is easier than rewarming. Temperature starts dropping AT INDUCTION
- Forced air blanket most effective
- Warm patient's environment - Surgery room, recovery cage, etc…
- Use warm fluids, warm scrub solution (and MINIMAL scrub solution), warm lavage solution, etc…
- **Minimize anesthesia time.**

Cat facts for the recovery phase of anesthesia Most unexpected anesthetic deaths occur in recovery (Brodbelt D, Vet J 2009;182:152-161). *(Table deleted due to too many tables)*

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TAILORING THE DIET TO THE PATIENT. WHAT TO FEED YOUR HEPATIC PATIENTS

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ABSTRACT BODY:

There are several disease processes that affect the liver that manifest in a variety of clinical signs and with different degrees of severity. Nutritional management of liver diseases is mainly a supportive treatment and varies depending on the aetiology and the clinical manifestation of the disease. A nutritional assessment (1) is essential to customize the feeding plan to each patient.

The goals of the nutritional management are as follows:

- Meet energy and nutrient requirements for the species and lifestage, and to maintain a stable body weight with ideal body condition score (BCS) and muscle condition score (MCS)
- Minimize hepatic damage (e.g. caused by copper accumulation or oxidation)
- Control metabolic derangements associated with some liver diseases (such as hepatic encephalopathy – HE – or urate urolithiasis).

Energy

Patients with these diseases can have increased energy requirements, due to increased losses (vomiting, diarrhoea, polyuria, hypermetabolic state). This, alone or together with the possibility of reduced food intake (anorexia, hyporexia), can predispose liver patients to malnutrition, which has negative impacts on prognostic. A negative energy balance can also increase protein catabolism and ammonia production, which can contribute to HE. However, liver patients can also be normal weight or even overweight.

Energy provision in thin animals or at risk of malnutrition is done using a highly digestible, highly caloric complete diet. In patients with reduced food intake, the use of feeding tubes might be required. High fat diets are useful to provide calories in a small volume, and is usually well tolerated unless the patient has a concurrent fat sensitive disease.

Protein

It is assumed that protein needs in liver patients are at least equal to healthy animals (2), but there is a lack of knowledge in the effects of disease on energy needs. However, some animals might have a lower tolerance to protein, such as patients with HE, usually associated with portosystemic shunts (PSS). The pathophysiology of HE is not completely known, and several hypotheses have been proposed, with a role attributed to bacterial nitrogen metabolites absorbed from the gut and blood ammonia, which cross the blood brain barrier and act upon the central nervous system. Other substances have been implicated, such as false neurotransmitters, mercaptans, and metals like manganese. HE can be intermittent, and some identified triggers are a high protein meal, gastrointestinal bleeding, and hypokalaemia.

In patients with HE, protein moderation might be beneficial to reduce ammonia and other nitrogen metabolites, but protein and amino acid needs must still be met. Otherwise, reduced regeneration can occur, plus endogenous muscle catabolism (which can aggravate HE). This protein must also be highly digestible and with an ideal amino acid profile.

Protein moderation might also be of interest if there is urate urolithiasis (which can be associated with PSS and other liver disorders), since purines (a urate precursor) are often abundant in protein sources.

Overall, protein moderation is only recommended if there is a need for it (HE or urate urolithiasis) and should not be attempted routinely in patients without these clinical manifestations, and, in all cases, always relative to current protein intake. It is not recommended in feline hepatic lipidosis unless HE is evident, since protein deficiency has been implicated in the pathogenesis of this disorder.

Protein source modification can also be attempted to help manage these signs without having to aggressively moderate the protein. Some protein sources like plant based, dairy or egg are lower in purines vs meat, fish, and organs. There is some evidence that plant based proteins might be better tolerated than animal ones in dogs with PSS (3). In all cases, a high digestibility is desired to minimize protein bacterial fermentation in the gut.

Fibre

High fibre diets with reduced calorie density are contraindicated in thin animals or those actively losing weight. High fibre diets can also reduce the diet's digestibility. However, moderate amounts of fermentable fibre might be of interest, by increasing nitrogen use by the gut bacteria (which will be excreted via faeces). Fibre that promotes bacteria that reduce lumen pH is also interesting, to reduce the uptake of intestinal ammonia into the body.

Other nutrients

Water soluble vitamin deficiencies can occur secondary to reduced food intake, decreased storage, decreased absorption, increased losses, and decreased synthesis (e.g., vitamin C). Fat soluble vitamins can also be affected in cases of fat malabsorption or dysbiosis, which can result in coagulopathies (vitamin K deficiency) and increased oxidative stress (vitamin E).

As for minerals, some liver diseases are associated with copper accumulation (primary or secondary), which contributes to liver damage. Dietary restriction can help with this issue (4). Zinc supplementation might also help. Sodium moderation is required in cases of ascites. Hypokalaemia is associated with HE, therefore, a complete diet that provides it is essential.

Antioxidant supplementation can also be considered. Some essential nutrients play a role in the antioxidant defence such as vitamins (C, E) and minerals (zinc, manganese, copper, selenium). Glutathione (synthesized from cystine, glutamate and glycine) is a very important antioxidant in this organ, and providing precursors is indicated in liver disease (such as SAMe). Some authors also suggest supplementing with L-carnitine and taurine in feline hepatic lipidosis (5).

Feeding plan

First, a complete nutritional assessment (1) is required in order to develop a feeding plan. A complete diet history is going to provide a lot of useful information, especially if the patient presents with HE or urate urolithiasis.

Diet choice: In cases without HE or urate urolithiasis or copper storage disease, a high quality, highly digestible diet (e.g., intestinal diets) adequate for the species and life stage can work well. Liver diets are not needed in those cases. Convalescence diets are a good choice for feline hepatic lipidosis, given their high energy density and usefulness in feeding tubes. Supplementation (e.g., with antioxidants, carnitine, taurine) can be considered.

If copper restriction is needed, a hepatic diet with low copper amounts is indicated. If protein moderation/modification is required (either due to HE or urate uroliths) then the current diet associated to these signs needs to be assessed to find another diet that will change the protein source +/- moderate the amounts. Liver diets can be considered (but are not always required). In all cases, all efforts should be made to choose a life-stage appropriate diet. Liver diets (in addition to being moderate in high quality protein and, in some cases, copper restricted) are also high in calories, moderate in sodium, and can have extra antioxidants and zinc. For cases with urate urolithiasis, liver diets (wet or with added water) can be used, and there are specific diets for this type of urolith that can also work. These diets also promote urine alkalinization.

Diet choice is also going to be affected by concomitant diseases (e.g., cats with triaditis).

Feeding amounts: Energy requirements need to be calculated according to species, life stage, BCS, and others (e.g., hospitalization vs outpatient). This can be done using formulas, label instructions, or through a complete diet history (the latter is better, given the high error of formulas). In all cases, amounts will be adjusted in +/- 10% intervals to achieve weight and BCS goals.

Feeding method: Multiple meals per day, even if portion controlled, can help reduce the metabolic load of both the gut and the liver. Free feeding can be considered in thin animals, with picky appetite.

Repeated nutritional assessments during follow up visits are important to adjust the plan as needed.

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RESPIRATORY DISEASES IN FISH

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ABSTRACT BODY:

Koi Herpes Virus Diseases

Koi herpesvirus disease (KHVD) is a worldwide viral disease of common carp (*Cyprinus carpio*) causing large-scale mortalities, up to 100%, especially in ornamental variants of common carp. KHVD is caused by cyprinid herpesvirus-3 (CyHV-3). Transmission of KHV is horizontal, but vertical transmission cannot be ruled out. Virus is shed via faeces, urine, gills and skin mucus. KHV outbreaks occur at temperatures over 16°C with optimal temperatures 23-25°C. The clinical signs usually appear in 3 days in optimal temperatures, but incubation period may be much longer in lower temperatures. Fish up to 1 year of age are more susceptible to the disease. Clinical signs include lethargic behaviour, erratic swimming, sunken eyes, pale gills and skin, gill necrosis and skin hemorrhages. Gross changes include necrosis, petechial haemorrhage and inflammation of gills, kidney and liver. Final diagnosis must be done by PCR detection of viral DNA or virus isolation and identification. There is no treatment for KHV. Fish that survive the disease may carry the virus and act as a source of infection for a very long time. Commercial live attenuated immersion vaccine is available (KoVax Ltd./Phibro Animal Health Corp.).

Carp edema virus disease/koi sleepy disease (CEVD/KSD)

The disease is caused by a poxvirus - Carp Edema Virus (CEV) and it is spread worldwide, often causing high morbidity and mortality in common carp and its ornamental varieties, mainly in juvenile stressed Koi carps. It is likely that virus shedding into the water occurs from gill and skin lesions of sick fish. Vertical transmission has not been confirmed. Optimal water temperature for development of clinical signs is between 15°C and 25°C, but outbreaks have been observed at much lower water temperatures. Decreased morbidity and mortality has been observed in temperature over 28°C. Sick fish typically lie motionless on the bottom of the pond and die within 2 weeks. Affected animals are lethargic, hanging just under the surface of the water, have sunken eyes (enophthalmos), erosive or hemorrhagic skin lesions with edema (especially in juvenile fish) and pale swollen gills. Definitive diagnosis is made by isolation of the virus or by PCR detection. There is no treatment for CEV but some measures may be applicable, such as 0.5% salt bath, which helps to prevent the disease. The salt does not treat infection, but decreases the amount of nitrates that can cause methemoglobinemia, which is a predisposing factor for infection. Immediate isolation and testing of sick fish showing suspect clinical signs is recommended, as well as removal of dead fish from ponds to minimize disease transmission. Freshly arrived koi carp should be quarantined at a temperature between 15°C and 25°C for at least 30 days and tested for the disease.

Bacterial gill diseases

Bacteria are a normal component of the aquatic environment and usually cause clinically apparent disease only in certain circumstances. Stress is the most important factor for establishment of bacterial infection, due to impairment of the immune system. The main stress factors are overcrowding, transport/handling, hypoxia, high ammonia and nitrates and inappropriate temperature for the fish species. Clinical signs are similar for many bacterial species and bacterial culture or molecular biology techniques are generally required to identify bacteria specifically. The most important bacteria in ornamental fish are Gram-negative bacteria from genera *Aeromonas*, *Flavobacterium*, *Citrobacter* and *Pseudomonas*. Gram-positive bacteria are less common and include mainly genera *Streptococcus*, *Lactococcus*, *Enterococcus*, and *Vagococcus*. Treatment includes correcting any water quality problems or removing stressors in the environment. Introduction of UV light and salt bath (0.5% salt bath) also often help to combat bacterial infections. Antibiotic treatment should always be based on culture and testing of sensitivity of the strain to specific antibiotics.

Fungal gill diseases

Mycotic disease in fish is often secondary, seen in immunosuppressed individuals often complicating bacterial and parasitic infections and also following trauma. Fungi belonging to the genus *Saprolegnia* or *Achlya* are the most commonly observed species. Microscopically, typical fungal hyphae are found. Treatment includes mechanical removal of the mycelia and medicated bath (formaldehyde, malachite green and slat. Gills can be primarily infected with Branchiomyces sanguinis or B. demigrans (gill rot) causing ischemic gill necrosis, typically in cyprinid fish.

Protozoan diseases

White spot disease (Ich)

White spot disease in probably the most common protozoan skin and gill disease in ornamental fish. It is caused by the ciliated protozoan parasite *Ichthyophthirius multifiliis. The parasite is highly infective and can cause severe mortality reaching 100%.* The disease presents as small white spots on the skin and changes in behaviour caused by irritation, such as flashing, gasping and lethargy. The infection may be limited only to gills. The diagnosis is made by clinical signs and microscopic examination of wet mounts of skin and gill scrapes, containing typical large, round to pear-shaped, slow motile ciliates with typical horseshoe nucleus and slow movement. Treatment should follow immediately after diagnosis and consists of moving the fish to a quarantine tank, increasing water temperature to 28-30°C and adding non-iodised salt 1-5g/l for 5 days. Some fish cannot tolerate high water temperature and in such cases the salt treatment should be prolonged to 7-8 days.

Oodiniosis

Oodiniosis - velvet disease is caused by the dinoflagellates *Piscinoodinium pillulare* and *P. limneticum* in freshwater fish and *Amyloodinium ocellatum* in marine fish. Trophonts are attached to the gill or epidermal epithelium by an attachment disc. *Piscinoodinium* has well-developed chloroplasts visible under the microscope. Clinical signs include a dense white dusting on the skin, dyspnoea, lethargy and death. Diagnosis is made by microscopic examination of skin and gill scrapes with typical appearance of parasites. The most effective treatment in freshwater is increasing water temperature to 25-28° C and prolonged salt bath (non-iodised salt 1-5g/l for 5 days). In marine species, the treatment of choice is copper. The copper, as a copper sulphate pentahydrate, is added to the system gradually over a period of several days until the free copper ion is at a concentration of 0.2 mg/l; this level is then maintained for up to 3 weeks. This treatment can kill all invertebrates and some fish species.

Other common gill protozoan parasites are caused by *Chilodonella, Trichodina, Costia* and *Brooklinella*.

Monogenean trematodes (flukes)

Monogenean trematodes are common skin and gill parasites with a direct life cycle in both marine and freshwater fish and can cause considerable damage to the host when present in higher numbers. The best known genera in freshwater fish are *Dactylogyrus* and *Gyrodactylus*, and in marine fish *Benedenia and Neobenedenia* (*Capsalidae*). They can be oviparous or viviparous and may complete their entire life cycle on a single host. In some viviparous species the cycle may be as short as 24 hours, if all environmental conditions are optimal. Clinical signs include lethargy, pale mucous membranes (anaemia), decreased appetite, flashing (rubbing against objects in the pond/aquarium), and excessive mucus production. Typical skin lesions are erythema, scale loss, white to grey irregular patches, erosions and ulceration. Gills are swollen and pale. Diagnosis is made by microscopic examination of wet mount/scrapes from gills and skin or by histopathology. Most freshwater monogeneans can be killed quickly with a 3 to 5 minute saltwater bath (0.3%). Another possibility is formalin bath 0.2 ml/l for 60 minutes every other day for 3 days. Praziquantel baths have also proved to be effective at dose 2–5 mg/l in a prolonged bath for 2–3 weeks.

Environmental diseases

Hypoxia

The amount of dissolved oxygen (DO) in water depends mainly on water temperature, but salinity and altitude also play a role. Minimal concentration of DO for the majority of fish species is 5.0 mg/l. Acute hypoxia is usually caused by failure of aeration or oxygen generators in aquaria or closed aquaculture systems, usually due to a power cut. Subacute hypoxia or suffocation is observed in ponds with large amounts of aquatic plants and/or algae, which are the major producers, albeit consumers of DO, and with no additional mechanical aeration. This event happens in early morning when the concentration of DO is lowest. Chronic hypoxia, due to long exposure to suboptimal concentration of DO, is observed in overcrowded tanks with low numbers of water plants. Clinical signs of acute hypoxia are crowding of fish near the water surface or near the water source, piping and tachypnoea. Fish that have died from asphyxia have wide open mouth and flared opercula. Chronic hypoxia causes anorexia, slow growth and reproduction, and higher susceptibility to infections. Definitive diagnosis is made by measuring the concentration of DO. Treatment of acute hypoxia includes intensive mechanical aeration and major water exchange if possible. Hydrogen peroxide can increase DO short term by spraying of 3% solution into water.

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RADIOLOGÍA DEL PASAJE NASAL

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RADIOLOGIA DEL PASAJE NASAL

La exploración radiográfica de la cabeza es extremadamente compleja. Sus características morfológicas varían mucho entre especies y en la especie canina, entre razas. (1) considerando ello, se deben tener presente que el pasaje nasal de las razas dolicocefálicas, mesocefálicas y braquicefálicas guardan diferencia la cual puede ser significativa. Por esto, resulta útil comparar con las imágenes obtenidas con las que se incluyen en los atlas de anatomía radiográfica normal. Cuando las lesiones afectan solo a un lado de la cabeza, la mitad contralateral también nos sirve como referencia de normalidad (1).

La interpretación radiográfica se puede realizar correctamente cuando la proyección radiográfica guarda los patrones de posicionamiento adecuado. Por eso, se recomienda realizar el estudio bajo anestesia general (1, 2, 3). Pequeñas cantidades de oblicuidad pueden llevar a una mala interpretación (3). Se debe realizar la intubación endotraqueal y si es necesario debe retirarse para evitar la superposición con las estructuras anatómicas en ciertas proyecciones. Se debe considerar la posición de la lengua, es necesario cambiarla de posición en las proyecciones ventrodorsal oblicua con boca abierta (1).

Las proyecciones radiográficas que se utilizan son: latero lateral, ventrodorsal con boca abierta y dorsoventral intraoral, para la observación exclusiva de la cavidad nasal intraoral dorsoventral y dorsoventral con la boca abierta; para la evaluación de los senos paranasales, la proyección indicada es la rostrocaudal, formando un ángulo de 90º con la columna (1, 2, 4, 5). Debe considerarse que las proyecciones ventrodorsal y dorso ventral comunes donde se incluyen el maxilar y mandíbulas aporta una imagen incompleta del pasaje nasal, sobreponiéndose el área craneal del pasaje nasal.

En un estudio realizado por Agut y *cols* (6), se determinó que los chasis flexibles con el sistema pantalla-película de mamografía es una buena alternativa al chasis convencional para la realización de las proyecciones intraorales para la evaluación de las fosas nasales en pequeños animales.

La cavidad nasal aparece dividida en dos mitades por el septo nasal compuesto por el hueso vómer y el cartílago nasal. El pasaje nasal se puede dividir a partir del tercer premolar hacia craneal los cornetes nasal y ventral, y hacia caudal los cornetes etmoidales. Se pueden observar, los cornetes nasal dorsal y ventral aparecen como finas líneas de densidad mineral en la parte rostral de la cavidad, los cornetes etmoidales o laberinto etmoidal se aparecen como espirales más gruesas también de densidad mineral en la zona caudal de la cavidad (1).

Según Coulson y Lewis (2) se puede encontrar una variación de la disposición del tabique nasal visualizándose radiográficamente con una desviación hacia en canal nasal izquierda y derecho causando una curvatura, pero ello guardando la línea de división intacta al igual que el hueso vómer; esto se puede presentar en perros y gatos.

La presencia de cuerpos extraños en la cavidad nasal, si estos son de densidad mineral o metal son fáciles de observar, en cambio, aquellos que tienen una densidad radiológica similar a la de los tejidos blandos y los líquidos (cuerpos extraños radiolúcidos), como las espigas, ramas de plantas, pequeños objetos de plásticos, etc.; no suelen visualizarse directamente en la imagen, aunque en muchos casos si se identifican los signos de rinitis focal asociada (1; 7).

Las fracturas que afectan al hueso nasal o a los senos frontales pueden ser difíciles de diagnosticar. Normalmente se produce depresión de los fragmentos, se acompañan de inflamación de los tejidos blandos adyacentes y, en muchos casos de enfisema subcutáneo por salida de aire a los tejidos adyacentes (1).

Las rinitis secundarias a infecciones bacterianas y rinitis linfoplasmocíticas suelen causar un aumento de la densidad radiográfica de la cavidad nasal, acompañado o no de un aumento de la densidad de los senos frontales, aunque se suele mantener una buena diferenciación de los cornetes en la imagen (1).

En procesos agudos pueden no aparecer alteraciones evidentes en la radiografia de la cavidad. Las rinitis crónicas hiperplásicas pueden producir junto con el aumento de la radiopacidad, una pérdida de diferenciación de los cornetes por engrosamiento de la mucosa y presencia de exudado entre ellos. Resulta más difícil evaluar la imagen de los cornetes en el gato ya que presentan un patrón más irregular que en el perro (1).

Las rinitis crónicas a menudo se caracterizan por áreas de incremento en la opacidad de tejidos blandos superpuestas a un patrón del cornete normal. Esta opacidad tiende a involucrar las porciones rostral y media de la cavidad nasal y frecuentemente es bilateral (4). En una rinitis crónica felina, ya que está puede causar una desorganización o destrucción de cornetes (1).

Las rinitis fúngicas, especialmente producidas por *Aspergillus* suelen presentar un patrón mixto en el que se combinan zonas de aumento de densidad con zonas osteolíticas, provocando una pérdida de diferenciación de los cornetes por destrucción de los mismos. En fases avanzadas, puede aparecer una desviación o erosión del hueso vómer, aunque no es muy frecuente. Se puede observar también un aumento de la densidad de los senos frontales, con o sin engrosamiento de la pared ósea (1).

La imagen radiográfica de los tumores nasales puede variar según el tipo de tumor y la antigüedad del mismo. Los tumores menos agresivos, así como los detectados en fases iniciales o tempranas presentan una imagen radiológica muy similar a la de la rinitis, caracterizada por un aumento localizado o difuso de la densidad radiográfica de la cavidad nasal, qué puede afectar uno a los dos senos frontales. Estos hallazgos suelen aparecer de forma unilateral en el caso de las neoplasias. Los tumores más agresivos, o los detectados en fases más avanzadas, suelen presentar un aspecto bastante destructivo, con pérdida del patrón característico de los cornetes nasales, desviación y/o destrucción del septo nasal y hueso vómer e invasión de otros huesos adyacentes (1).

En ocasiones, los tumores nasales se extienden hasta planos superficiales causando la aparición de una masa de tejido blando externa que produce deformación facial, lo cual es más típico de los tumores que de la rinitis. Las neoplasias nasales también pueden invadir la lámina cribiforme y penetrar así en la cavidad craneal, pudiendo afectar al encéfalo (1).

La destrucción del cornete nasal es indicativo de un proceso morboso muy agresivo y ocurre principalmente por rinitis erosivas o neoplásicas. Las rinitis destructiva esta ocasionada en general por infecciones fúngicas y afecta mas comúnmente a perros mesocefalicos o dolicofefalicos de menos de 4 años de edad (4). El linfoma nasal en gatos puede provocar un aumento de la densidad radiográfica de la cavidad nasal, acompaña o no de destrucción de hueso (1).

La radiografía tiene sus limitaciones, pero si se requiere evaluación más precisa, detallada, incluyéndose tejidos blandos son necesarias la tomografía, resonancia magnética y endoscopia, elegimos la tomografía para analizar con más precisión la afectación de las estructuras óseas, mientras que la resonancia sería más adecuada si el animal presenta signos neurológicos que hagan sospechar de una extensión secundaria del tumor hacia el encéfalo a través de la lámina cribosa (1).

Pueden estar destruidas grandes áreas del tabique nasal y esa destrucción puede ser evidente radiográficamente. Es más habitual que en estos animales ocurra reconocimiento de penetración septal con destrucción del vómer o destrucción o desviación del tabique nasal. Los signos periféricos de edematización de tejido blando, destruccion y formación de nuevo hueso periostico están usualmente asociados con neoplasias muy agresivas (4; 7).

La destrucción de los cornetes nasales y su sustitución por material de radio densidad de tejido blandos sugieren la presencia de un tumor, pero el diagnóstico definitivo requiere a realizar una biopsia (8). Los hallazgos en las imágenes deben ser confirmadas mediante histología otros medios. aunque son raras las neoplasias nasales benignas pueden ocurrir y son indistinguibles de las patologías malignas, independientemente de la técnica de diagnóstico por imagen empleada (7). Los pólipos nasales inflamatorios son indistinguibles clínica y radiográficamente de una enfermedad nasal maligna (7).

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DERMATOPHYTOSIS IN CATS: MORE THAN THE WOOD'S LAMP

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Dermatophytosis in dogs and cats.

Dermatophytosis is a dermatosis caused by superficial fungal infection normally affecting keratinized skin structures; stratum corneum, hair shafts, nails, generated by fungi from a zoophilic, geophilic or anthropophilic origin.

The most common dermatophyte species affecting dogs and cats are *Microsporum canis*, *Microsporum gypseum* and *Trichophyton mentagrophytes*. Due to the potential zoonotic risk and its infectious nature is an important disease in small animal medicine, potentiated by the close relationship and human animal bond existent in our present time.

Depending of the host immune competence, these could be self-limiting diseases within weeks or months. Immune response and underlying condition must be considered and evaluated particularly in patients not responding to conventional therapies.

There are more than 30 different dermatophyte species. Dermatophyte species in animals are isolated as asexual forms, called anamorphs which are identified as belonging to the genus *Microsporum* or *Trichophyton* on the basis of macroscopic and microscopic characteristics of the organism grown in culture.

Several studies have evaluated the normal composition of the cutaneous micobyota in different species. Healthy pet cats and dogs have shown that *M. canis*, the most common cause of dermatophytosis, is not part of the normal skin microbiome of dogs or cats. Cats have been identified as heathy carriers of *Microsporum canis*, harvesting the disease and the hability of transmission without presenting clinical signs in some cases.

Physicians often blame cats when diagnosing dermatophytes in humans, and advice owners to isolate them and in some cases to get rid of the cat claiming that the primary cause of the disease in human patients must be zoonotic transmission from the family cat.

Host	Cutaneous site	Micobiota
Human	Most cutaneous sites	*Malassezia*
	Feet	*Malassezia, Aspergillus, Cryptococcus, Rhodotorula* and *Epicoccum*
Dog	Mucous surface and mucocutaneous junctions	Environmental fungi Ascomycoyta, *Alternaria*, *Cladosporium* and *Epicoccum* less abundant Basidiomycota as *Cryptococcus* and *Malassezia*
Cat	Haired skin	Environmental fungi Ascomycota, *Cladosporium*, *Alternaria* and *Epicoccum*, less abundant Basidiomycota as *Cryptococcus*
	Mucous surfaces, oral cavity	

The pathogenesis of the disease occur through the infective form of the dermatophyte: the arthrospore, which is formed by fragmentation of fungal hyphae into very small infective spores. These can be transmitted by direct contact between an infected and uninfected animal or by fomite transmission, including grooming appliances, bedding, collars, ectoparasites and exposure to a contaminated environment.

Affectation of the skin barrier and microtrauma to the skin is an important factor in development of clinical signs. *Microsporum canis* infections are typically due to contact with an infected animal, mainly cats. Transmission from contaminated environments is not an efficient route of transmission. Most Trichophyton infections are suspected to be due to contact with infected rodents or their nests. *Microsporum gypseum*infections are less common and are presumed to be due to contact with contaminated soil as this is a geophilic organism. Increased microtrauma to the skin from pruritus and self- trauma, humidity and ectoparasites help to develop the clinical infection.

Secretion fo endoproteases and exoproteases digest keratin and degrade it, reducing its keratin cysteine disulphide bonds. The proteases secreted by dermatophytes may be host specific and result in inflammation and immune response, altering macrophage function and inhibiting lymphocite by cell wall mannans of the fungi, and decrease of the

keratinocyte turnover. Clinical cure and protection against re-infection depends on a strong cell-mediated immune response involving effector cells such as macrophages and neutrophils, and cytokines such as interferon gamma.

CLINICAL SIGNS

The clinical presentation of the disease is related to the affectation of the keratinized structures. The most common clinical signs include hair loss, papules, scales, crusts, erythema, follicular plugging, hyperpigmentation and changes in nail growth. Lesions usually are asymmetrical with variable pruritus, but in general is minimal to absent. If pruritus is present, areas of self-trauma or eosinophilic lesions in cat may be present and mimic the current condition.

Variations in clinical presentation may reflect the susceptibility of host immune response. Multifocal and diffuse lesions are most commonly seen in animals with concurrent skin or systemic disease and/or physiological stress. Dogs and cats may develop nodular lesions due to dermatophytes infections and this could be diagnosed cytologically or thought histopathology. These lesions may be present as kerion, pseudomycetoma and mycetoma. Normally these lesions have nodular, dome shaped appearance, may be exudative. Cytologically and histologically present granuloma and pyogranulomatous reactions with hair shaft present in some cases and fungal spores and/or hyphae. The nodules may ulcerate and drain purulent to serous material with tissue grains.

DIAGNOSIS OF DERMATOPHYTOSIS

Despite the fact there are several techniques to detect and diagnose dermatophytes, no test is considered a gold standard and each of them may be helpful in different clinical scenarios.

WOOD´S LAMP EXAMINATION

An invention of Robert William Wood in 1903, produces an ultraviolet light generated from a filter consisting in a high-pressure mercury arch filtered through a component made of barium silicate with a 9% of nickel oxide. Wood lamp filter is selective for wavelengths between 320nm and 420nm with a peak on 365nm. When filtered light is absorbed by tissue, usually in visible specter, it is emitted by the fluorescence process. Normal skin has a blue fluorescence with examination with Wood lamp, while some dermatosis may create specific fluorescence patterns.

In the case of dermatophytes, the characteristic green fluorescence observed on *M. canis* infected hair shafts is due to a water-soluble chemical metabolite (pteridine) located within the cortex or medulla of the hair. This fluorescence is due to a chemical interaction that occurs as a result of the infection and is not associated with spores or infective material.

Wood lamp fluorescence is often confusing for some clinicians, “green apple” fluorescence must be found on the hair shaft. Topical medications, ointments may create false fluorescence. Scale fluorescence may exist with a white color and create confusion for the inexperienced clinician. Crust may be removed in search for infected hairs that create fluorescence. Fluorescing hairs are most likely to be found in untreated infections; fluorescence may be difficult to find in treated cats. False positive and false negative results are most commonly due to inadequate equipment, lack of magnification, patient compliance, poor technique or lack of training.

DERMATOPHYTE CULTURE

Although dermatophyte culture media (DTM), a modification of the Sabouraud agar may be considered a gold standard, it is important to know that false positive may appear depending of the sample region and technique. Collecting fluorescing hairs may increase the success, toothbrush collecting technique (Mackenzie technique) may be helpful in collecting large amount of infecting material.

BIOPSY

Histopathology is a useful diagnostic technique and it is recommended in nodular and elevated lesions. Results are usually received in some cases before the DTM shows growth.

PCR

PCR detection of dermatophyte DNA can be helpful; however, a positive PCR does not necessarily indicate active infection, dead fungal organisms from a successfully treated infection will still be detected on PCR, as will noninfected fomite carriers. Negative PCR in a treated cat is compatible with cure. Negative fungal culture from a cat with no lesions and a negative Wood’s lamp (except for glowing tips) is compatible with cure.

ENVIRONMENTAL DISINFECTION

Cleaning and disinfection of carpets, bedding, clothes must be addressed to reduce the infecting material in the environment. Diluted sodium hypochlorite is effective in removal of infective material.

TOPICAL THERAPY

Topical therapy is a complement and helps reducing the infective material in the patient. Formulations containing enilconazole, chlorhexidine plus miconazol and lyme sulfur have shown efficacy.

SYSTEMIC THERAPY

Systemic therapy is indicated in most cases. Different antifungal treatments are used in dogs and cats. Itraconazole (5-10mg/kg) (noncompounded) and terbinafine are the most effective and safe treatments for dermatophytosis. Griseofulvin (microsized 25mg/kg, ultramicrosized 5 -10mg/kg) is effective but also has more potential adverse effects compared to itraconazole and terbinafine (10-30mg/kg). Ketoconazole (5-10mg/kg dogs) and fluconazole (10mg/kg) are less effective treatment options and ketoconazole has more potential for adverse effects specially in cats. Lufenuron has no in vitro efficacy against dermatophytes, does not prevent or alter the course of the disease and it is not recommended. Antifungal vaccines do not protect against challenge exposure but may be a useful adjunct therapy.

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EARLY SPAY-NEUTER: THE PRO'S AND THE CON'S

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ABSTRACT BODY:

EARLY AGE SPAY-NEUTER: THE CONS

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Introduction:

Early age sterilization is frequently recommended by high quality high volume spay-neuter (HQHVSN) clinics for population control in dogs. Unfortunately, recent studies have indicated an association between loss of gonadal hormones and future development of many deleterious health conditions. Included in this list are metabolic, behavioral, orthopedic, neoplastic, endocrine, immunologic, neurologic and urologic disorders (Table 1, to be discussed in **bold**). The purpose of this lecture is to review several of these conditions and provide relative risk where available.

Table 1: Conditions reported with increased frequency after gonadectomy in dogs

- Metabolic issues
  - **Obesity**
- Urologic disease
  - **Urethral sphincter mechanism incompetence (USMI)**
  - Recessed vulva
- **Behavioral issues**
- Neoplasia
  - **Osteosarcoma**
  - **Hemangiosarcoma**
  - Brain tumors
  - Transitional cell carcinoma
  - Mast cell tumor
  - Lymphosarcoma
  - Prostate carcinoma
- Orthopedic disorders
  - **Cranial cruciate ligament injury (CCL)**
  - Canine hip dysplasia (CHD)
- Endocrine disorders
  - Hypothyroidism
  - Diabetes mellitus
  - Cushing's disease
- Immune disorders
  - Atopy
  - Autoimmune hemolytic anemia (AIHA)
  - Immune-mediated thrombocytopenia (ITP)
  - Inflammatory bowel disease (IBD)
- Neurologic disease
  - Intervertebral disk disease
  - Cognitive dysfunction

Obesity

Obesity is by far the most common disease in small animal practice, and gonadectomy approximately doubles the risk in both males and females, unrelated to age[1,2]. In a recent Banfield report 51% of dogs ≥2 years of age were overweight, less than 10% of owners were successful in having their pet lose 10% BW and 40% of those that did lose weight regained it within 12 months[3].

Urethral sphincter mechanism incompetence (USMI)

There is conflicting evidence regarding relative risk of developing USMI after early versus standard age sterilization, but overall risk is less than 1% for intact bitches compared to 5-20% after gonadectomy[4]. USMI can occur any time after surgery and larger dogs (>20kg) are more likely to develop the problem. Certain breeds such as the Rottweiler, Boxer, Old English

Sheepdog and Doberman Pinscher are more frequently affected, with 65% of Boxers reported to develop USMI after gonadectomy in one study[4]. While both medical and surgical treatments are available, outcomes are often discouraging.

Behavioral issues

Contrary to popular belief, dogs are not better behaved after gonadectomy. Gonadectomy is associated with decreased roaming, mounting and marking in some male dogs but overall dogs are more sensitive to handling, demonstrate excessive barking, beg for food more often and are more fearful of strangers and other dogs[5,6]. Effects on aggression are variable. Of note is that aggressive behavior is generally not "learned", so gonadectomy once the behavior is demonstrated has the same likelihood of decreasing it as early gonadectomy would have prevented it[7].

Neoplasia

Recent studies indicating an association between gonadectomy and increased risk of several cancers, especially in certain breeds, is likely the biggest issue bringing the question of routine early sterilization of all dogs to the forefront. Relative risk for appendicular osteosarcoma is 1.3-2.0 in gonadectomized dogs, and 3.1-3.8 in gonadectomized Rottweilers[8]. Mean survival with amputation alone is about 6 months, or 13 months with amputation and chemotherapy. Relative risk for hemangiosarcoma is about 2.5 after gonadectomy, with mean survival time of 2 months for ruptured splenic hemangiosarcoma[9,10]. Additional tumors reported with increased frequency after gonadectomy include brain tumors, lymphosarcoma, mast cell tumor, prostatic carcinoma, and transitional cell carcinoma[11,12]. Mammary tumors are less likely to occur after gonadectomy in female dogs, especially if performed early[13]. Other than benign perianal adenoma, there are no non-reproductive tumors reported with decreased frequency after gonadectomy in male dogs.

Cranial cruciate ligament disease

Cranial cruciate injury is by far the most common orthopedic injury in dogs. Gonadectomy doubles the risk of CCL injury regardless of age of sterilization in both males and females, and for all breeds and sizes of dogs[14].

Summary:

The decision to spay or neuter a dog should be made on an individual basis, taking into consideration the pet's breed, intended use, household environment and temperament. Owner input is important in the decision-making process, and the veterinarian's role is to explain both the positive and negative consequences of gonadectomy. Early age sterilization, often performed before ownership has been established may be appropriate in some, but not all situations.

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EARLY SPAY-NEUTER: THE PRO'S AND THE CON'S

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ABSTRACT BODY:

EARLY AGE SPAY-NEUTER: THE PROS

Early spay-neuter can be considered as neutering performed before the traditional neutering age, which is typically around 6 months of age and sometimes older in certain dog breeds. In some places, animals are neutered as early as 6 weeks of age.

The main benefit of performing early neutering is for animal welfare organisations to overcome pet overpopulation. Being able to sterilise animals at a younger age means that accidental pregnancies can be avoided, as many animals are able to reproduce before the traditional neutering age. At the same time, young kittens and puppies are highly sought-after for adoption. Being able to sterilise these animals prior to rehoming can help organisations and shelters relief the burden of either keeping these animals until they can be neutered or to spend resources on following up to ensure adopted animals return for neutering when they are older.

Early age neutering is generally easier to perform due to easier identification of the ovaries and uterus, better visualisation, less abdominal fat, and more accurate haemostasis of the blood vessels[1]. Animals can be neutered faster, through a smaller incision, as well as reduced use of suture materials for closure. All these also translates to lower post operative complications such as infections or suture breakdown as compared to those neutered at a later age[1].

Younger animals may react to anaesthetics differently from older animals. They can be more sensitive to certain drugs, have prolonged effects, and limited capacity for cardiovascular compensations[2]. However, safe anaesthetic drugs are widely available and mortality rates are comparable with neutering of older animals[2]. The procedure can be considered low risk as long as precautions are taken to ensure animals are well hydrated and steps are taken to prevent hypothermia and hypoglycaemia. These young animals also recover faster from anaesthesia compared to those neutered at a traditional age.

There are some concerns regarding early spay-neuter, and some of these myths have been debunked by evidence. Below are a few of the major concerns regarding early spay-neuter:

Stunted growth

Contrary to the concern, early neutering delays the physeal closure and causes slight lengthening of the long bones. However, these does not cause any visible differences in size of the animals[3]. In cats, there are no evidence that the lengthening causes any clinical adverse consequences[2,4].

Increased obesity

Although neutering at any age can decrease the metabolic rate, there are no difference between those neutered early or at a traditional age[3]. Obesity is generally manageable through managing the diet and levels of activity of pets.

Increased FLUTD in male cats

Feline lower urinary tract disease (FLUTD) in male cats has been a major concern because of the difficulty in the extrusion of the penis. However, neutering does not cause a reduction in urethral diameter and there are no evidence of an increase in the cases of FLUTD in cats neutered early[2,4,5].

Early age neutering can also bring about health and behavioural benefits to cats and dogs. One of the major health benefits is the significant decrease in mammary tumours for both cats and dogs[3]. In terms of behavioural benefits there are less sexual behaviours, urine marking and aggression in cats[4]. For dogs, there are reports of decreased separation anxiety, escaping behaviours, inappropriate elimination when frightened, and relinquishment to shelters[6].

There are some growing concerns in dogs regarding development of joint disorders and certain types of cancers, particularly for certain large sized purebred dogs. However, these risks are not seen in smaller breeds[7]. Early studies show that these concerns might not affect mixed breed dogs as much. Occurrence of lymphoma, mast cell tumour, haemangiosarcoma, and osteosarcoma of neutered mixed breed dogs were comparable to intact dogs. However, there were increased in joint disorders of mixed breed dogs weighting 20kg or more[7].

In summary, there are a lot of benefits to early age neutering of cats and very few cons, most of which are manageable such as obesity. Many vets around the world are increasingly being encouraged to perform early neutering for cats, not just for shelter or rescue animals, but for all pet cats. However, a case-by-case risk-benefit analysis should be considered before neutering large-sized dogs.

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ENDOCRINOPATÍAS EN URGENCIAS: ADDISON Y DKA

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ABSTRACT BODY:

ENDOCRINOPATIAS EN URGENCIAS: ADDISON Y DKA

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El Hipoadrenocorticismo o enfermedad de Addison cursa con disminución de los niveles circulantes de gluco y/o mineralocorticoides y puede ser primario (atrofia adrenal bilateral) o secundario (falta de produccion de ACTH) en su origen.

Los animales con Addison en la sala de emergencias se pueden presentan en "**crisis addisoniana",** la forma más severa del hipoadrenocorticismo primario.

Los **signos clínicos** son shock hipovolémico grave, pero con bradicardia severa y arritmias. La falta de aldosterona produce una intensa natriuresis y la consecuente pérdida de agua produciéndose una severa reducción en el volumen circulante y una disminución de la tasa de filtración glomerular. Esto producirá una azotemia pre renal, una retención de potasio, fosforo e hidrogeniones que darán lugar a la acidosis metabólica. El animal puede presentarse en anuria u oliguria. Las pérdidas en forma de vómitos y diarreas contribuyen aún más a la deshidratación. También puede observarse que la hipotensión no responda totalmente al tratamiento con fluidos por la deficiencia de glucocorticoides.

Muchas veces es difícil diferenciar la crisis addisoniana del fallo renal agudo en el momento de la admisión del paciente al hospital.

En las **analíticas**, podemos encontrar hemoconcentración, acidemia, azotemia, hiponatremia, hipocloremia e hiperkalemia (con una ratio Na/K <27). Una Ratio < 27 tiene una sensibilidad del 89% y una especificidad del 97% en el diagnostico de Addison anemia moderada no regenerativa con neutropenia, eosinofilia y linfocitosis; hipercalcemia; hipoglicemia (aunque la falta de hipoglicemia no excluye el Addison).

En el **ECG** encontramos cambios relacionados con la hiperkalemia (bradicardia, parada atrial, ondas T picudas, QRS anchos, ondas R reducidas y bloqueo AV).

El test de estimulación con ACTH es la prueba diagnóstica del hipoadrenocorticismo que en el caso de la crisis addisoniana, realizaremos justo después de la resucitación del paciente.

Un buen test de screening en perros con sospecha de Addison es la medición del **cortisol basal** · Valores de cortisol plasmático > 2 mg/dl descartan Addison con buena fiabilidad.

El **tratamiento** requiere fluidoterapia agresiva con una solución cristaloide equilibrada (ringer lactato, plasmalyte, isolyte, normosol). Este tipo de fluidos consigue corregir la acidosis y al tener una concentración menor de sodio en su composición (130-140 mEq/L), evita rápidos incrementos en la concentración de sodio del paciente que podrían derivar en alteraciones neurológicas (síndrome de desmielinización osmótica). Se recomienda una corrección máxima de la hipernatremia de 10-12mmol/L/d (o 0,5 mmol/L/h).

La frecuencia de infusión será de **20-90 ml/Kg/h** durante la primera hora, administrándose en bolos de 15-20ml/kg en 15 min hasta alcanzar los objetivos finales de la resucitación (mejora estado mental, pulso, frecuencia cardiaca y presión arterial sistémica). Con los fluidos compensamos las pérdidas de agua y la deshidratación, la hiperkalemia e hiponatremia, la acidemia y la hipercalcemia. También aumentamos la filtración glomerular y la producción de orina (idealmente 1-2 ml/kg/h).

La fluidoterapia suele ser suficiente para normalizar la hiperkalemia leve y acidosis.

Corrección hiperkalemia severa (hiperkalemia (K>10 mEq/L), con bradicardia inferior a 40 ppm) (ver proceeding obstruccion uretral)

Tenemos que monitorizar electrolitos y la glicemia y administrar bolos o CRI de dextrosa si es necesario.

Tratamiento hormonal:

Usaremos dexametasona hasta realizar el test ACTH. El uso de la hidrocortisona ha demostrado reducir el tiempo de corrección de la hiperkalemia y la estancia hospitalaria. La suplementación con mineralocorticoides debe iniciarse en cuanto sea posible (oral o subcutánea).

Recientes estudios han cuestionado los intervalos de dosificación y las dosis de la DOCP. Parece que la duración del efecto es superior a un mes y las dosis bajas (1.1 mg/kg) son suficientes para controlar signos clínicos y evitan sobredosificación. Se recomienda ajustar el tratamiento a cada paciente en base a

monitorización de signos clínicos y electrolitos.

Dosis fisiológicas de glucocorticoides serán necesarias de por vida en pacientes no tratados con fludrocortisona.

Diabetes cetoacidotica: DKA

La DKA es la presentación más severa y con mayor riesgo para la vida del paciente diabético. El 50 % de los gatos y hasta el 70 % de los perros con DKA son nuevos diabéticos.

En la patofisiología de la DKA intervienen la hiperglicemia propia de todo diabético debida a la deficiencia insulínica, la acidosis metabólica y en la mayor parte de las ocasiones, una enfermedad concurrente que exacerba y complica los signos (p.ej pancreatitis, infecciones urinarias, colangiohepatitis, etc).

Sin insulina la célula empezara a usar otros sustratos en el metabolismo celular como los triglicéridos, llevando a la acumulación de cuerpos cetónicos y cetoacidosis. La glucosuria llevará al paciente a la perdida de agua por diuresis osmótica. El aumento de cuerpos cetónicos circulantes y la enfermedad concurrente pueden también ser causa de vómitos y deshidratación y la situación de acidosis metabólica se ira agravando. Los mecanismos de homeostasis de glucosa mediante las hormonas del stress (adrenalina, noradrenalina, cortisol y glucagón) estimularan la glucogenólisis, gluconeogénesis y lipolisis para intentar corregir la situación.

Cuando la acidosis metabólica es severa, el K+ empezará a salir de la célula intercambiándose por H+ en un intento de restablecer la neutralidad. Esto disminuirá la reserva intracelular de K y deberemos considerarlo en el momento del tratamiento. Aunque el paciente no se presente inicialmente hipokalémico, deberemos suplementar los fluidos con KCl. La acidosis puede afectar la función cardiaca (reducción GC, arritmias, hipotensión).

Los pacientes en DKA suelen presentarse con vómitos, deshidratados, dolor abdominal y con mayor o menor inestabilidad cardiovascular.

La **analítica** mínima en estos pacientes incluirá hemograma, bioquímica, uroanálisis con cultivo de orina. Radiografías de tórax y ecografía abdominal complementaran el examen y descartaran otras patologías concurrentes (pancreatitis, colangiohepatitis y neoplasia entre otras). A nivel laboratorial presentaran hemoconcentración, hiperglicemia/ glucosuria, cetonemia/ cetonuria, desordenes electrolíticos (suplementación con K siempre indicada. Aunque inicialmente esté en niveles normales, el K circulante disminuirá al empezar el tratamiento con insulina; monitorear el fósforo. Puede disminuir también y llevar al paciente a hemolisis

El tratamiento del paciente en DKA requiere:

- Rehidratación +/- resucitación
- Corrección de la acidosis
- Administración de insulina (reduccion cetosis)
- Mejorar el estado general del paciente (antieméticos, antiácidos, antibióticos si se confirma infección urinaria, analgésicos)

Fluidoterapia:

Se recomineda empezar rehidratando al paciente por al menos 6 h antes de empezar la terapia con insulina. (Aunque un estudio no acabo de demostrar la utilidad de esta practica). Esto mejorará el volumen circulante, los niveles de K, glucosa y la acidosis metabólica. Los fluidos de elección serán isotónicos, equilibrados con solución tampón (RL, normosol, plasmalyte, etc). Plantear la rehidratación en 24 h para evitar cambios drásticos en la osmolaridad y electrolitos.

Suplementar siempre con potasio, aunque no esté bajo inicialmente

Si se observa hipofosfatemia, suplementar al 50% en KCl y KPO4

Insulina:

En los estados iniciales de tratamiento elegiremos una insulina regular (acción rápida y fácil de absorber). El método de administración dependerá de nuestras preferencias, disponibilidad de personal para monitorizar al paciente, servicios veterinarios (hospital 24h). En la actualidad disponemos de sistemas de monitorización de glucosa intersticial que permiten evaluar constantemente los niveles de glucosa. Aunque la correlación entre esta glucosa y la plasmática no es perfecta, esta práctica nos permite evaluar la tendencia de la glucosa con mínima interacción/ stress, reduciendo el número de venipunciones.

La pauta ideal que suprime más eficientemente la formación de cuerpos cetónicos es la insulina iv en CRI. Este método requiere intensa monitorización de los niveles de glucosa (cada 1-2 h). Los pacientes suelen permanecer en CRI de insulina hasta que se resuelven los trastornos metabólicos, recuperan el apetito y se puede iniciar en la insulina de larga duración.

En el gato además se han descrito otras pautas utilizando insulina de larga duración, más sencillas con excelentes resultados que pueden reducir los costes para el propietario;

Tratamientos complementarios:

La terapia con antieméticos, gastroprotectores, analgésicos y antibióticos si se confirma infección

urinaria u otra fuente de infección será necesaria.

Aproximadamente el 70 % de los pacientes en DKA sobrevive y consigue pasar a una situación de diabetes estable. Generalmente requieren un tratamiento intensivo en UCI no siempre al alcance de los propietarios. El pronóstico depende del tipo de patología concurrente y de la severidad de la DKA.

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MIELOPATÍAS AGUDAS

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ABSTRACT BODY:

Las mielopatías son frecuentes en la clínica diaria de pequeños animales. Éstas pueden resultar un reto para el veterinario, ya que se han descrito más de 40 mielopatías diferentes, muchas de ellas con una presentación clínica similar. Frente estos casos, es imprescindible realizar un buen diagnóstico diferencial, que nos ayudará a poder elegir e interpretar de manera objetiva las pruebas diagnósticas más óptimas en cada caso.

Para ello, es muy importante usar el "razonamiento clínico". La información que obtendremos con la anamnesis y la reseña del paciente, la localización neurológica de la lesión, así como del inicio y la progresión del problema, es imprescindible para poder ordenar, de más a menos probable, las posibles enfermedades causantes del motivo de consulta de nuestro paciente.

La **reseña** incluye, principalmente, la especie, raza, edad y sexo del paciente, ya que estas características predisponen a sufrir ciertas enfermedades.

La **localización** de la lesión (determinar la parte del sistema nervioso afectada que me justifica los signos clínicos del paciente), así como la **simetría o asimetría** de los signos clínicos y determinar la **presencia o no de hiperestesia espinal**, lo obtendremos realizando un completo examen neurológico.

El **inicio y el curso/ progresión** del problema, lo valoraremos acordándonos de la palabra VITAMIND. Esta regla nemotécnica sirve para valorar las diferentes categorías de enfermedades que pueden causar problemas neurológicos, ya que cada una de las letras de esta palabra corresponden a una entidad clínica determinada (vascular, inflamatorio-infeccioso, traumático, anomalía congénita, metabólico, idiopático, neoplásico, degenerativo) , que se comportan diferente en cuánto a inicio (agudo o crónico) y progresión (progresivo o no progresivo). .

Cuando tengamos un paciente con una **mielopatía de inicio agudo**, nuestros principales diagnósticos diferenciales serán los siguientes (el orden de los mismos dependerá de la historia clínica, la reseña, la localización y simetría de los signos clínicos y la presencia de dolor o no):

MIELOPATÍAS AGUDAS MÁS FRECUENTES:

TRAUMATISMOS- FRACTURAS/ LUXACIONES ESPINALES

Son lesiones de la columna vertebral por acción de fuerzas externas, siendo los más frecuentes los accidentes de tráfico y las caídas. A pesar de que pueden afectar a toda la columna vertebral, las zonas más frecuentemente afectadas son la zona lumbosacra, la zona toraco-lumbar y la zona atlantoaxial, debido a su mayor rango de movimiento.

Los animales con fracturas o luxaciones vertebrales son pacientes politraumatizados, y antes de valorar el daño neurológico, es imprescindible estabilizarlos y detectar otros posibles procesos concomitantes como neumotórax, hemoabdomen, uroabdomen, otras fracturas... Una vez estables, es importante valorar el daño neurológico de manera muy cuidadosa pero completa, con la mínima manipulación del paciente para no producir más daño iatrogénico. Los signos clínicos dependerán de la localización y de la severidad de la lesión, siempre con un inicio hiperagudo. El diagnóstico concreto de las lesiones se basa en los signos clínicos y el resultado de las pruebas de imagen, básicamente radiografías simples, a pesar que con el TAC y la RM podemos conseguir un diagnóstico más detallado de las estructuras óseas y los nervios respectivamente. Es importante valorar toda la columna, ya que el 40 % de los pacientes tienen más de una lesión vertebral y es imprescindible relacionar los hallazgos radiológicos con los signos clínicos, ya que se ha descrito un 22-28 % de falsos negativos con las radiografías simples para fracturas y luxaciones.

El tratamiento médico o quirúrgico dependerá del tipo de lesión, de los signos clínicos del paciente, de las condiciones del propietario y, en los casos quirúrgicos, de las preferencias del cirujano.

MIELOPATIA VASCULAR

Las mielopatías vasculares son aquellas alteraciones medulares que se producen debido al compromiso vascular de una parte determinada de la médula espinal (M.E). Son secundarias a una disminución o ausencia de flujo sanguíneo focal, debido a la oclusión o a la rotura de un vaso sanguíneo espinal o vertebral. Como consecuencia, se produce una isquemia medular del territorio irrigado por el vaso afectado. Esto provoca cambios metabólicos y bioquímicos que pueden acabar con daño neuronal y gliosis, principalmente afectando la sustancia gris debido a su superior demanda metabólica.

Como característica clínica, las mielopatías vasculares

tienen un inicio hiperagudo (<6h) o agudo (7-24h), y una evolución no progresiva, a pesar que se ha descrito que puede haber un empeoramiento de los signos clínicos durante las primeras horas debido a la inflamación o daño medular secundario. Los signos clínicos son asimétricos por lo general y no provocan dolor. La neurolocalización será focal y dependerá del segmento medular afectado en cada caso, y el grado de disfunción neurológica dependerá de la extensión de la lesión.

Dentro de las mielopatías vasculares isquémicas primarias, se destacan dos diferentes procesos:

- Embolismo fibrocartilaginoso
- Enfermedad tromboembólica

Mielopatía isquémica por embolismo fibrocartilaginoso (EFC):

Es la causa más frecuente de mielopatía isquémica en perros. Está causado por la oclusión de un vaso sanguíneo espinal o vertebral por material fibrocartilaginoso. El material embolizado es fibrocartílago, con las mismas características histológicas que el núcleo pulposo de los discos IV. Existen varias teorías referentes a como llega el material dentro de los vasos afectados.

Se presenta con más frecuencia en perros de razas grande y gigantes, con una edad media de 5-6 años, aunque se ha descrito también en razas pequeñas y desde los 2 meses a los 13 años de edad. La presentación es la típica de una mielopatía vascular. En gran parte de los afectados el cuadro ocurre durante el ejercicio. La localización más frecuente es a nivel de intumescencia lumbosacra.

El diagnóstico se consigue con pruebas de imagen (RM). El tratamiento se basa en reducir el daño secundario de la médula espinal, manteniendo una buena perfusión medular, así como fisioterapia temprana. Se han descrito varios factores pronósticos como la presencia de sensibilidad, la localización, la simetría, el tamaño del paciente y el resultado de las pruebas de imagen.

Enfermedad tromboembólica

A pesar de que el EFC es la causa más frecuente de MI, también existen émbolos bacterianos, parasitarios, neoplásicos o de grasa así como condiciones médicas asociadas (cardiomiopatías, hipotiroidismo, hiperadrenocorticismo, fallo renal crónico e hipertensión), que pueden causar la oclusión de un vaso. La presentación clínica y los hallazgos en la RM son idénticos a los casos de EFC.

En la especie felina es frecuente la oclusión de la **arteria espinal ventral**, especialmente en gatos adultos- viejos y con condiciones médicas concurrentes. Los signos clínicos son hiperagudos, no progresivos y simétricos. En los gatos, la arteria espinal ventral es más estrecha a nivel de C2 (o C3), por lo que está predispuesta a una oclusión embólica. Tienen predisposición los gatos de más de 12 años y con condiciones médicas asociadas como fallo renal crónico, cardiomiopatía hipertrófica e hipertensión. Neurológicamente, se corresponde con una mielopatía C1C5. Los signos neurológicos suelen ir acompañados de ventroflexión cervical. El diagnóstico requiere imágenes de RM. El pronóstico es favorable, con un tiempo medio de recuperación de 5.7 días para la ambulación. Hay recurrencia en el 37 % de los casos.

PROCESOS DEGENERATIVOS: ENFERMEDAD DEL DISCO IV – EXTRUSIONES DISCALES

Se ha descrito que la enfermedad del disco intervertebral (IV) es la causa más frecuente de mielopatía tanto en perros como en gatos.. Están compuestos externamente por un anillo fibroso (fibras de colágeno), el cual rodea un área gelatinosa, conocida como el núcleo pulposo (NP) y compuesto principalmente por agua. Estos componentes se degeneran de manera fisiológica con la edad. Existen 2 diferentes tipos de degeneraciones discales: metaplasia condroide (típica de razas condrodistróficas y edades jóvenes) y metaplasia fibroide (más frecuente en animales no condrodistroficos y adultos).

A pesar de que por definición las hernias discales se encuentran dentro de la entidad de “procesos degenerativos” por la fisiopatología causante, y éstos tienen, en general, una evolución crónica progresiva, hay ciertos tipos de hernias discales (la mayoría) que difieren por su presentación clínica, teniendo un inicio agudo.

Actualmente se describen 7 diferentes tipos de hernias discales: Hernia discal Hansen I, Hernia discal Hansen II, extrusión aguda no compresiva del NP (antes mal llamada Hansen III), Extrusión aguda del NP hidratado, extrusión aguda del NP intradural / intramedular, extrusión aguda del núcleo pulposos con hemorragia epidural extensa asociada y extrusión de disco IV traumática.

Pueden ocurrir en cualquier raza y edad, a pesar que la Hansen I tiene predisposición en razas como teckel (en la que se ha demostrado un componente genético que produce una degeneración precoz del disco), bulldog francés y pequinés en edades jóvenes (2-6años). Exceptuando la tipo Hansen II, el resto de las hernias discales tienen una presentación aguda o hiperaguda y asimétrica. Las que suelen producir más dolor son la extrusión discal Hansen I y la extrusión aguda con hemorragia epidural extensa asociada.

El diagnóstico de las hernias discales requiere pruebas

de imagen avanzadas (Mielo-TAC, TAC o RM). El TAC y el mielo TAC pueden ser poco sensibles en los casos en los que el disco no está degenerado o no hay compresión.

El tratamiento depende del tipo de hernia discal, de los signos clínicos del paciente y de las condiciones del propietario.

SARCOPENIA AND CACHEXIA IN DOGS AND CATS

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ABSTRACT BODY:

Cachexia

Cachexia is muscle loss secondary to chronic inflammation or disease1. The Society on Sarcopenia, Cachexia and Wasting Disorders defines it as lean body mass (LBM) loss, although fat loss may occur simultaneously2. In humans, three of five criteria are required: decreased muscle strength, fatigue, anorexia, low fat-free mass index, and increased inflammation markers2. Decreased muscle function is associated with frailty, morbidity and mortality. Frailty scores exist for humans, have been developed for dogs3, and are in development for cats.

Cachexia is often seen in pets with chronic diseases, e.g. cancer, chronic kidney disease (CKD), congestive heart failure (CHF), or diabetes mellitus. Cachexic pets often show weakness abd anorexia, and have a poor quality of life, which can contribute to an owner's decision to euthanize the pet.

Causes of cachexia are multifactorial and include inflammatory mediators (e.g TNF-α), oxidative stress, upregulation of the ubiquitin-proteasome proteolytic pathway, and hormonal mediators. Increased inflammatory and counter-regulatory mediators decrease metabolic adaptations for fat utilization. Protein is then used as a primary energy source causing rapid and often profound LBM catabolization and muscle loss4. In cats, which use more protein than carbohydrates for energy, this may be evenmore pronounced.

Diagnosing cachexia early can be challenging as LMB loss may precede significant weight loss. The six-minute walk test, i.e. distance walked in six minutes, for dogs to assess stamina5 is less useful in cats; however, muscle condition scores should be performed routinely and ultrasound techniques have been developed to assess LMB6,7.

Sarcopenia

Sarcopenia, loss of LBM and muscle function with ageing, occurs without overt disease. In humans, decreases in maximal strength are three times greater than muscle mass loss.8.

Older Labrador retrievers showed a decrease in epaxial muscle area compared to younger dogs, with no difference in inflammatory mediators7. Cats over 12 years old are likely to lose fat and lean tissue. By 15 years, cats have a mean LBM < 2 kg; 1/3 less than the mean 3 kg during their adult lifestage. Speed of decline is a predictor of time of death9,10.

The mechanism of sarcopenia is also multifactorial and involves decreased physical inactivity (disuse atrophy), increased cytokines ("inflammaging"), decreased growth hormone, IGF-I, and testosterone, changes in type II muscle fibres, insulin resistance, decreased stem cell/progenitor cell function (cellular ageing), decreased mitochondrial function, oxidative stress, and decreased protein synthesis4,11. Aged mammals have decreased muscle regenerative capacity, partly caused by reduced number and function of muscle satellite cells, the progenitor cells involved in muscle repair and maintenance11. Declining muscle tissue IGF1 and anabolic cytokines reduce synthesis of myofibrillar (e.g. myosin heavy chain) and mitochondrial proteins 4,11.

Sarcopenia, cachexia, and malnutrition are thus the main causes of muscle wasting. As chronic diseases are common in older pets, cachexia and sarcopenia are often concurrent. An increase in fat mass and sarcopenia can occur simultaneously so weight may not be greatly altered. Like cachexia, sarcopenia has clinical implications and an association with increased morbidity and mortality.

Muscle mass is thus reduced physical activity, central nervous system ageing, loss of motor units, myocyte changes, and altered hormonal, cytokine and oxidative status. An inadequate diet contributes to muscle loss.

Treatment of Sarcopenia and Cachexia

Exercise Is Vital For Muscle Condition

Exercise may be walks or other exercise for dogs; for cats this may involve interactive play. For both species treatment of arthritis is recommended for activity and well being.

Dysfunctions in Satiety Hormone Regulation and Fatty Acid Oxidation

Appetite is controlled by orexigenic (stimulating eating), and satiety factors (e.g. adiponectin, serotonin, insulin and proopiomelanocortin), which may be increased in cachexia4. During disease, disturbances of hypothalamic pathways controlling energy homeostasis and appetite occur. The 'brain-muscle axis' coordinated by the hypothalamus mediates onset of anorexia, the balance between anabolism and catabolism, and tissue

wasting by centrally influencing energy homeostasis. In particular, the hypothalamic melanocortin system does not respond appropriately to energy depletion; its activity is diverted toward promoting catabolism (i.e., reduced energy intake, increased energy expenditure, increased muscle proteolysis, and fat loss). Hypothalamic proinflammatory cytokines and serotonin are keys in triggering hypothalamic resistance11,12.

Fatty acid metabolism within hypothalamic neurons affects food intake and energy metabolism in a leptin-independent way. Inhibition of fatty acid synthase blocks upregulation of orexigenic neuropeptides and down-regulation of anorexigenic neuropeptides, thereby decreasing food intake. Proinflammatory cytokines, particularly TNFα and IL-1, inhibit fatty acid oxidation13.

Stimulating appetite and calorie intake

Decreased calorie intake is common in older pets and may be due to disease. Healthy older people also have decreased taste perception which may occur in dogs and cats. Dental disease or arthritis may make eating or accessing food uncomfortable, so feeding management should be addressed to ensure easy, comfortable food access.

Some pets, especially cats, will eat if a variety of foods are fed, e.g. rotating around different foods. Flavour enhancing pastes or "soups" or small amounts of palatable foods may encourage intake, though should comprise < 10% of calories. Fish oil supplements may increase palatability and are beneficial in modulating inflammatory cytokines. Flax seed or other plant oils are ineffective omega-3 fatty acids for dogs and cats. Diets vary greatly in calorie density (kcal/g). EU regulations do not require calorie content on the label but calorie content can be roughly estimated from label macronutrient information.

Mirtazapine has appetite stimulating and anti-emetic/anti-nausea effects with good efficacy for increasing cats' appetites.14. The half-life is longer in older (over 10 yr) cats and those with CKD14.

Ghrelin, an endogenous hormone increased during fasting, stimulates appetite, has anti-inflammatory effects and decreases muscle atrophy by inducing muscle differentiation and fusion15. While ghrelin itself has a short half-life, receptor agonists are being developed. The veterinary drug capromorelin is a selective ghrelin receptor agonist and appetite stimulant although effects on muscle tissue have not been investigated.

Protein

Sufficient and probably increased amounts of highly digestible protein should be fed to many older cats.

Many cats have decreased protein and fat digestibility with age, especially those over 12 years9,16. About 20% of cats over 14 yr have low protein digestibility (<77%) and low fat digestibility2,9,16,. Management in these cases is similar as for cats with histologically confirmed IBD: diet (highly digestible, novel antigen or hydrolysed diets), prebiotic or probiotics, correction of low serum cobalamin or folate concentrations, vitamin E supplementation, and possibly antibiotic treatment or immunomodulation15.

Dogs appear to have less decrease in nutrient digestibility with age, although may be present in individual dogs17.

Older humans show anabolic resistance, i.e. more protein is required for muscle protein synthesis; therefore, recommended daily protein is increased. The mechanism responsible for this is not fully understood. The gut microbiota are implicated in many postulated mechanisms, either directly or indirectly. Gut microbiota change with age and are influenced by dietary protein so might modulate individual responses. Research also implies a role for the gut microbiome in skeletal muscle function18.

Possible Future Treatment

Leucine and β-hydroxy-β-methylbutyrate (HMB)

Leucine, a branched chain amino acid, may help preserve LBM. Amino acids are potent modulators of protein turnover, and muscle cells are highly sensitive to their availability. Amino acid abundance increases activity of mTORC1, driving protein synthesis, and leucine is the most potent stimulator of mTORC1. Leucine supplementation is popular in humans for anabolic effects and potential for overcoming the anabolic resistance of aging19.

Although leucine enhances acute protein synthesis in healthy exercising humans, supplementation may not enhance protein synthesis during muscle wasting. Some studies in humans show no beneficial effects on muscle mass or function20. A leucine metabolite, β-hydroxy-β-methylbutyrate (HMB), has positive effects on LBM and strength when combined with exercise, including in older humans21,22.

Other Ideas

A few other ideas in human and rodent research into muscle loss in the older populations include: myostatin antagonism, selective androgen receptor molecules, ursolic acid, β-agonists, anti-oxidants, anti-inflammatory drugs, and vitamin D23. Supplemental carnitine may aid fatty acid metabolism. The breadth of the research indicates that no one intervention has overwhelming success.

Summary

Earlier diagnosis of muscle mass loss will allow for opportunities for better interventions. A good diet history will provide information about calorie and protein intake. Consistently monitoring body weight, body condition and muscle condition scoring will help address changes. Small decreases in weight are important, especially if it is a trend. Ultrasound techniques may provide more quantitative assessment of muscle condition. Determination of frailty scores may also be useful.

Addressing appetite, feeding management, and changing to a higher calorie, more digestible and possibly higher protein diet may be needed. In some cases, a hydrolysed or novel protein diet can be useful. Supplementing with omega 3 fatty acids is often beneficial; other supplements to consider include cobalamin, folate, vitamin E as an anti-oxidant, (possibly vitamin D in some cases) prebiotics and probiotics, carnitine, and amino acid supplements. Supplementing with leucine and other branched chain amino acids may show benefits, although more research is needed. Watch for new treatments, although ensure that safety and efficacy studies have been done for dogs and cats.

References available on request

023 / #36

MYCOBACTERIAL INFECTION IN FISH

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ABSTRACT BODY:

Mycobacteriosis in fish is a chronic progressive bacterial disease, very frequent in tropical fish, occasionally seen in cold water fish. Many species of mycobacterium have been isolated from the lesions, but main pathogens are *Mycobacterium marinum*, *M. gordonae* and *M. fortuitum*. Myocbacterial infection often causes a systemic disease with granulomatous type of inflammation in many organs and tissues, mainly in kidney, liver, skin and mesenteric adipose tissue and also in gills. Emaciation, anorexia, skin ulcerations and lethargy are the main clinical signs, occasionally with grossly visible granulomas in affected organs and tissues. Microscopic examination of compression fresh tissue preparations and histopathology show multiple granulomas in the infected tissues. Special stain Ziehl-Neelsen reveals myriad of acid-fast rods. Definitive diagnosis is made by culture in specific media (Lowenstein-Jensen) and/or PCR. Treatment is very difficult and is generally not attempted in practice; sick fish should be euthanized. Mycobacterium has a unique cell wall containing long chain 3-hydroxy mycolic acid causing great resistance to disinfectants and requires long contact times for most disinfectants to be effective; 5% phenol, 1% sodium hypochlorite, iodine solutions, glutaraldehyde and formaldehyde are usually effective. Fish mycobacteriosis has zoonotic potential, mainly in immunocompromised humans, and ranges from focal dermal fish tank granuloma, most often on fingers, to severe systemic illness leading to death.

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CUANDO LO COMÚN NO ES SINÓNIMO DE FÁCIL! SHUNT PORTO SISTÉMICO EXTRAHEPÁTICO (CASOS)

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Generalidades

Las derivaciones portosistémicas son anomalías vasculares que permiten la comunicación entre el sistema porta y la circulación sistémica, eludiendo el pasaje a través del hígado. Se clasifican en intra o extrahepáticas, múltiples o simples, congénitas o adquiridas. Los signos clínicos que las caracterizan son los de la encefalopatía hepática; debilidad, ataxia, presión de la cabeza contra objetos, desorientación, marcha en círculos, cambios conductuales, amaurosis, convulsiones y coma. Aunque se sabe que el amoníaco tiene un papel principal en la patogénesis de la misma, nuevos estudios confirman la participación de otros factores como la inflamación y el manganeso. En las derivaciones portosistémicas también se presentan signos gastrointestinales o urinarios, especialmente cristaluria de biurato de amonio.

Las derivaciones portosistémicas o anomalías vasculares portosistémicas congénitas en perros consisten en vasos anómalos que permiten que la sangre proveniente del estómago, intestinos, páncreas y bazo pase directamente a la circulación sistémica elu- diendo el hígado (Greenhalgh et al., 2010), lo cual resulta en un inadecuado desarrollo he- pático, disminución del metabolismo y producción de proteínas, reducción en la elimi- nación de toxinas y medicamentos, y posible falla hepática (Karen y Berent, 2018). Los sistemas afectados son el sistema nervioso central, gastrointestinal y urinario. Los síntomas neurológicos suelen ser inespecíficos, y pueden incluir ceguera, ataxia, convulsiones, disminución de la propiocepción y presión de la cabeza, signos que, se relacionan con el consumo de alimentos en cerca de la mitad de los pacientes (Weisse y Berent, 2017).

También denominada fístula arteriovenosa hepática. Destaca por la presencia de múlti- ples comunicaciones (malformaciones) que se unen en una única comunicación (fístula). Una rama de la arteria hepática, a través de numerosos vasos, comunica directamente con la vena porta generando un sistema de alta presión. Esto provoca el desarrollo de shunts extrahepáticos secundarios a un aumento de la presión portal.7

Anatomía de la vasculatura hepática y el drenaje venoso abdominal

La vena porta provee hasta el 80% del flujo de la sangre y el 50% del contenido del oxígeno del hígado; el resto es aportado por la arteria hepática. Las tributarias de la vena porta, desde caudal a craneal, incluyen los vasos mesentéricos que forman la vena mesentérica craneal, la vena mesentérica caudal, la vena esplénica y la vena gastroduodenal. La primera drena el intestino delgado; la segunda el colon y la porción proximal del recto; la tercera el bazo y estómago; y la última mencionada el páncreas, duodeno y estómago. Los conductos biliares y las ramas de la arteria hepática suelen localizarse sobre la superficie ventral de la vena porta, aunque algunas ramas arteriales pueden ubicarse por dorsal (Slatter, 2006).

Ecografía

La ecografía se ha convertido en la herramienta de diagnóstico de elección para la visualización de las DPS (Fossum et al., 2009). Es un medio no invasivo, rápido que puede efectuarse sin sedación o anestesia pero que depende mucho de la experiencia del operador (Ettinger y Feldman, 2002

Recientemente, el uso de la inyección transesplénica de solución salina agitada y sangre heparinizada ha sido evaluado para ayudar en el diagnóstico ecográfico de DPS en perros. Las microburbujas se siguen ecográficamente a través del sistema del portal, éstas no pueden atravesar la barrera sinusoidal del hígado en animales normales; por lo tanto, la presencia de microburbujas en la circulación sistémica es el criterio utilizado para diagnosticar DPS. El transductor del ultrasonido se posiciona para seguir las microburbujas a través del sistema del portal. La solución salina agitada mezclada con 1 ml de sangre heparinizada se inyecta en el bazo. Esta técnica es capaz de diferenciar entre DPS intrahepática y extrahepática. También es capaz de diferenciar entre derivaciones portoácigos y portocava y es útil para el monitoreo posoperatorio luego de la atenuación de la derivación (Thieman Mankin, 2015).

Tratamiento

Las derivaciones portosistémicas congénitas pueden ser tratadas médica o quirúrgicamente. El objetivo del tratamiento quirúrgico consiste en ocluir el flujo sanguíneo de la derivación, redirigiendo la sangre a través de la vasculatura portal disponible. La finalidad de la terapia médica o conservadora es disminuir el transporte de los factores absorbidos desde el tracto gastrointestinal a la circulación sistémica. Tanto el tratamiento médico como el quirúrgico desempeñan un papel en el manejo de perros con DPS y, a menudo, se usan en combinación (Thieman Mankin, 2015). La tasa de supervivencia a largo plazo en animales afectados clínicamente y sometidos a cirugía fue significativamente mayor que en aquellos sometidos a la terapia conservadora. Además, hay evidencia de que la cantidad y la frecuencia de los signos clínicos luego del tratamiento son más bajos en los pacientes tratados quirúrgicamente, lo que sugiere que pueden tener también, una mejor calidad de vida (Greenhalgh, 2014).

Tratamiento Medico

Se recomienda el empleo de antibióticos para reducir la flora entérica que produce la mayor parte de las toxinas (amoníaco), que se creen que son responsables de la encefalopatía hepática. Frecuentemente, se utiliza neomicina oral (10-15 mg/kg,

2 a 3 veces por día). Metronidazol (7.5 mg/kg, VO, 3 veces al día) o ampicilina (22 mg/kg, IM o SC, 3 a 4 veces por día) también reducen las concentraciones intestinales de amoníaco. La lactulosa es un disacárido sintético que acidifica el contenido colónico y capta iones de amonio del lumen (Fossum et al., 2009).

Cuando la depresión marcada del sistema nervioso central o el coma impiden la administración oral de lactulosa y neomicina, estos fármacos son administrados mediante enema (Ettinger y Feldman, 2002). El diazepam suele ser ineficaz en perros con encefalopatía que presentan convulsiones, el fenobarbital tiende a ser más eficaz y a algunos animales se les puede anestesiar con propofol (Fossum et al., 2009). Tambien se recomienda alimento balanceado para problemas hepáticos.

Aparatos de oclusión

Constrictores ameroides

Debido a que la oclusión abrupta y la ligadura parcial han sido asociadas a complicaciones posquirúrgicas importantes agudas y crónicas, se han desarrollado dispositivos (tales como los constrictores ameroides) para la oclusión, completa gradual del vaso de derivación. Un constrictor ameroide tiene un anillo interno de caseína que está rodeado por una vaina de acero inoxidable. La caseína es una sustancia higroscópica que se hincha a medida que absorbe agua corporal. La vaina de acero inoxidable fuerza a la caseína a hincharse hacia adentro, comprimiendo el vaso anómalo. El constrictor ameroide nunca provoca un cierre completo pero la reacción tisular fibrosa que éste estimula producirá la oclusión del vaso en unas 4 a 5 semanas. El cierre es más rápido durante los primeros 3 a 14 días después de la colocación; posteriormente, la velocidad de cierre disminuye. El tiempo para ocluir el vaso depende del tamaño del mismo y del constrictor, y de la rigidez del anillo externo (Slatter, 2006).

Bandas de celofán

Se han utilizado bandas de celofán para proveer una oclusión gradual de las derivaciones portosistémicas congénitas. La banda está formada por el plegamiento (en 3 veces) de una banda de 1,2 cm de ancho de celofán esterilizada con óxido de etileno. El extremo es hecho en punta para facilitar su pasaje, y la banda es pasada alrededor del vaso anómalo. Los hemoclips pueden deslizarse a lo largo del celofán para ajustar la comprensión; una vez determinada la colocación final, el celofán es suturado para evitar que se afloje. La fibrosis estimulada por el celofán provoca, en la mayoría de los perros, la oclusión del vaso anómalo en unas 8 semanas de colocada la banda. Cuando el diámetro interno de la banda es mayor a 3 mm, es poco probable que se produzca una oclusión completa del vaso de derivación (Slatter, 2006).

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025 / #173

PRACTICAL FELINE DERMATOLOGY WITH INTERACTIVE CASES

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ABSTRACT BODY:

The aim of the consultation process should be to obtain a full history, undertake a full physical examination, and consider what further actions or investigations may be required in conjunction with the owner, while ensuring the cat remains as stress-free as possible. Irrespective of the suspected cause of the skin disease, a full history and full clinical examination should never be overlooked as there may be concurrent disease present and/or a systemic cause of the skin condition. . Even with the best environment and approach, some cats will remain very anxious and a full physical examination may not always be possible at the first attempt.

The whole process should be performed gently and quickly, in order not to stress the patient.

In exceptional cases, some cats are so fearful that a full examination is not achievable even with the most patient of handling. This is rare, but heavy restraint (scruffing the cat and pinning it to the table) will only make the experience much worse for the cat. In such cases, consider using chemical restraint to facilitate the examination.

The main question to be answered when we have a feline patient with alopecia is to differentiate among self-inflicted or spontaneous alopecia. If we are able to answer this our differential diagnosis is reduced and we can start our practical approach.

Cats are considered “hidden scratchers”, their behavioral characteristics made difficult for the owner to identify pruritus or self-inflicted wound in the majority of cases. Approach to the owner may be cautious due to the fact, most owners will not believe their cat is scratching. In these cases, the clinical evaluation will be basic; clinician may use tools like trichograms, cytology, skin scraping, dermatophyte tools for diagnosis and dermatoscopy.

A practical tip to identify self-inflicted alopecia is to look closely and if possible, with magnification the alopecic area in the skin. In most self-inflicted alopecia a remain of hair shaft will be visible and in most of the “alopecic” areas remaining of trimmed hair is present. Most cats even if there is a severity of licking won’t be able to eliminate the base of the hair showing a “superficial shaving” appearance. This evaluation may be easier if we hold the affected skin between our fingers applying a mild pressure like folding the skin when clinical observation is performed.

Trichogram is helpful in identifying self-traumatic alopecia, when we will observe broken hair shafts or tips or in some cases fungal spores or ectoparasites attached to the hair shaft. This diagnostic tool is one of the main we need to use in our initial approach.

If we suspect of dermatophytes, Wood´s lamp examination, dermatophyte test medium or cytology may be useful in ruling out or confirming our diagnosis

If our initial clinical approach demonstrates a spontaneous alopecia, additional test may be required like CBC, blood chemistry, endocrine panel and diagnostic imaging. Differential diagnosis of spontaneous alopecia may include: hyperadrenocorticism, hyperthyroidism, thymoma, paraneoplastic syndrome, follicular dysplasia

Papules, pustules, furuncles, abscesses and crusts are common lesions in cats. With the exception of abscesses, they are often observed in combinations, repre- senting different stages of the same disease evolving into one another. In general, these lesions are the expression of inflammatory diseases, with infectious, para- sitic, allergic or autoimmune pathogenesis. Clinical presentations of papules, pustules, furuncles, abscesses and crusts and their preferential localization in selected feline diseases are described, together with useful diagnostic hints com- ing from signalment and history. A feline-specific clinical presentation called miliary dermatitis is characterized by multiple, small crusted papules and pruri- tus. The diagnostic approach to papules, pustules, furuncles, abscesses and crusts requires performing the diagnostic tests in a systematic way.

Complete blood count, biochemistry, and urinalysis are useful when a meta- bolic disease such as xanthomas or calcinosis cutis due to renal failure is sus- pected, based on the results of histopathological examination. If results of cytology and/or histopathology suggest a diagnosis of leishmaniosis, serology should also be performed. FIV and FeLV serology should be also carried out, especially in cats affected by infectious diseases.

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026 / #40

ADVANCES IN METHODS OF SURGICAL STERILIZATION IN DOGS AND CATS

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ABSTRACT BODY:

ADVANCES IN METHODS OF SURGICAL STERILIZATION IN DOGS AND CATS

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The purpose of this lecture is to introduce refinements and advances in methods of surgical sterilization in dogs and cats.

Scrotal castration in dogs

While historically avoided, orchiectomy using a scrotal incision is gaining increased popularity, especially in high-quality-high-volume-spay-neuter (HQHVSN) clinics. Scrotal orchiectomy has been associated with less self-inflicted post-surgical trauma and decreased surgery times, especially in small (0.9-11.4kg) dogs[1,2]. Other than location of the incision and closure, the surgical procedure is the same as with the pre-scrotal approach (2). Deep tissues are either not closed or apposed with 1-2 simple interrupted absorbable sutures. The skin is either closed with skin glue or allowed to heal by second intention.

Suture-less castration in dogs

Small dogs can be castrated safely using the auto-ligation method described for male cats. An additional suture-less method for open castration is to transect the vas deferens near the testicle, then tie the vas to the spermatic vessels with several square knots.

Auto-ligation of the ovarian pedicle in cats

Auto-ligation of the ovarian pedicle is quick, safe and avoids the necessity of using suture [3]. This method is easier in larger cats, and surgeons learning the procedure should avoid kittens until comfortable with larger patients[4].To perform auto-ligation the ovarian pedicle is fanned out and the suspensory ligament cut. A window is made in the mesovarium and a single overhand throw is started in the pedicle with a needle driver. After placing the first half of the throw, the jaw of the needle driver is clamped. Finally, the pedicle is transected between the knot and ovary, and the overhand throw completed by pushing the knot over the tip of the needle driver.

Surgical knots with inherent knot security on the first throw

Traditionally, the first throw on a knot providing vascular occlusion has been either a square knot or a surgeons throw. Square knots are acceptable if the tissue is thin, but if the tissue is thick, the knot may loosen before the second throw can be placed. A surgeons throw helps decrease slippage of the first throw but loosens somewhat when tensioning. Utilizing a knot that has more inherent resistance to slippage of the first throw (Miller's, constrictor, and strangle knots) is preferable, especially for inexperienced surgeons[5].

Methods of surgical hemostasis

Recent advances in methods of surgical hemostasis improve the safety, efficacy and speed of vessel ligation during surgical procedures. Vascular clips, self-locking ties and bipolar electrosurgical forceps are available in multiple sizes and configurations, and provide a quick and secure method of ligation. Vessel sealing devices are available that use electrothermal energy and pressure to fuse collagen within vessel walls, resulting in vascular occlusion[6]. Vessel sealing devices create minimal thermal spread and can occlude vessels measuring up to 7mm in diameter.

Analgesic techniques

Liposome encapsulated local anesthetics provide excellent analgesia for 72 hours without significant systemic side effects.

Laparoscopic ovariectomy

Laparoscopic surgery is less painful, provides improved visualization and safety and is being requested by owners with increasing frequency[7,8]. To perform laparoscopic ovariectomy the urinary bladder is first emptied by manual expression or catheterization. Dogs are positioned in dorsal recumbency and secured to an operating table that allows for medial to lateral rotation. The first port placed is for the telescope and generally located 2-4 cm caudal to the umbilicus in an average 40kg dog. Abdominal insufflation with carbon dioxide to a pressure of 12-14 mm Hg provides optimum visualization while avoiding adverse physiologic effects. An instrument port is then placed 2-4 cm cranial to the umbilicus under visual guidance. The patient is rotated 45 degrees lateral and the ovary on the up side located. Once identified the proper ligament is grasped through the instrument port, held against the body wall and a large swaged-on needle with #2 suture material passed through the abdominal wall, ovarian pedicle

and then back though the abdominal wall to "skewer" and stabilize the ovary. In this position both the ovarian pedicle and soft tissue attachments between the uterine horn and ovary can be electrocoagulated, clipped, or sealed with a vessel sealing device. Once free, the ovary is retrieved through the instrument port. The patient is then repositioned into the opposite lateral recumbency and the procedure repeated on the opposite side. Port sites are closed routinely.

Hysterectomy (Ovary-Sparing Spay)

The surgical incision for hysterectomy extends from just caudal to the umbilicus to the pubic brim. Identification and exposure of both the uterus and ovaries are the same as for ovariohysterectomy. Once the first ovary is identified it is elevated with a hemostat placed on the proper ligament and a window made in the mesosalpinx at the junction with the uterine horn. Several methods of ligation can now be used with success, but whatever method is utilized, it is critical that the entire uterine horn be removed. In one method, 3 clamps are placed across the mesosalpinx and two ligatures are placed. The first ligature lies between the ovary and most cranial clamp and the second in the crush of the most cranial clamp. The mesosalpinx is then sharply transected between the two remaining clamps[9].

Debate exists regarding removal of the contralateral ovary. Removal has the advantage of potentially decreasing the risk of subsequent ovarian cancer, but gonadal hormone levels may not be the same as when both ovaries are left in situ. For this reason, maintaining both ovaries is recommended.

It is critical with a hysterectomy procedure that the entire uterus be removed as persistent gonadal hormones will predispose to the development of cystic endometrial hyperplasia complex/pyometra. Many methods for ligation and transection at the level of the cervix or cranial vagina can be used successfully, but regardless of technique the uterine arteries should be individually transfixed. This location is very caudal in the abdomen and exposure can be difficult. A surgical assistant is helpful for retraction when performing this part of the procedure.

In one common technique, 2 clamps are placed on the cranial vagina/caudal cervix. Both uterine arteries are individually transfixed caudal to the clamps, and then a third circumferential ligature placed caudal to these. The vagina/cervix is then transected between the two clamps. Some surgeons prefer to place all ligatures without clamps, and then cut cranial to the ties, leaving at least 3 mm between the sutures and cut surface.

Vasectomy

Vasectomy in male dogs is a simple, quick and atraumatic procedure easily performed by general practitioners[10]. In most dogs, the spermatic cords can be palpated as they extend from the scrotum toward the inguinal rings. A 3-5cm pre-scrotal midline incision is made slightly cranial to the site for routine pre-scrotal orchiectomy. Careful dissection in a lateral direction exposes the spermatic cord. If localization is difficult, it is useful to manipulate the testicle caudally and cranially to tense and relax the cord. Once the spermatic cord is identified it is isolated by circumferential passage of a hemostat. A small incision is then made in the parietal vaginal tunic with a scalpel blade, tenotomy scissor or performed bluntly with fine hemostats. Extreme care should be taken to avoid any disruption of the pampiniform plexus. The vas deferens is identified as a turgid white structure. Circumferential ligatures are placed 1-2 cm apart and a short section of vas deferens excised between them. Closure of the vaginal tunic can be performed with fine (4.0-5.0) absorbable suture material although the necessity of this step is unclear. Closure is the same as pre-scrotal castration.

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EL PACIENTE EN INSUFICIENCIA RESPIRATORIA: ¿ORIGEN CARDIACO O PULMONAR?

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ABSTRACT BODY:

Distrés respiratorio: ¿origen cardiaco o pulmonar?

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El oxígeno es la fuente de la vida, necesario para infinidad de funciones.

Los animales que se presentan en la sala de urgencias con insuficiencia respiratoria plantean un desafío para los veterinarios. Es una situación estresante para paciente y veterinario en la que la vida del paciente puede correr peligro y que requiere rápida intervención.

El veterinario debe mantener la calma en todo momento y adaptarse a la tolerancia del paciente en cuanto a la realización de diagnósticos. No siempre podemos ni debemos realizar pruebas complementarias para llegar al diagnóstico hasta que el paciente se encuentre estabilizado.

La primera pregunta que nos surge es ¿Por qué está respirando así este animal? En ocasiones la reseña (razas braquiocefálicas), historia (mordedura de cable eléctrico, inhalación de humo, historia de colapso traqueal, bronchitis) o examen físico básico (soplo cardiaco de alto grado, silbidos, crepitaciones, patrón respiratorio) nos pueden orientar en el diagnóstico.

El origen de problema puede localizarse en la vía aérea (patología respiratoria de vía alta o baja como parálisis laríngea, colapso traqueal, obstrucción vía aérea, neumonía, bronquitis, asma) o a nivel cardiaco (insuficiencia cardiaca congestiva).

Es muy importante prestar atención al patrón respiratorio del paciente (disnea inspiratoria, espiratoria, mixta, frecuencia y esfuerzo respiratorio).

Todos estos pacientes, sea cual sea el origen de problema, se van a beneficiar del uso de sedación suave para mejorar la calidad de la respiración y de la suplementación de oxígeno tan pronto como llegan a la sala de emergencias. El uso de butorfanol a dosis de 0,1-0.4 mg/kg es segura y la adición de acepromacina a dosis bajas (0,01-0,02 mg/kg) en braquiocefálicos debe ser considerada. Esa debe ser nuestra primera actuación en pacientes en distrés, pero aun estables.

Si SpO2 < 93-95% el paciente está en hipoxemia y deberemos suministrar oxígeno. El objetivo será aumentar la difusión de oxígeno a nivel tisular. La tolerancia del paciente a la suplementación es mayor si el aire está humidificado.

Métodos de suministrar oxigeno	**FiO2 conseguida**
Sonda de oxígeno alrededor de la cara	25-40% si flujo es 2-3 L/min Poco eficiente
Máscara facial	50-60% si la máscara esta ajustada y flujo 8-10 L/min. Controlar la liberación de CO2 para evitar Re-inhalación
Campana de oxígeno (con collar isabelino o caja metacrilato)	30-40 % una vez la campana se ha oxigenado y con un flujo de 1-2 L/min
Jaula de oxigeno	40-60%. Riesgo de hipertermia. Alto consumo de oxigeno
Gafas nasales	25-40% a un flujo 2-3 L/min
Cánula nasal/nasofaríngea	30-70 % a flujos de 50-150 ml/kg/min. Flujos mayores serán incomodos para el paciente

Algunos se presentan en peor condición (cianóticos, con esfuerzo respiratorio severo, en riesgo de parada respiratoria) y será necesario tomar la decisión de intubar para salvar la vida del paciente y asistir en la ventilación hasta que tengamos más clara la causa del problema. Mientras el paciente se estabiliza, podremos conversar con su propietario para obtener más información y procederemos a realizar más pruebas cuando el paciente lo permita.

Debemos realizar un examen físico completo incluyendo auscultación pulmonar y torácica para localizar el problema (vía alta, baja, inspiratorio/espiratorio), medir el grado de oxigenación y función pulmonar (pulsioxímetro o gasometría arterial)

Las radiografías muchas veces no pueden ni deben realizarse inmediatamente por el temor a empeorar

la situación por el estrés añadido durante el procedimiento.

Actualmente el uso del ecógrafo como herramienta de examen complementario rápido en urgencias es de gran utilidad. Las técnicas de **POCUS** ("point of care ultrasound"), **TFAST, AFAST, VetBLUE** son utilizadas a diario en la sala de emergencias y no solo en pacientes traumatizados. No deben sustituir a la auscultación, pero proporcionan información valiosísima para el diagnóstico.

No requiere de sedación, puede realizarse durante el proceso de estabilización con mínimo estrés para el paciente (en decúbito esternal, sin afeitar el pelo) y nos permite evaluar la presencia de líquido en cavidad torácica/parénquima pulmonar, tamaño del corazón y cámaras cardiacas, medir ratio LA/Ao, presencia de derrame pericárdico en pocos minutos y sin estrés.

Se habla de pulmón húmedo (edema pulmonar principalmente) si se observan líneas B también llamados "cohetes pulmonares" que son artefactos similares a rayos láser que parten de la superficie pleural y ocupan todo el campo de exploración ecográfica y se mueven sincrónicamente con la respiración. Su presencia indica fluido en el parénquima y su ausencia hace muy poco probable el diagnostico de edema pulmonar cardiogénico.

La medición de la **ratio LA/Ao** (atrio izquierdo/aorta) es de especial interés en la diferenciación del origen cardiaco o respiratorio de la disnea. Requiere de cierto grado de entrenamiento, pero es relativamente sencillo. Consiste en medir el diámetro de la aorta y del atrio izquierdo en un corte bidimensional de eje corto paraesternal derecho. Una ratio 1:1 se considera normal y hablaremos de dilatación atrial y sospecha de ICC si la ratio es >1.5.

No obstante, la ecografía en urgencias nunca debe substituir a una ecocardiografía completa.

El diagnóstico de ICC no es fácil en gatos ya que los hallazgos en radiografía cardiaca pueden ser muy inespecíficos y muchas veces no se presentan con soplo cardiaco. Otra forma indirecta de diagnosticar ICC es midiendo niveles de NT-ProBNP que serán altos en casos de dilatación/estiramiento del musculo miocárdico y cardiomegalia. Actualmente hay test rápidos (SNAP proBNP) al alcance de los clínicos de emergencias[3]. Un valor normal o bajo descarta la causa cardiaca en pacientes con distrés respiratorio.

Si queremos evaluar **la ventilación/oxigenación alveolar**, podemos medir PaO_2 y $PaCO_2$ en una muestra arterial sanguínea y calcular diferentes índices para evaluar la función pulmonar:

Regla del 120 PaO_2 +$PaCO_2$>= 120	Si < 120 posible hipo oxigenación arterial No útil en pacientes con suplementación de oxígeno. Poco fiable
Ratio PaO_2/FiO_2 (P/F) **Buena correlación SpO_2/FiO_2 y P/F**	Ratio P/F 200-300: enfermedad respiratoria severa Ratio P/F <200 : sospecha ARDS (síndrome de distrés respiratorio agudo) No considera hipoventilación
Gradiente alveolo-arterial A– a= [150-$PaCO_2$]-PaO_2 A nivel del mar y con % oxigeno atmosférico (21%)	A– a < 10 es normal > 20 indica desajuste V/Q (ventilación/circulación) pulmonar (ej neumonía). Mayor numero indica mayor severidad. No usar en pacientes suplementados con oxígeno

El tratamiento de estos pacientes dependerá del diagnóstico definitivo. Un paciente con soplo cardiaco, aumento del esfuerzo y la frecuencia respiratoria, con crepitaciones pulmonares y una ratio LA/Ao por encima de lo normal es altamente sugestivo de ICC y requerirá del uso de diuréticos +/- toracocentesis si también presenta efusión pleural.

Pacientes con problemas de vía alta (colapso traqueal, síndrome de vías altas en braquiocefálicos, parálisis laríngea, obstrucción traqueal) pueden necesitar intubación inmediata (traqueal vs traqueostomía de emergencia), ventilación asistida o simplemente intubación temporal y suplementación con oxigeno hasta normalizar oxigenación y ventilación.
Pacientes con patología bronquial requerirán del uso de broncodilatadores y esteroides (inhalados +/- parenteral)

Pacientes con patología de vía baja (neumonía severa, edema no cardiogénico, síndrome de distrés respiratorio agudo (en inglés ARDS)) pueden presentar niveles de oxigenación bajos y necesitar de tratamientos más agresivos si no responden favorablemente a la suplementación y oxigeno inicial (cánula nasal de alto flujo, ventilación mecánica).

La cánula nasal de alto flujo es de especial interés ya que permite alcanzar flujos de oxígeno de hasta 60L/min administrando aire humidificado mejor tolerado por el paciente. Ha demostrado ser de utilidad en pacientes en insuficiencia cardiaca congestiva que no están respondiendo a la suplementación tradicional

con oxígeno, en pacientes braquiocefalicos en distrés y en recuperacion post cirugia de via repiratoria altas, pacientes hipoxémicos (edema pulmonar no cardiogenico, pneumonia). Es el paso previo a la ventilación mecánica.

Pacientes en los que el origen de la disnea sea neurológico (p ej: botulismo, tétanos, polirradiculoneuritis, mielomalacia) necesitarán de ventilación mecánica asistida en todos los casos. La suplementación con oxígeno en estos casos no será suficiente porque falla la capacidad de ventilación.

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MIELOPATÍAS CRÓNICAS

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ABSTRACT BODY:

Las mielopatías son frecuentes en la clínica diaria de pequeños animales. Estos casos pueden resultar un reto para el veterinario, ya que se han descrito más de 40 mielopatías diferentes, muchas de ellas con una presentación clínica similar. Frente estos casos, es imprescindible realizar un buen diagnóstico diferencial, que nos ayudará a poder elegir e interpretar de manera objetiva las pruebas diagnósticas más óptimas en cada caso.

Para ello, es muy útil utilizar el "razonamiento clínico". La información que obtendremos con la anamnesis y la reseña del paciente, la localización neurológica de la lesión, así como del inicio y la progresión del problema, es imprescindible para poder ordenar, de más a menos probable, las posibles enfermedades causantes del motivo de consulta de nuestro paciente.

Si nos centramos en los procesos crónicos, destacan las anomalías congénitas (que estarán presentes desde el nacimiento), la mayoría de procesos inflamatorios-infecciosos (de progresión rápida) y las neoplasias y la mayoría de procesos degenerativos, éstos últimos con una progresión más lenta.

Por tanto, cuando tengamos un paciente con una **mielopatía crónica**, nuestros principales diagnósticos diferenciales serán los siguientes (el orden de los mismos dependerá de la historia clínica, la reseña, la localización y simetría de los signos clínicos y la presencia de dolor o no):

MIELOPATÍAS CRÓNICAS MÁS FRECUENTES:

PROCESOS DEGENERATIVOS:

- MIELOPATIA DEGENERATIVA

La mielopatía degenerativa (MD) es la mielopatía neurodegenerativa más frecuente en perros. Su fisiopatología es desconocida, aunque se han hipotizado posibles mecanismos inmunológicos, metabólicos, nutricionales, de estrés oxidativo y factores genéticos como posibles causas. Histopatológicamente consiste en una degeneración no inflamatoria de los axones y de la mielina, y astrogliosis, de la médula espinal, con afectación principalmente e inicialmente de la parte dorsal de los funículos laterales y del funículo dorsal de la región torácica.

Puede afectar a cualquier raza mediana o grande, aunque se ha visto cierta predisposición en los Pastores Alemanes y Bóxers. Los signos clínicos pueden aparecer a partir de los 5 años de edad, con una edad media de presentación de 9 años. No se ha descrito predisposición de sexo, exceptuando los Pembroke Welsh Corgi, con más afectación de las hembras en relación 1.6: 1.

La enfermedad tiene una evolución progresiva lenta. Los signos clínicos iniciales consisten en una ataxia espinal de las extremidades posteriores y paraparesia espástica asimétrica. Una característica clínica típica de la enfermedad es que, en estadios iniciales y debido a la distribución de las lesiones medulares, se suelen apreciar más déficits propioceptivos (ataxia espinal) que motores (paresia). A medida que progresa la enfermedad, se manifestarán signos clínicos de neurona motora inferior (NMI) en las extremidades posteriores, con disminución de reflejos espinales e incontinencia fecal y urinaria. Posteriormente aparecerá debilidad en las extremidades torácicas, hasta llegar a tetraplejia. Otro factor clínico importante a tener en cuenta relacionado con la MD, es la ausencia de hiperestesia espinal.

El diagnóstico definitivo requiere histopatología de las lesiones de la médula espinal. El diagnóstico presuntivo se basa en la presencia de los signos clínicos característicos, la exclusión de otras enfermedades medulares, y un resultado positivo del test genético. Éste detecta la mutación del gen SOD1, considerado como alto factor de riesgo para desarrollar la enfermedad. Se trata de un gen autosómico recesivo con penetrancia incompleta. Así, los animales homocigotos para la mutación tienen alto riesgo de manifestar la enfermedad clínica. En cambio, los pacientes heterocigotos son portadores pero es poco probable que desarrollen la enfermedad clínica.

No existe ningún tratamiento eficaz demostrado actualmente para la MD. Se han realizado estudios experimentales con vitaminas, glucocorticoides, ácido aminocaproico, … pero sin resultados concluyentes hasta el momento. El pronóstico a largo plazo es reservado, con una media de progresión hasta la no ambulación de entre 6 y 12 meses. Se ha demostrado un mayor tiempo de capacidad para ambular así como del tiempo de supervivencia en animales sometidos a fisioterapia intensiva.

- PROTRUSION DISCAL

Es el único tipo de enfermedad del disco IV que tiene una presentación crónica y progresiva. El disco IV

sufre un proceso degenerativo relacionado con la edad llamado metaplasia fibroide. Se produce degeneración del núcleo pulposo y del anillo fibroso, produciendo debilitamiento del mismo y una protrusión progresiva del disco anormal hacia el canal vertebral, provocando una compresión medular focal, moderada y progresiva, causante de los signos clínicos. Generalmente va acompañado de hiperestesia a la palpación espinal. Afecta principalmente a razas medianas y grandes de perros no condrodistróficos y de edades adultos-geriátricos. Los discos más afectados son de T11 T12 hasta L2L3, y L7S1 en perros y a nivel de L4L5 Y L7S1 en gatos. En muchas ocasiones son múltiples. El diagnóstico requiere pruebas de imagen avanzadas (mielografía, TAC, RM) que evidenciará los discos degenerados y la compresión medular.

El tratamiento puede ser médico (reposo, analgesia, control del peso y fisioterapia) o quirúrgico, dependiendo de las características del paciente y del propietario, de los signos neurológicos, del grado de hiperestesia y de la respuesta a tratamientos médicos previos.

NEOPLASIAS

Las neoplasias espinales tienen un curso clínico crónico y progresivo lento, generalmente con signos asimétricos. Son más frecuentes en animales de edad avanzada, aunque ciertos tumores son típicos de pacientes jóvenes o en crecimiento. En la mayoría de los casos son dolorosos de manera focal, básicamente por dilatación meníngea o por alteraciones de flujo del LCR (síndrome siringomiélico) secundario.

A nivel de la médula espinal, las neoplasia se clasifican según su origen (primarias - cuando provienen de células de médula espinal, meninges o tejidos periespinales - o secundarios o metastáticos), y según su localización e histopatología, de la siguiente forma:

- Tumores extradurales: Representan un 50 % de los tumores espinales, y los más frecuentes son los tumores óseos, los linfomas y los tumores de células grasas.
- Tumores intradurales- extramedulares: Representan un 35 % de los tumores espinales, y se incluyen los meningiomas, los tumores malignos de vaina nerviosa y los nefroblastomas.
- Tumores intramedulares: Representan el 15 % de los tumores espinales y se incluyen diferentes tipos de tumores de células gliales.

El diagnóstico definitivo requiere análisis histopatológico de las lesiones, y el presuntivo se basa en los resultados de las pruebas de imagen, siendo la RM la técnica de elección en la mayoría de los casos.

El tratamiento y el pronóstico dependerá principalmente del tipo de tumor y de su localización.

PROCESOS INFLAMATORIOS- INFECCIOSOS

Consisten en un grupo heterogéneo de enfermedades que incluyen las inflamaciones de etiología infecciosa, y las denominadas “de etiología desconocida”, las cuales tienen un origen inmunomediado y predisposición genética. Éstas últimas son las más frecuentes en pequeños animales y se reconocen diferentes subtipos. Pueden afectar a todas las razas y edades, aunque es más frecuente en perros de raza pequeña y jóvenes de > 6 meses de edad. Contrariamente a la creencia popular, la mayoría de procesos inflamatorios no infecciosos, no suelen provocar signos sistémicos por lo que es poco frecuente detectar alteraciones en la exploración general o en la analítica sanguínea.

Pueden afectar las diferentes estructuras del sistema nervioso, ya sea encéfalo, médula espinal o SNP; sin embargo, la mayoría de veces tienen una distribución difusa o multifocal; es decir, afectando más de una parte del sistema nervioso. Solamente un 18% de los casos tiene una presentación puramente espinal (es decir, causando solo signos clínicos de mielopatía).

Los signos clínicos son crónicos y progresivos en el 52 % de los casos, con una evolución rápida de los signos clínicos. La simetría de los signos clínicos dependerá de la distribución de las lesiones. En cuanto a la presencia de dolor, dependerá del tipo de etiología, pero en general son dolorosos (71% de los casos) y más cuando hay afectación meníngea.

El diagnóstico definitivo requiere histopatología de las lesiones. El diagnóstico presuntivo se basa se basa en el resultado del análisis del LCR, que mostrará pleocitosis. La confirmación que el origen no es infeccioso se basa en los resultados negativos del LCR.

El tratamiento de las de “origen no infeccioso o etiología desconocida” se basa en corticosteroides +- otros fármacos inmunosupresores.

THE MICROBIOME AS A THERAPEUTIC TARGET IN GASTROINTESTINAL DISEASE

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ABSTRACT BODY:

What is intestinal dysbiosis?

Intestinal dysbiosis is the term applied to an imbalance in the composition of the enteric microbiome.

Dysbiosis across species

- *Chronic enteropathies and Lymphoplasmacytic enteritis in dogs and cats*
- Expansion of proteobacteria and reduction in beneficial firmicutes
  - Granulomatous colitis in dogs
- Comparative sequence based analysis reveal dysbiosis characterized by enrichment in *E.coli* and depletion in Clostridiales. I situ analysis indicates an association with intracellular *E.coli* in PAS+ GC in French bulldogs, Boxers and mastiff cluster breeds.
  - HGE/AHDS
- HGE/AHDS associated with *Clostridium spp* (culture and molecular studies) and most recently NetF producing *C.perfringens*
  - Crohn's disease
    - Expansion of proteobacteria such as AIEC *E. coli* and reduction in beneficial firmicutes such as *F.prausnitzii*

What causes dysbiosis?

- Genetic makeup
  - Defects impacting Paneth cell function
    - E.g. ATG16L, NOD2, Xbp1,IRGM
  - Defects associated with innate immunity
    - E.g. NOD2, CD48/SLAM, NCF
  - Defects associated with barrier function and endoplasmic reticulum stress
    - E.g. Sel1L, AGR2
- Antimicrobial therapy
  - Metronidazole
- Diet
  - Dietary composition may favor growth or repression of certain species:
    - E.g. prebiotics that stimulate growth of Clostridiales and *Faecalibacterium*. Dietary fat and sugars may suppress symbionts and promote growth of pathosymbionts: "Western diet"
- Intestinal Inflammation of any type

How does dysbiosis develop?

- Does dysbiosis precede overt inflammation in a genetically predisposed host?
  - Genes such as NOD2 and ATG16L have been associated with dysbiosis, but in and off itself the dysbiosis associated with genetic polymorphisms in the absence of inflammation, is not as extreme as that associated with inflammation.
- Do good bugs suppress bad bugs?
  - Yes! They can secrete products that suppress Gram negative paythosymbionts e.g. *Lactobacillus*
- Do bad bugs suppress good bugs?
  - Yes! They can directly inhibit the growth of resident symbionts . E.g. *Klebsiella*
- Does inflammation in and of itself cause dysbiosis?(Figure !)
  - Yes! It is has been clearly shown that inflammation in the absence of genetic susceptibility can create marked dysbiosis. The degree of dysbiosis is related to the severity of inflammation

Figure 1. Inflammation Drives Dysbiosis in the absence of genetic susceptibility: Genetic defects associated with IBD impact the threshold and magnitude of floral shifts

Why do beneficial bacteria become depleted in the inflamed intestine?

- Altered mucosal niche?
  - Loss of mucus, loss of surface area (villus atrophy), loss of metabolic capacity of a related symbiont, loss of nutrients/metabolites associated with a healthy flora
- Inability to utilize inflammation associated substrates
  - Lack of metabolic plasticity to adapt to a changing intraluminal microenvironment
- Direct attack by proliferating pathosymbionts

Why do "bad bugs" proliferate in the inflamed intestine?

- Loss of surveillance from good bacteria

- Resident beneficial bacteria such as *Lactobacillus* can secrete products that antagonize the growth of proteobacteria such as *E.coli*

- Ability to adapt to the inflamed environment
  - Dysbiotic bacteria are frequently adept at resisting oxidative and osmotic stress and injurious bile salts
- Ability *to utilize inflammation associated substrates*
  - Dysbiotic bacteria have a portfolio of metabolic pathways that enable them to utilize many different nitrogen and carbon sources.
  - Many Gram negative dysbiotic bacteria are enriched in pathways that enable them to use chemicals that are increased in the inflamed gut. E.g ethanolamine, glutamine.
  - Dysbiotic bacteria can be seen feasting on cellular material associated with IBD and the magnitude and type of dysbiosis may correlate with levels of host DNA (tissue damage) in feces.

Opportunities for therapeutic intervention

- Elimination of known pathogens (bacteria, algae, protozoa etc)
  - Antimcrobials , bacteriophage, environmental modification
- Dietary modification
  - avoidance of foods inducing clinical signs and intestinal inflammation e.g. food responsive enteropathy. Prebiotics to support probiotics.
- Eradicate pathosymbionts
  - Antibiotics, bacteriophage, modify luminal environment,
- Selectively restrict growth and virulence of pathosymbionts
  - e.g., inhibit growth by modifying luminal microenvironment, antagonzing metabolism, reduce motility and virulence gene expression, block adhesion and invasion
- Anti-inflammatory agents
  - Prednisolone at dose to restore epithelial function and reduce inflammation without immunosupression, ASA (dual effect on bacteria and host), rifaximin (dual effect on bacteria and host)
- Immunomodulation
  - Reduce or eliminate inflammation that is driving dysbiosis
  - Enhance protective immunity
- *Replenish beneficial symbionts*
- Probiotics: “live microorganisms, which when administered in adequate amounts, confer a health benefit on the host”
- Prebiotics
- Symbiotics
- Microbiome transplantation

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030 / #41

HOMEMADE DIETS: WHEN AND HOW TO USE THEM

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ABSTRACT BODY:

HOMEMADE DIETS: WHEN AND HOW TO USE THEM

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Homemade diets (HMDs) are an alternative to commercial diets and their use varies amongst countries. There are a variety of reasons why a pet owner might choose to home-cook for their pet (1). One of the reasons includes the lack of commercial options (due to lack of palatability or when multiple nutrient sensitive diseases are present). Whatever the reason, the HMD must be complete, balanced and adequate for the patient in order to safely meet their nutrient and energy needs. HMD and commercial diets have both pros and cons (2) and careful assessment is needed to decide if an individual patient can benefit from each method of feeding.

How are homemade diets formulated?

The formulator requires basic information, such as the nutrient needs of the patient and their energy needs. The former can be obtained from the literature for both healthy pets (3,4) and those with disease. Energy needs can be obtained by formulas (3,5), who have error, or via a complete diet history. Pets with lower energy needs will require nutrient fortification to compensate for the low food intake. Any maximums must also be known, to avoid toxicity issues. Finally, the nutrient profile of the ingredients of choice is also needed. This is usually obtained from public databases. Ingredients are then combined (with supplements, usually) to meet the needs for each pet.

Risks associated with homemade diets

In the author's experience, the risks of HMD include cost (in money (6) but also in time, space, and resources), lower consistence, and nutritional inadequacies (2). Nutritional inadequacies are expected when the diets are inadequately formulated and can be associated to clinical signs of deficiency or toxicity, this can take a long time to appear, especially in adults. Generic recipes found in books and internet are notorious for their nutritional issues (7,8), especially given the lack of specific instructions in many cases. Other problems of these recipes include outdated strategies or nutritional requirements, over-complexity or simplicity, and use of dangerous ingredients (such as garlic). And they do not have the main benefit of HMDs: customization to the pet. However, nutritional inadequacy can also occur in properly formulated diets, for example due to diet drift (9), limitations of the nutrient database of ingredients and, most importantly, due to the lack of testing of HMD, as opposed to commercial pet food from reputable companies.

Pros of homemade diets: when to use them

There are some situations where HMDs can be very helpful, such as in finicky patients (although some will still prefer commercial diets, especially if the diet requires protein or fat moderation), when very highly digestible diets are needed, or in some patients with co-morbidities, as long as they are complete and balanced, and the patient has frequent veterinary visits for nutritional assessment (10). Homemade diets can be fully customized to patient and owner, which is of particularly interest if there are no commercial diets that suit the situation.

Resources

In the USA and Europe, pet owners and veterinarians can consult with a boarded veterinary nutrition specialist from ACVIM (Nutrition) or ESVCN. Other countries might have their own veterinary nutrition specialization organization, and the veterinary schools or the veterinary licensing bodies can also be contacted for information. Many services can work remotely through a referring veterinarian.

Patients eating homemade diets should be visited at least twice yearly (or even more, depending on the health status of the patient) to undergo a complete nutritional evaluation assessing the pet, the diet (in detail), and the environment, to ensure the homemade diet is being used properly and to assess if adjustments are needed. The WSAVA website has useful tools to perform this evaluation

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031 / #42

SELECTED CASES FROM PET FISH VETERINARY PRACTICE

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ABSTRACT BODY:

Whether the full-time dedicated pet fish veterinarian or the small animal- exotic veterinarian who sees the occasional “wet” patient, pet fish cases are always interesting and often quite challenging. Despite having a well-equipped facility, the varied life support equipment, multitude of species differences and inherent difficulties in using supportive diagnostics readily available for our furry friends can make successful resolutions hard to achieve. For those who are up for the challenge, this presentation will explore some of the more interesting cases seen in multiple species of pet fish.

EMERGING CONCEPTS IN FELINE VECTOR-BORNE DISEASES

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ABSTRACT BODY:

INTRODUCTION

Ticks on domestic cats are often overlooked as parasites and vectors of infectious disease agents. This oversight may result from the belief that cats are fastidious groomers and remove ticks before attachment takes place and thus insufficient time for transmission of infectious agents to occur. As some cats spend a majority of their time indoors, owners may be led to believe that their cats are not exposed to ticks. Feline vector-borne diseases (FVBD) have, however, emerged in recent years, showing a wider geographic distribution and increased global prevalence. In addition to their veterinary importance, domestic cats play a central role in the transmission cycles of some FVBD agents by acting as reservoirs and sentinels, an occurrence that requires a One Health approach.

CYTAUXZOONOSIS

Cytauxzoon felis infection is transmitted by *Amblyomma americanum* (lone star tick) and *Dermacentor variabilis* (American dog tick) and is considered the most severe disease transmitted by ticks to cats. Infected bobcats act as the wild animal reservoir and chronically infected domestic cats are the urban reservoirs. Cytauxzoonosis is seen in domestic cats throughout the central, southeastern, and south-central USA.

Seasonal activity of ticks is a key component in the transmission of *C. felis* as the number of cases increase with increased tick activity. In addition, outdoor cats living in wooded habitats are at higher risk for infection. Neither age nor breed predisposition for infection has been reported.

Typical clinical signs are pyrexia, inappetence, anorexia, dyspnea, and icterus with the disease having a severe acute nature, which rapidly progresses with death occurring 1-7 days after onset of clinical signs.

Diagnosis of cytauxzoonosis was historically based on finding the parasites in blood smears by light microscopy; however, the current gold standard is the use of PCR, which also allows the detection of infection prior to onset of clinical signs.

Recommended treatment for cytauxzoonosis is atovaquone (15 mg/kg PO TID) together with azithromycin (10 mg/kg PO SID). Imidocarb dipropionate has been used but not recommended due to its lack of efficacy and side effects. In the debilitated cat, administration of azithromycin intravenously as a slow infusion can be considered.

BABESIOSIS

Babesiosis is a tick-borne protozoan disease caused by parasites of the genus *Babesia* belonging to the *Piroplasmida* group. Multiple species of *Babesia* organisms have been documented in cats including *Babesia felis*, *B. herpailuri*, *B. cati*, *B. vogeli*, *B. canis subsp. presentii*, *B. canis subsp. canis*, *B. pantherae*, *B. microti*, *B. leo*, *B. venatorum*, *B. hongkongensis B.* l*ohae*, and *B.* v*ulpes*. The level of genetic diversity within *Babesia* species suggests that new subspecies can develop easily. Reports of occurrence of *B. felis* and other *Babesia* species in domestic cats should only be accepted if they are supported by credible molecular evidence.

The pathogenicity of *Babesia* spp. is dependent on many variables including species, strain, age of the host, and immunologic response. The species and strain appear to be the most important factors in determining pathogenicity.

Common clinical signs are anorexia, lethargy, weakness, and rough hair coat. Pyrexia and icterus are uncommon. Both the clinical signs and laboratory changes can often be attributed to the hemolytic anemia that occurs secondary to the disease.

Babesiosis can be diagnosed on peripheral blood smear examination. However, when the level of parasitemia is low, detection of the organism in blood smears can be difficult. In addition, the different *Babesia* species and some other blood parasites, including *Cytauxzoon felis*, are morphologically indistinguishable. The best method for definitive diagnosis is PCR, which also allows differentiation between species and determination of co-infections.

The drug of choice for *B. felis* is primaquine at 0.5 mg/kg PO SID for 1–3 days, then repeated every 7 days for four doses. Although primaquine is capable of reducing parasitemia, it does not eliminate the parasite. Rifampicin and sulfadiazine–trimethoprim have shown to have some anti-parasitic effect but less effective than primaquine. Diminazine aceturate and imidocarb diproprionate have shown promising results in the

treatment of the large babesial species of cats.

Unfortunately, there are no controlled clinical trials evaluating therapeutic regimens in feline babesiosis and such trials are desperately needed. Combination therapy with azithromycin and atovaquone or clindamycin and quinine, doxycycline, clindamycin, and metronidazole may all have some efficacy against the Babesial spp of the cat.

Prognosis depends on the severity of disease, which in turn depends on both the organism and host factors. Mortality rates of 15–20% have been reported. Concurrent infection with *Mycoplasma hemofelis*, FeLV or FIV has a negative impact on the response to treatment and outcome of disease.

FELINE GRANULOCYTOTROPIC ANAPLASMOSIS

Feline granulocytotropic anaplasmosis is caused by *Anaplasma phagocytophilum* reported in Germany, Austria, Poland, Switzerland, Italy, the UK, Finland, Sweden, and the USA. It is transmitted by *Ixodes* and *Dermacentor* species of ticks. Although the disease is common in both dogs and humans, it has been poorly described in cats but is of zoonotic significance.

Clinical signs of feline anaplasmosis include pyrexia, lethargy, anorexia, arthralgia, lameness, lymphadenomegaly, weight loss, and neurologic signs.

Diagnosis can be based on clinical signs, history of tick exposure, and the microscopic detection of morulae within the neutrophils in acute infection. However, morulae are often difficult to find, as the parasitemia can be very low. Although, serology and PCR are the preferred methods confirming the diagnosis, studies have reported negative PCR results from cats with clinical illness and positive on serological tests. The latter, however, may merely indicate prior exposure to the parasite. Possible explanations for PCR negative cats would be that *A. phagocytophilum* is sequestered in low numbers in tissues, or that infected cats quickly clear the parasite.

Recommended therapy is doxycycline at 10 mg/kg PO SID for 28 days.

FELINE MONOCYTOTROPIC EHRLICHIOSIS

Feline monocytotropic ehrlichiosis is caused by an *Ehrlichia canis*–like agent, however, even though a large number of suspected feline ehrlichiosis cases have been reported, *E canis*–like DNA has been demonstrated from only a few infected cats.

Clinical signs include pyrexia, lethargy, inappetence, weight loss, and polyarthritis.

Diagnostic criteria for feline ehrlichiosis have not yet been clearly established, although positive serology and organism detection in the blood supports the diagnosis.

For cats with suspected clinical ehrlichiosis, administration of doxycycline at 10 mg/kg PO SID for a minimum of 28 days is recommended.

TULAREMIA

Tularemia is caused by *Francisella tularensis*, which is a non-motile, aerobic, encapsulated gram-negative coccobacillus. The disease is a notifiable zoonotic disease. Cats can be exposed to *F. tularensis* through direct contact with infected wildlife (cottontail rabbits, hares, and rodents), vectors, or contaminated environment and are more susceptible to the disease than dogs. Vector transmission is via *Amblyomma americanum*, *Dermacentor variabilis*, and *D. occidentalis*.

Clinical signs include pyrexia, anorexia, marked depression, dehydration, lymphadenopathy, splenomegaly, hepatomegaly, icterus, and acute oral or lingual ulcers.

Diagnosis is most commonly based on clinical signs and confirmed by paired serum titers.

Therapy of choice is gentamicin at 5 mg/kg SC, IV, or IM SID for 7–14 days. Although doxycycline and fluoroquinolones may be effective, relapses are common.

LYME BORRELIOSIS

Lyme borreliosis is caused by *Borellia burgdorferi* and despite antibodies to *B. burgdorferi* being found in cats from endemic areas, a distinct clinical entity has to date not yet been described. Cats are hosts for the tick vectors, *Ixodes scapularis* and *Ixodes pacificus,*and a prevalence of antibodies to *B burgdorferi* (up to 47%) has been reported in endemic areas of the northeastern USA.

Cats experimentally infected with *B burgdorferi* show signs of arthritis and meningitis. Recommended treatment for suspected Lyme borreliosis in cats is doxycycline at 10 mg/kg PO SID for 28–30 days.

HEMOPLASMOSIS

Hemoplasmosis is caused by *Mycoplasma*, of which currently there are 3 species, namely *M. haemofelis*, *M. haemominutum*, and *M. turicensis*. The incubation period of the disease varies between 6-17 days and has a range of 20-34 days. The disease can be divided into the pre-parasitemic, acute, recovery, and carrier phase.

Infection with *M. hemofelis* often causes a severe hemolytic anemia, whereas *M. hemominutum* infections do not usually induce clinical signs, although some reports have documented a mild or moderate anemia. It has also been suggested

that co-infection with *M. hemominutum* and feline retroviruses may result in anemia. The pathogenic potential of *M. turicensis* seems to depend on cofactors but has been reported to result in hemolytic anemia.

Clinical signs are generally non-specific. Infected animals may exhibit depression, lethargy, anorexia, weight loss, weakness, and pale mucous membranes. Icterus is variably present. In the acute phase, pyrexia may be apparent whereas chronically infected cats tend to be normo-thermic. Hepato-splenomegaly and generalized lymphadenopathy may be present.

Hemoplasmosis can be diagnosed on a peripheral blood smear examination. However, when the level of parasitemia is low, detection of the organism in a blood smear can be difficult and often it is difficult to distinguish the organisms from Howell-Jolly bodies, basophilic stippling, and /or stain deposit. In addition, *Babesia felis* and *Cytauxzoon felis* create additional confusion. The best method for definitive diagnosis is PCR, which also allows differentiation between species and determination of co-infections.

Therapy is doxycycline, together with corticosteroids; the latter as the anemia associated with hemoplasmosis has an immune mediated pathogenesis.

033 / #262

10 THINGS TO HELP YOU TAKE WOW BIOPSIES

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ABSTRACT BODY:

How to take great biopsies and obtain the right results

- Know your limits; consider referral if a biopsy procedure exceeds your skill set.
- Take a picture before taking a biopsy, and afterward, label carefully, especially if you have multiple biopsy sites.
- Choose the biopsy location wisely.
- Choose incisional biopsy over excisional if the lesion can not be completely excised with adequate margins.
- Choose your biopsy instrument wisely.
- Prepare the site aseptically and use sterile instruments.
- When in doubt, take multiple biopsies.
- Plan for proper fixation of the tissue.
- Charge appropriately.
- Communicate with your pathologist.

034 / #48

10 THINGS TO HELP YOU TAKE WOW BIOPSIES

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ABSTRACT BODY:

Because this is a lecture shared by two speakers, five of the biopsy suggestions are in abstract prepared by Dr. Jolle Kirpensteijn.

- *Know your limits; consider referral if a biopsy procedure exceeds your skill set.*

The decision on whether or not to take a biopsy yourself or to refer the patient to someone with better surgical skills depends on tumor size and location and also if knowing the tumor type before hand impacts surgical planning. Biopsy of diffuse skin lesions with a biopsy punch is simple and every veterinarian can perform this procedure. The exception to this might be widespread cutaneous mast cell tumor because of the risk of bleeding if the tumor cannot be completely resected. Soft tissue sarcomas range from the low malignancy grade I tumor to the more metastatic grade III tumor. Knowing the grade in advance of surgery helps to determine how urgent the surgery is and how important wide margins are. Tumors can occur in difficult anatomic locations such as toes, near the rectum, vulva or eyes. Excisional biopsy by someone with excellent surgical skills benefits the patient and facilitates tumor control.

2. Take a picture before taking a biopsy and afterward, label carefully, especially if you have multiple biopsy sites.

Photographs of the lesion before and after biopsy helps you remember exactly where you biopsied but also helps the pathologist reading the biopsy to understand where the tissue came from. Preoperative photos are useful to help the surgeon identify where the lesion to be resected is found. Photographs are also an important component of medical record keeping.

3. When in doubt take multiple biopsies.

Obtaining multiple biopsies helps to determine the type and extent of a disease process. The term multiple biopsies can be interpreted several ways. It might encompass multiple samples of the same large tumor, multiple samples of a multifocal lesion such as chronic skin lesions or in the case of exploratory laparotomy, biopsies of multiple organs. Pets may have more than one disease or especially in the case of enteropathy, the abnormalities may not be distributed evenly throughout the gastrointestinal tract. Large tumors frequently have necrotic areas preventing accurate diagnosis. Finally, biopsy of regional lymph nodes, even if they are normal size, will help the oncologist direct treatment and issue a prognosis.

4. Plan for proper fixation of the tissue.

10% formalin is used to preserve samples for histopathology. The pathology laboratory provides formalin in containers for biopsy submission. Small samples like endoscopic or punch biopsies should be submitted in a tissue cassette. Check with your laboratory about submitting samples larger than the container. Do not force a sample into the opening of the container as when fixed, the tissue becomes stiff and will be difficult to remove from the container. Check with your pathology laboratory about packaging samples for shipping.

5. Communicate with your pathologist.

Multiple avenues exist for communication between the pathologist and clinical veterinarian. The conversation starts when choosing a pathology laboratory. The pathology laboratory's website provides sample submission information and usually a sample submission form where history, location, lesion description, type of biopsy can be communicated to the pathologist. Inking or marking margins tells the pathologist where to focus their attention regarding the completeness of excision. The histopathology report represents the communication from the pathologist to the veterinarian. If you have questions about the biopsy or additional testing that has been recommended, the pathology reports list the telephone number and email of the pathologist for ready access to the answers.

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PROBLEMAS DE CONDUCTA Y SALUD: UNA RELACIÓN INSEPARABLE

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ABSTRACT BODY:

El hecho de que un animal se comporte de una determinada ante una situación dada depende de multitud de factores. El estado de salud del animal es uno de ellos. El cambio en el comportamiento del animal es uno de los principales signos clínicos de muchas patologías. Con frecuencia, el cambio en el comportamiento del animal es el principal motivo por el cual los propietarios acuden al veterinario.

El objetivo de esta charla será en explicar la relación existente entre cada patología y la etología clínica, no profundizar en la fisiopatología de cada problema. Las patologías que se incluirán han sido elegidas por, por su frecuencia, porque suponen un reto diagnóstico para el clínico y por su interés científico.

Problemas neurológicos

El sistema nervioso central (SNC) tiene un papel directo sobre el comportamiento de los animales. Cualquier alteración en el funcionamiento del SNC puede cambiar el comportamiento del animal.

Desde un punto de vista práctico los problemas de comportamiento debido a problemas neurológicos se pueden dividir en 4 grandes grupos (Tabla 1). Aquellos problemas neurológicos que cambian el comportamiento a la vez que producen alteraciones en el examen neurológico y en las pruebas complementarias (Grupo 1). Algunos problemas de neurológicos producen problemas de comportamiento, alterando el examen neurológico, pero sin modificar las pruebas complementarios (Grupo 2). Son problemas poco frecuentes, normalmente problemas degenerativos del SNC, pero potencialmente muy graves que suelen terminar con la vida del animal.

En el caso de los animales del grupo 3, muestran problemas de comportamiento debido a un problema neurológico, pero, sin embargo, el animal no muestra déficits en el examen neurológico. Si se le realizaran pruebas complementarias, se podría llegar a un diagnóstico. Sin embargo, estos casos resultan un reto ya que cuando se realiza el historial etológico se observa un cambio de comportamiento (suele ser el motivo de consulta de los propietarios) pero, sin embargo, cuando se realizan las pruebas médicas básicas, al no observarse alteraciones en el examen neurológico, suelen rechazarse las pruebas complementarias más invasivas o costosas que podrían poner de manifiesto el diagnóstico problema. Existen algunos estudios muestran que lesiones que afectan especialmente a la zona más rostral del cerebro podrían estar en este grupo, ya que esta es una zona del SNC que no tiene su reflejo en el examen neurológico. Especialmente aquellos tumores de crecimiento lento que no producen alteraciones secundarias asociadas al tumor de forma repentina (como el aumento de la presión intracraneal), pueden cambiar el comportamiento sin alterar el examen neurológico.

Finalmente, los perros del Grupo 4 son un importante reto diagnóstico puesto que son animales con un problema de neurológico que les cambia el comportamiento, pero no modifica ni el examen neurológico ni las pruebas complementarias rutinarias y más utilizadas actualmente. Los dos problemas más comunes que se incluyen en este grupo son las epilepsias (totales y parciales) y los traumatismos craneoencefálicos leves.

Tabla 1. Problemas neurológicos y de conducta.

	Déficits del examen neurológico	Alteraciones laboratorio y/o imagen
Grupo 1	+	+
Grupo 2	+	-
Grupo 3	-	+
Grupo 4	-	-

Patologías que cursan con dolor y los problemas de comportamiento

Los cambios de comportamiento son el signo de dolor más común. Estos cambios de comportamiento inducidos por el dolor pueden clasificarse en dos grandes grupos, “pérdida de ciertos comportamientos” y “aparición de determinadas conductas”. Tanto la pérdida de estas conductas como la aparición de otras son indicadores de falta de bienestar del animal.

Dolor y agresividad

El dolor puede inducir agresividad mediante varios mecanismos. Está ampliamente aceptado que puede producir una reacción defensiva con la finalidad de evitar el contacto físico para evitar así un mayor daño. En otras palabras, si un animal tiene una extremidad fracturada y alguien va a manipularla, con toda probabilidad el animal puede reaccionar

de forma agresiva para evitar dicha manipulación y, consecuentemente, sentir más dolor.

Sin embargo, el dolor también puede facilitar la aparición de conductas agresivas por otras vías. Existen dos patrones diferentes de expresión de la agresividad por dolor. Las diferencias entre los dos patrones de expresión dependen de si ya era agresivo antes del inicio del problema de dolor. En el primer grupo, los perros que nunca habían sido agresivos antes del inicio del dolor, la agresividad se relacionaba con más frecuencia en contextos en los que el propietario intentaba manipular al animal y, además, era una agresividad más impulsiva. Sin embargo, en el segundo patrón de expresión de agresividad por dolor, los animales que ya eran agresivos por otras causas antes del inicio del problema de dolor, lo que se observaba es que los animales aumentaban la frecuencia y la intensidad de los ataques en los mismos contextos en los que previamente se mostraban agresivos. Además, eran menos impulsivos que los perros del primer grupo. Este aumento en la intensidad y la frecuencia podría deberse a la disminución del umbral de la agresividad causada por la respuesta de estrés crónica inducida por el dolor.

Dolor y miedo, fobias y ansiedad

El papel que ejerce el dolor en la aparición de miedos, fobias y/o ansiedad no está todavía del todo claro y podrían estar implicados, al menos, dos mecanismos.

El primer mecanismo está ampliamente aceptado. Las vías neurológicas del miedo, el dolor y la ansiedad están íntimamente relacionadas (química y funcionalmente). El dolor actúa como un estímulo incondicional que induce una respuesta de miedo. En otras palabras, cuando un animal se encuentra en una situación que le causa dolor, intentará crear asociaciones entre el estímulo causante del dolor y otros posibles estímulos inicialmente neutros que estén presentes en el momento y que puedan ayudarle a predecir una situación similar en el futuro. Por ejemplo, si un perro acude al veterinario y le explora y le hace daño, el animal rápidamente podrá asociar al veterinario y la sala de exploración en general a ese dolor que ha experimentado. De esta manera, en próximas exploraciones en el mismo entorno, el animal puede mostrar miedo, aunque el animal ya no tenga dolor o, ni siquiera, estésiendo explorado.

El segundo mecanismo no es tan intuitivo y está siendo estudiado en la actualidad. Aunque todavía no está demostrado en perros y gatos, se asume que el dolor puede inducir un sesgo negativo en el animal que favorecería la aparición de problemas de miedo y ansiedad.

Dolor y conductas repetitivas

El dolor puede tener diferentes papeles en el desarrollo de las conductas repetitivas. En primer lugar, de una forma directa ya que algunas conductas repetitivas, como por ejemplo el exceso de acicalamiento en el gato o la dermatitis acral por lamido (DAL) en el perro, pueden deberse a un problema de dolor localizado.

Dolor y disfunción cognitiva

Está ampliamente demostrado que el dolor puede afectar al ciclo sueño-vigilia, mediante la estimulación del sistema reticular activador en el SNC. Además, como ya se ha comentado anteriormente, el dolor también puede disminuir la actividad del animal (igual que ocurre en la disfunción cognitiva). De la misma manera, los animales con dificultades para la movilidad pueden empezar a eliminar de forma incorrecta en el interior de casa simplemente porque al disminuir su actividad los paseos sean más cortos, o porque prefieran no salir al exterior de la casa para orinar o defecar para evitar la situación de dolor (andar o bajar escalones, por ejemplo). Por lo tanto, tres síntomas comunes en la disfunción cognitiva pueden justificarse también por la presencia de dolor (especialmente si es crónico) en el animal.

Hipotiroidismo y problemas de comportamiento

El hipotiroidismo se ha asociado con frecuencia a diferentes alteraciones del comportamiento. Las más frecuentes son cambios en la conducta sexual, cambios en la actividad del animal y, finalmente, a la aparición de la agresividad y el miedo. Sin embargo, la evidencia científica al respecto, al menos en perro, es escasa. Sin embargo, estudios recientes con técnicas de neuroimagen funcional, realizados en medicina humana y en animales de laboratorio, muestran que el hipotiroidismo altera la función de importantes centros del SNC y que, además, el tratamiento farmacológico revierte dichas alteraciones. Además, se ha visto que, en perros, la agresividad no altera la ni la concentración de T4 ni la TSH.

Conclusiones

El estudio del comportamiento animal no debería entenderse como aislado. El comportamiento el animal depende directamente del funcionamiento de todos sus órganos y sistemas. Por lo tanto, ante el diagnóstico de cualquier problema de comportamiento siempre debe incluirse una valoración del estado de salud del animal.

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QUE HEMOS APRENDIDO EN 30 AÑOS DE PROFESIÓN DE: PATOLOGÍA DE RODILLA

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ABSTRACT BODY:

Que hemos aprendido en 30 años de profesión de: Patología de rodilla

La principal causa de osteoartrosis (OA) de rodilla en el perro es la rotura del ligamento cruzado anterior (LCA). La etiopatogenia de la rotura del LCA va ligada a varios factores: edad >4 años, raza: Golden, Labrador, Rotweiller,etc…, sexo: más en hembras que en machos , peso: > 25 kgrs, particularidades anatómicas : fosa intercondílea estrecha, pero actualmente se considera que los dos hechos más importantes son : la degeneración gradual y crónica del ligamento que está relacionada con una inclinación de la meseta tibial excesiva y un empuje craneal excesivo del fémur sobre la tibia.

El diagnóstico generalmente es clínico: cojera, extremidad extendida al sentarse: “sitting-test” y en demostrar la inestabilidad de la rodilla mediante el signo del cajón y la traslación craneal de la tibia al flexionar el tarso. Es útil chequear la otra extremidad para comparar y evaluar mejor la inestabilidad de la extremidad afectada. Siempre se debe hacer con el animal sedado o anestesiado. Un 25% de las roturas del LCA son parciales y dificultan el diagnóstico. En casos de duda la radiografía nos puede ayudar ya que podremos observar un desplazamiento craneal de la grasa debido a la presión del derrame articular. La punción y citología del derrame nos muestra un líquido sinovial degenerativo. La RM y artroscopia son útiles en caso de duda y para inspeccionar los meniscos.

El tratamiento de la rotura del LCA generalmente es quirúrgico. Los animales sometidos a tratamientos conservadores suelen desarrollar más OA que los tratados quirúrgicamente

Clásicamente se han usado técnicas de estabilización estática: Las técnicas intracapsulares usan mayormente una tira de fascia lata que se ancla la cóndilo femoral lateral mediante un tornillo y una arandela con dos variantes : “over the top” y “over and under the top” en la que la tira de fascia se pasa por debajo del ligamento transverso de la rodilla

Las técnicas extracapsulares usan cerclaje metálico o nilón que va desde la cresta tibial a la inserción del músculo gastronemio en el fémur distal a nivel del sesamoideo. El objetivo es que la fibrosis peri articular que se desarrolle estabilice la rodilla. Últimamente se han descrito puntos isométricos que se unen con filamento politrenzado.

Las técnicas de estabilización dinámica son cada vez más populares y se han ido abandonando las técnicas de estabilización estática ya que las primeras confieren mejores resultados clínicos.

La estabilización dinámica se inicia a partir de los trabajos de Slocum en que consideraba que la causa principal de la rotura del LCA es la excesiva inclinación de la meseta tibial y propone nivelar el ángulo de la meseta a 5º mediante una osteotomía en cuña de la tibia proximal :TWO (Tibial Wedge Osteotomy). Posteriormente se desarrolló un corte circular de la tibia proximal que se estabiliza con una placa especifica TPLO (Tibial Plateau Levelling Osteotomy)

La TTA (Tibial Tuberosity Advencement) es otra técnica de estabilización dinámica propuesta por Montavon en Zúrich que demuestra que biomecanicamente avanzando la tuberosidad tibial de manera que el ligamento tibio-rotuliano sea perpendicular a la meseta tibial se elimina el empuje craneal y se obtienen resultados clínicos similares a la TPLO.

Existen además otra técnicas de estabilización dinámica como la TTO (Triple Tibial Osteotomy) o la basada en el cora: CBLO (Cora Based Levelling Osteotomy) pero las más usadas en nuestro entorno son las dos primeras: TPLO y TTA

Existe controversia en la decisión de que técnica elegir. La elección vendrá determinada por cada caso en particular, la experiencia del cirujano y las posibilidades técnicas decidirán el procedimiento .

Estudios clínicos recientes indican mejores resultados con la TPLO con una tasa de complicaciones muy parecida a la TTA. Dos metanálisis realizados bajo criterios de Medicina Basada en la Evidencia concluyen que la TPLO es superior funcionalmente a las técnicas extracapsulares pero que no hay suficientes datos para afirmar cuál de los dos procedimientos ,TPLO y TTA , es mejor y que se necesitan más estudios comparativos.

Con la evolución de los implantes han disminuido las complicaciones en ambos procedimientos. La TTA no estaría indicada en casos en que la inclinación de la meseta tibial sea de 30º o más y tampoco si la tuberosidad tibial es muy estrecha o la inserción del

ligamento rotuliano es muy bajo.

Existe también controversia en cómo manejar el menisco. Por razones anatómicas el menisco más afectado es el medial .En rodillas con rotura de ligamento cruzado anterior se evidencia un 40-45% de lesiones de menisco cuando la rodilla se explora mediante artrotomía. Si la exploración se realiza mediante artroscopia estos porcentajes se incrementan por un ratio de 1.5-1.8 ya que la artroscopia evidencia lesiones más pequeñas no detectables mediante artrotomía. Por otro lado sabemos que no toda lesión de menisco necesita cirugía desde el punto de vista clínico por ejemplo algunas lesiones longitudinales y transversas y que una artrotomía o artroscopia inducen cierto grado de sinovitis y OA. En caso de menisectomía hay que preservar la zona periférica del menisco que es la más vascularizada y ante un menisco asintomático con una lesión moderada radial o longitudinal es posible que la mejor opción sea no extraerlo. En un estudio sistemático en que se evalúan 1792 referecias y se selecciona 89 no se obtienen resultados de forma categórica sobre el diagnóstico y tratamiento de las lesiones meniscales relacionadas con la rotura del LCA. La liberación preventiva del menisco basada en un corte transversal del mismo para liberarlo y evitar lesiones posteriores del mismo es otra práctica en discusión y no hay unanimidad sobre su utilidad.

La progresión de signos de artrosis radiológica es un problema frecuente después de la reparación del ligamento cruzado en todas las técnicas, aunque muchas veces no manifiestan sintomatología clínica. Concretamente en la TTA la aparición posterior de OA se relaciona con lesión del cartílago previa a la cirugía y poco avance de la tuberosidad tibial.

Dado que las técnicas de estabilización dinámica no contrarrestan totalmente las fuerzas de tracción ni las de rotación, algunos autores proponen combinar la TTA o la TPLO con sutura lateral extracapsular.

Estudios recientes proponen técnicas intracapsulares (hueso-hueso), utilizando injertos a partir de tendones flexores de los dedos obtenidos de cadáveres. Estos tendones se someten a un proceso de “decelularización” para evitar fenómenos de rechazo y se implantan al receptor intrarticularmente siguiendo en trayecto fisiológico del LCA.

037 / #235

LOCAL BLOCK MADE EASY

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ABSTRACT BODY:

Local anaesthetics inhibit membrane depolarization, nerve excitation and conduction by blocking primarily inward Na^+ currents through voltage-gated Na^+ channels. These drugs are inexpensive, not scheduled, and are readily available world-wide. Therefore there is the potential for widespread applicability of local anaesthetics for the management of pain in cats and dogs.

Local anaesthetic drug pharmacology

The most commonly used local anaesthetic drugs in small animals are lidocaine, mepivacaine, bupivacaine and ropivacaine. All of these agents are classed as aminoamide local anaesthetic drugs with their structure comprising of an aromatic group connected to a terminal amine via an amide linkage. Local anaesthetics are weak bases and therefore equilibrate within the body according to their pKa. The pKa of a drug is the pH at which 50% of the drug is in the ionized and 50% of the drug is in the non-ionized form. This is important for local anaesthetics because it is the unionized form of the drug that can cross the neuronal cell membrane to access the voltage gated Na^+ channel, whereas it is the ionized form of the drug that binds to the Na^+ channel receptor to block Na^+ ion entry into the neuron. Therefore local anaesthetics with a low pKa nearer physiological pH such as lidocaine will have a more rapid onset of action because a greater proportion of the drug will be unionized at physiological pH. The other physiochemical properties that determine the characteristics of local anaesthetic drugs are molecular weight, lipid solubility and protein binding.

Molecular weight is inversely related to the ability of the local anaesthetic to diffuse through tissue

Lipid solubility will determine the potency of the local anaesthetic and duration of action. Local anaesthetic drugs with a low lipid solubility have a lower potency and a shorter duration of action because they don't penetrate the nerve membrane as well as drugs with a high lipid solubility. Drugs with high lipid solubility also tend to have a slower onset of action because the drug becomes trapped in the myelin surrounding the neuronal cell membrane

Protein binding determines the duration of action of local anaesthetic drugs. Drugs that are highly protein bound bind more firmly to the receptor site within the Na^+ channel and therefore have a more prolonged duration of action.

Physiochemical properties of different local anaesthetic drugs

Local anaesthetic	pKa / Onset time (mins)	Protein binding / duration of action (hours)	Lipophilicity / potency	Maximum recommended dose (mg/kg)
Lidocaine	**7.8 / 5-10**	**Moderate / 1-1.5**	**Moderate**	**Canine: 6-10** **Feline: 3-5**
Bupivacaine	**8.1 / 20-30**	**Long / 3-10**	**Potent**	**Canine: 2** **Feline: 1-1.5**
Ropivacaine	**8.1 / 20-30**	**Long / 3-6**	**Potent**	**Canine: 3** **Feline: 1.5**
Mepivacaine	**7.7 / 5-10**	**Moderate / 1.5-2**	**Moderate**	**Canine: 5-6** **Feline:2-3**

Systemic toxicity of local anaesthetics

Systemic toxicity of local anaesthetics is most likely to occur through accidental overdose and is therefore more likely in smaller patients such as cats and small dogs than larger breeds of dog.

Factors affecting systemic toxicity include:

- The site of injection: Vascular injection sites lead to a more rapid absorption of the drug into the systemic circulation and therefore higher plasma concentrations of the drug with a greater risk of toxicity. Inadvertent intravenous or intraarterial injection is also a big risk factor for toxicity especially for local anaesthetics such as bupivacaine. Some local anaesthetic preparations contain epinephrine (e.g. lidocaine with epinephrine)

as a vasoconstricting agent to delay the absorption of the drug into the systemic circulation. As well as prolonging the duration of action of the local anaesthetic this will reduce the risk of systemic toxicity.

- Drug used: For example, bupivacaine is particularly cardiotoxic due to its slow dissociation from voltage gated Na^+ channel receptors in the heart.

Cardiovascular effects of local anaesthetics

Due to a combination of slowing of conduction in the myocardium, myocardial depression and peripheral vasodilation, hypotension, bradycardia and cardiac arrest can occur when systemic concentrations of local anaesthetics reach toxic levels.

Central nervous system effects of local anaesthetics

Local anaesthetics are lipid soluble and have a low molecular weight and therefore readily cross the blood-brain-barrier. At sub toxic doses, local anaesthetics can act as anticonvulsants, sedatives and analgesics. At higher concentrations they cause convulsions followed by generalized CNS depression.

Management of local anaethetic toxicity

Treatment of local anaesthetic drug toxicity is largely supportive. Benzodiazepines can be administered to manage seizures along with oxygen therapy and endotracheal intubation and ventilation if needed. Fluid support and inotropes may be needed to manage cardiotoxicity. Lipid solutions can directly counter local anaesthetic toxicity by creating a lipid compartment within the plasma that attracts lipophilic compounds such as local anaesthetics, thereby separating them from the aqueous phase of the plasma[1].

Small animal local anaesthetic techniques

Currently local anaesthetic techniques are often neglected as part of small animal anaesthetic and analgesic regimens. However use of a local technique can often reduce the dose of other anaesthetic drugs required for maintenance of anaesthesia and contributes to a multi-modal analgesic technique. Use of specific nerve blocks to prevent the relay of nociceptive information from the site of injury to the spinal cord can also provide pre-emptive analgesia, and prevent or reduce the development of central sensitization.

Key points:

- Calculate the safe maximum dose and do not exceed this dose. If more volume is needed for wider distribution of the drug then dilute the local anaesthetic with sodium chloride.
- Use appropriately sized needles and syringes. This will minimise tissue trauma during injection, while using a suitably sized syringe will allow accurate dosing.
- Avoid inadvertent intravascular injection by drawing back on the syringe and checking that no blood is aspirated before injection.

Local anaesthetic / analgesic techniques

Topical anaesthesia (transmucosal): The application of some local anaesthetic drugs to mucous membranes produces analgesia rapidly (within 5 minutes). Common areas for the topical administration include the cornea for ocular examinations, the nasal passages (e.g. prior to placement of a nasal oxygen cannula and the larynx during intubation. The depth of analgesia produced in tissues is usually small (1 – 2mm).
The absorption of local anaesthetic through the skin (stratum corneum) is generally poor. However EMLA cream (a mixture of lidocaine and prilocaine) is capable of producing anaesthesia if applied to skin and covered with a non-permeable dressing for 30 – 60 minutes. This is useful to provide local analgesia prior to intravenous catheter placement in cats and dogs.

Infiltration anaesthesia: The infiltration of local anaesthetics is commonly performed in veterinary practice; it is safe, reliable and does not require extensive experience. Sterile sharp needles should be used. For example local anaesthetics can be infiltrated along the incision line after abdominal surgery or prior to hemilaminectomy[2].

Regional Anaesthesia: Use of a nerve stimulator or ultrasound to locate peripheral nerves can significantly increase the accuracy of drug deposition and therefore effectiveness of the block. It can also allow a lower total volume of the local anaesthetic drug to be used, reducing motor side effects and the risk of toxicity due to absorption of local anaesthetic agents into the systemic circulation.

e.g. Brachial plexus block (loss of sensation and motor function distal to the elbow)

Epidural anaesthesia: Lumbosacral epidural anaesthesia may be used to provide analgesia for all procedures caudal to the thoracolumbar junction. It is especially useful for orthopaedic procedures in the hindquarters.

Dental nerve blocks: Maxillary and mandibular nerve blocks are extremely useful to provide analgesia for dental procedures or mandibular or maxillary surgery. The maxillary and mandibular nerves can be blocked as they exit from the infra-orbital and mental foramens respectively or they can be blocked more proximally to provide a wider area of analgesia.

Intraperitoneal disposition of local anaesthetics: The WSAVA-GPC guidelines recommend IP and incisional

anaesthesia for the management of pain, particularly as adjuvant techniques for pain relief in dogs and cats undergoing abdominal surgery[3]. Both techniques are described and demonstrated in the following link (https://www.youtube.com/watch?v=76dwKuirqt0).

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APPROACH TO THE INAPPETENT CAT

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ABSTRACT BODY:

Introduction

Cats with disease are prone to malnutrition for a variety of reasons, including inappetence. This can be exacerbated during stress situations, such as hospitalization. Hospitalized cats may have reduced appetite (1) even though they have access to food. In some cases, the inflammation associated with their disease can promote a hypermetabolic state, which can result in loss of both fat and lean mass, resulting in accelerated malnutrition, which can affect prognosis.

Malnutrition has negative consequences for recovery and should therefore be prevented/treated in all cases. The ISFM has recently published a set of consensus guidelines on how to manage inappetent hospitalized cats (2). More detailed information on this topic can be found there.

The guidelines are open access and include a variety of supplemental material, including videos on the placement of naso-oesophageal and oesophagostomy feeding tubes, example forms (diet history for cat caregivers, hospital feeding records) and information for caregivers regarding managing the cat with low appetite or with a feeding tube in place.

Causes of inappetence

Reduced feed intake is not always related to inappetence: cats might want to eat, but not be able to due to functional issues (e.g., jaw fracture) or conditions associated with dysphagia like oral disease.

Inappetence, however, is the main reason for reduced food intake during hospitalization, such as disorders associated with nausea, ileus, pain, or stress. Some diseases can affect appetite via inflammation. Dehydration, constipation, and side effects of medication/treatment should also be considered. It is important to try to identify the cause(s) of inappetence in each case, in order to tailor the treatment. For example, in nausea, the treatment will include addressing the underlying cause and antiemetics, while analgesia is indicated when pain is suspected.

Management of inappetence

First, all aspects of the management must be assessed to ensure that they do not affect appetite, such as medication choice and delivery route and avoiding offering unfamiliar diets or force feeding. Stabilizing the patients (re-hydration, correction of electrolyte and acid-base unbalances, etc.) before feeding them is also important. Reducing stress is also important, and that might require familiar litter, noise reduction, comfortable housing, and areas to hide, amongst others.

Establishing a feeding plan

All hospitalized cats should receive a nutritional assessment (3) in order to design a feeding plan, which will include answering what to feed (diet), how much (allowance) and how (method). The assessment collects information on signalment, history (medical and diet) and physical exam, including weight, body condition (BCS) and muscle condition (MCS) scores. Bloodwork can also provide some clues, but any changes are non-specific and not very sensitive. The information taken together can be used to classify cats in malnutrition risk group: low, moderate or high (4). This will allow for patient selection, identifying those cats that require nutritional support or assisted feeding as early as possible. Overall, main reason for intervention is 3 days of anorexia, taking also into account the days previous to admission.

The preferred feeding route is orally, and there are some interventions that can be done to promote voluntary intake mentioned above (like stress reduction, providing a palatable diet, ensuring adequate hydration, etc.). Adapting to the cat's feeding behaviour will help us feed them in a "cat friendly" manner. Asking caregivers what the cat usually likes is also important. Appetite stimulants such as mirtazapine can also be considered, these can be given while working up the causes of the inappetence, close follow up of food intake, weight, BCS and MCS is needed to move on to assisted feeding if they are not enough to promote adequate calorie intake. In general, these are more indicated in hyporexia or short-term use (while working up the cat) but in some situations like critically ill patients or those unable to eat, they are not the best choice.

If oral feeding is not sufficient to maintain a stable weight, feeding tubes are the next options, the most used are naso-oesophagic, NO, (or naso-gastric, NG) and oesophagostomy tubes (O tubes). NO and NG are less invasive, plus do not require anaesthesia for placement, but are only indicated for short term with liquid diets. O tubes are invasive and required short anaesthesia for placement but can be used long term and allow for a larger choice of diets. Gastrostomy

tubes are also an option, especially for longer term use or in patients with oesophageal disease. In the few cases where enteral feeding is not possible/tolerated, parenteral (iv) nutrition is the next option, but enteral feeding is preferred if at all possible, in order to maintain intestinal integrity.

Diet choice

The diet should meet the feline nutritional requirements. Cats are strict carnivores, which results in some nutritional particularities that makes them different than dogs, such as having a high protein (nitrogen) requirement and having to receive certain nutrients in the diet that are abundant in animal tissue, such as pre-formed vitamin A (retinol), arachidonic acid, cholecalciferol, and taurine (5). This is hypothesized to be due to cats evolving eating pray (6). If a feeding tube is in place, convalescence diets (liquid or slurry) are a very practical choice, unless contraindicated.

Allowance

The amount of food provided depends on the energy needs of the patient. Energy needs of cats can be estimated using formulas (5,7) and vary depending on life stage and other factors such as neuter status and activity level. For hospitalized cats, energy requirements are estimated to be closer to their RER (resting energy requirements) to approximate their basal metabolic needs for cats in cage rest. The RER should be calculated using current body weight. The RER can be divided by the energy density of the diet of choice to obtain the daily allowance (in g or ml per day).

Once 100% RER is reached (over a few days), the allowance can be adjusted in 10% intervals to achieve our weight goals, since energy needs are variable and different diseases might result in higher or lower energy expenditure. Usually weight maintenance is the goal, but weight gain can be considered in stable animals with low BCS and in growing kittens.

Feeding schedule

If fed orally, divide the allowance over several meals and start slow.

For tube fed cats, start slowly (at 25-33% of RER per day) and divide the allowance over 3 to 6 boluses per day, depending on tolerance, calorie goals, and personnel availability. With liquid diets, a constant rate infusion can also be considered, especially helpful if there is volume intolerance.

Follow up

The follow up includes the specific one for the disease(s) plus those needed during hospitalization and the nutritional assessment, including serial weights, BCS, MCS and a tight record of food intake (orally or via feeding tube). The food intake must be compared to the RER and evaluated together with weight trends. We also need to look out for tube feeding complications.

Once the cat is feeling better, we can start offering food orally before each tube feeding. The tube can be removed once the cat is eating the RER consistently for 3-5 days.

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039 / #47

DO NO HARM. WHEN GOOD DIET INTENTIONS GO WRONG

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ABSTRACT BODY:

INTRODUCTION

Pet food recalls, human diet and nutrition trends, and a growing appreciation for the role of diet in health and disease have led pet owners to seek out diets they believe will "optimize" the health of their companion animals. Feeding a complete and balanced diet is known to promote wellness in dogs and cats, but it can be challenging for pet owners to evaluate their options in the face of marketing efforts by manufacturers and retailers. Many owners, and some veterinarians, will advocate feeding home-prepared foods exclusively (raw or cooked, or both) and either cite perceived health benefits or a general mistrust of the pet food industry. Home-prepared diets can have multiple nutrient deficiencies if not formulated correctly and these deficiencies can have long-term and potentially fatal consequences for individual dogs or cats, not to mention the risk that food-borne pathogens pose to pets and people. It is important for veterinarians and supports staff to understand the benefits and risks of any given diet type and to help ensure that, no matter the diet selected, the nutritional needs of the individual animal are being met.

BASIC NUTRIENT REQUIREMENTS FOR DOGS AND CATS

Despite both dogs and cats being classified into the order Carnivora, cats are obligate carnivores while dogs are adapted to an omnivorous diet. As such domestic dogs are able to thrive on a wide variety of plant and animal-based ingredients, from more heavily meat-based to completely vegan, while cats require animal-based ingredients in their diets. As evidence of this nutritional flexibility, dogs are able to taste sweet; can synthesize adequate amounts of taurine from the sulfur amino acid precursors methionine and cysteine; can split b-carotene found in fruit and root vegetables into two vitamin A subunits; and have the ability to convert linoleic acid (found in high concentrations in plant seeds and grains) into arachidonic acid. Cats in contract lack all of these metabolic features.

Despite differences between dogs and cats, they have similar requirements for specific essential amino acids, essential long-chain fatty acids, macrominerals, trace minerals, water-soluble vitamins, and fat-soluble vitamins. Both dogs and cats can synthesize vitamin C in the liver; neither species can synthesize vitamin D from UV exposure to skin; and both lack salivary amylase for the initial step in carbohydrate metabolism but have pancreatic amylase activity and glucose receptors on enterocytes for further carbohydrate utilization. Altogether dogs require 37 essential nutrients in their diets and cats require 41 and it is important that the diet selected and fed on a regular basis meets these ongoing nutritional needs irrespective of form.

COMMERCIAL DIETS

Complete and balanced commercial diets (dry, moist, pasteurized, or raw-meat based frozen or dehydrated) are designed to be fed as a sole source of nutrition to dogs and cats and include adequate amounts and ratios of all essential nutrients. Nutrient levels are either formulated on an energy basis (nutrient per 1000 kcal of diet) or on a Dry Matter basis (nutrient per kg of food). Pet food formulation and labeling requirements may vary by region, but most countries have adopted guidelines recommended by the European Pet Food Industry Federation (FEDIF) and the Association of American Feed Control Officials (AAFCO). FEDIAF and AAFCO have established model guidelines for the countries and states regarding pet food labels, ingredient definitions, what can and cannot go into pet foods, and levels of specific essential nutrients required for a given life stage. Any pet food with a FEDIAF or AAFCO label of adequacy must have met these guidelines, though it is up to individual nations or states to regulate and enforce these recommendations. Commercial diets will then be labeled as having gone through "feeding trials" to ensure nutrient adequacy or as having been "formulated" to meet these requirements. Commercially produced fresh-food diets (raw or cooked) still must meet industry labeling requirements and any diet or brand lacking this basic, required information should be viewed with concern.

RISK OF UNBALANCED AND RAW MEAT DIETS

Home-prepared diets have grown in popularity over the last few years and so has the frequency of nutrient-deficient diseases. Conditions such as pansteatitis[1], hypo-[2] and hypervitaminosis D[3], and thiamine deficiencies[4] are being seen more frequently in clinical practice. Proponents of home-prepared foods claim that these diets are a safe and natural way to feed animals yet published reviews on the nutritional

adequacy of home-prepared (cooked and raw) diet recipes in recent years found that less than half of the recipes used by pet owners provided a complete and balanced source of nutrients.[5-7] Unless someone with veterinary nutrition training developed the diet, there are a few consistent deficiencies found in most home-prepared recipes irrespective of the ingredients used. Most home-prepared diets for dogs and cats are lacking a sufficient source of calcium; a source of trace minerals (such as iodine, selenium, copper, and zinc); a source of linoleic acid; and a source of essential fat-soluble and water-soluble vitamins.

Fresh meat whether it is fed raw or cooked is palatable to most dogs and cats, can be highly digestible, and depending on the cut of meat selected is higher in fat than most dry kibbles. The result is an animal that readily eats its food, has low stool volume, and has a shiny coat. These positive aspects are often held up as "proof" of nutritional superiority to commercial foods and these perceived benefits are reinforced daily to the owner, while the potential negative consequences are largely ignored. Nutrient deficiencies in adult animals are insidious and can lead to long-term complications, which can vary from poor skin and coat health to chronic diarrhea, osteopenia, anemia, altered drug metabolism, and hepatic lipidosis depending on the specific nutrients lacking in the diet.

The negative aspects of raw meat diets can be disastrous not only for the animals but the people in the household as well. Any raw meat ingredients can be a potential source of parasitic and bacterial exposure, which can include *Neospora*, *Toxoplasma*, *Salmonella*, *E. coli*, *Campylobacter*, and *Cryptosporidium*;[8-10] raw and cooked bones specifically carry a risk of gastrointestinal obstruction/perforation and oral trauma;[11] and are a poor source of essential minerals (such as calcium, phosphorus, and magnesium) due to the poor digestibility of larger bones within the canine and feline digestive tract.[12] Ultimately, the animal's acceptance of a home-prepared diet does not change significantly when the meat is cooked or when more bioavailable sources of nutrients are used.

SUMMARY

Individual animals may vary in their response to specific commercially-available diets and there is no one diet-that-fits-all. If an owner elects to feed a home-prepared diet they should be counseled on the risks of this feeding strategy and cautioned that nutritionally-related disease can mimic other forms of chronic illness. Any animal eating a home-prepared diet should have at least an annual physical exam and health screening, including serum biochemistry (with T4), hematology, and urinalysis profiles. While blood work and urinalysis results will give you a general overview of the animal's health status, they will not pick out specific deficiencies or excesses. A complete diet history (all foods and supplements) should be collected from the owner at each visit. Any home-prepared diet recipes should be obtained from a reputable, trained source.

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MINIMUM STANDARDS FOR NEUTERING IN WELFARE PROJECTS

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ABSTRACT BODY:

Control of reproduction via sterilisation is an important method to humanely manage dog and cat populations.[1,2] Surgical sterilisations are widely performed in shelters and rescue organisations, as well as projects targeting pets who are unlikely to be presented to a veterinary clinic[3] or targeting free-roaming animals in large scale Trap-Neuter-Release programmes.[4] All surgical sterilisations should aim to achieve the best possible outcomes for the animals. That means lowest morbidity and mortality from the procedure. On top of that, the entire procedure should be least stressful for the animals throughout the entire procedure from the start, including capture and handling, to the end when the animal is returned to its rightful location. Surgical sterilisation done in welfare projects such as high-volume spay neuter clinics may not achieve the ideal practices in private practices to assess and reduce the risks associated with sterilisation. These limitations can be due to the limited access to the animal as it may only be caught once, limited handling without sedation for some animals, and the use of limited resources to maximise its benefits. It is very important to weigh the risks each of these limitations with the benefits of sterilisation for the individual animal and ensure that its health and welfare are not compromised.

Basic minimal standards applicable to sterilisation in all situations are as follows:

Analgesia

All surgical procedures are painful and provision of adequate analgesia is mandatory for every surgery. The choice of the analgesic protocol depends upon the age and health of the patient, degree of surgical trauma, and availability of various analgesics. If an accurate body weight cannot be obtained before drug administration, safety is increased by using reversible agents, avoiding drugs that result in marked cardiorespiratory depression, and estimating body weight as accurately as possible.[5] Analgesia should begin before surgery, be maintained throughout surgery, and continue for a period of time after surgery. Multimodal analgesia is recommended whenever possible because analgesics (non-steroidal anti-inflammatories (NSAIDs), opiods, local anaesthetics) work synergistically to control pain, usually with fewer side effects than when single analgesics are used, and with improved analgesia. Patient hydration status and the presence of preexisting hepatic, renal, or gastrointestinal disease or clotting abnormalities should be considered when using NSAIDs. Local anaesthetic techniques are particularly appropriate as they may provide excellent analgesia without causing systemic side effects.[6]

Anaesthesia and perianaesthesia monitoring

General anaesthesia is required when performing surgical sterilisation in dogs and cats. General anaesthesia involves the administration of a combination of drugs in order to safely produce analgesia, loss of consciousness, muscle relaxation, and immobility without patient compromise. The choice of anaesthesia should be tailored to the individual patient based on any risks identified during a pre-anaesthetic evaluation as well as the availability of anaesthetic drugs and equipment.[7-8] Anaesthetic premedication should achieve sedation and stress reduction, safe handling, analgesia, balanced anaesthesia and calm recovery.[8] Anaesthesia can be induced and maintained with either an injectable anaesthetic agent or inhalant gas or a combination of the two.Clinical observation and assessment by a vigilant anaesthetist are essential for safe patient care from the induction of anaesthesia to recovery.[8] Patient monitoring should be performed by skilled and well-trained person and should not be the person performing the procedure. The minimum monitoring standards of an anaesthetized patient include assessing and accurate recording of physiologic parameters (e.g., heart rate, respiratory rate).[8] Clinical monitoring can be supplemented with continuous monitoring devices that measure cardiac rhythm, blood pressure, arterial oxygen saturation, body temperature, etc.

Asepsis

Single-use sterile surgical gloves are a minimum requirement for all abdominal surgeries and for the castration of adult dogs. For routine cat and puppy castration, either single-use sterile gloves or examination gloves are acceptable. Routine perioperative use of antimicrobials is not recommended. Antimicrobial use should be reserved for specific indications, such as pre-existing infection

(e.g., pyometra) or a when a break in surgical asepsis occurs.

Good surgical techniques

Using good surgical techniques (following Halsted's principles) will optimize surgical success while decreasing the risk of complications. When surgical practices are efficient, surgery times are shorter and patient recovery is improved.In order to reduce postoperative morbidity, surgeons should strive to reduce surgical trauma by using gentle tissue handling techniques, control hemorrhage, preserve blood supply, minimize tissue tension, and eliminate dead space.

Post-operative monitoring

After recovery, the post-operative care will transition to the animal caretakers. Clear, written instructions should be provided regarding the post-operative care. Post-operative care generally includes monitoring for signs of pain or discomfort, care and monitoring of the surgical incision, offering food and water, ensuring necessary exercise restriction, and administering medications.

Below are some additional considerations[5] for sterilisation in welfare projects:

Surgical area

Aside from stationary or mobile clinics, a temporary *MASH* style clinic may be set up in a location more convenient for access to the target animals e.g. in rural areas. These clinics can be set up in public or private spaces large enough to accommodate the number of surgeries, such as community halls or gymnasiums. No matter the location, the clinics should be in a clean environment, have access to water and electric or adequate lighting, and where foot traffic can be controlled.

Transport, handling and restraint

Feral or free-roaming animals unaccustomed to human contact may find any type of restraint or handling highly stressful. Different methods of capture for these animals may be employed, depending on situation, catcher ability, and equipment available. Methods of capture can include hand capture, netting, trapping or chemical capture. Regardless of the methods used, the manner that causes the least amount of stress for the animal should be selected and the entire process from capture to transport shall be kept as short of a duration as possible.

Physical examination

Some of the more fearful animals may be brought into the clinic via a trap. Since human contact with these animals will increase the fear and distress, it may not be feasible to adequately conduct a physical examination prior to sedation or anaesthesia. Veterinarians will need to assess suitability for surgery via visual examination and history from the caregivers before sedation, and then a more thorough physical examination can be done after sedation or induction of anaesthesia.[2]

Blood work

Pre-anaesthetic blood work may not be feasible for all animals undergoing sterilisation. A vet should use their discretion to decide if further diagnostic tests are warranted based on the physical examination and available patient history.

Animal identification

To reduce the risks of misidentification and unnecessary surgery, it is recommended that all animals are marked with a permanent identification. This can include microchipping if costs permit, or low-cost methods such as tattooing or ear notching/tipping. The latter methods allow for visual identification of the animal from afar, thereby also decreasing the unnecessary stress of undergoing capture and handling.

Discharge of patients

As with any surgical patient, they can be discharged to the caregiver once the animal has recovered from the anaesthesia, is alert, responsive and at a minimum at sternal recumbency. Incision site and pain should be assessed, and whenever possible, assess mucous membrane and respiration as well before discharge. Prolonged confinement can be very stressful for animals and may affect recovery, and one must balance the need for confinement with safe post operative recovery. Most free-roaming animals can be safely released to its original location the day after surgery. Caregivers can be utilised to keep monitoring these animals post operatively. The veterinarians in charge must use their discretion to decide on what is best for the patient. Risks must be weighed against the benefits of sterilisation and the overall objective of the welfare project. In order to decrease morbidity and mortality, good record keeping and data analysis are essential to ensure continuous refinement of the entire process.[4]

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MINIMUM STANDARDS FOR NEUTERING IN WELFARE PROJECTS

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ABSTRACT BODY:

#261 MINIMUM STANDARDS FOR NEUTERING IN WELFARE PROJECTS

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CUYES EN PERÚ COMIDA O MASCOTAS?

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ABSTRACT BODY:

Introducción - Algo de historia del cuy en el Perú

Los cuyes (*Cavia porcellus*) son una especie que en el Perú han sido domesticados para ser animales de producción cárnica desde épocas precolombinas, siendo muchas veces una alternativa para la Seguridad alimentaria del poblador altoandino, selvático y costeño. Hay datos que muestran a los primeros criadores en Pichimachay (Ayacucho) y Telarmachay (Junín) a más de 4000 msnm ([1]). Culturalmente se han visto cerámicos e incluso pinturas que incluían al cuy como parte de la cotidianeidad y cultura popular del poblador peruano y latinoamericano como es el caso de "La última cena" de Marcos Zapata de 1753, que representa a la tradicional última cena de Jesucristo con sus discípulos con un plato de comida en base a cuy en el centro de la mesa ([2]).

Según la Resolución Ministerial N° 0338-2013-MINAGRI del año 2013 el segundo viernes del mes de octubre de cada año ha sido declarado como el "Día Nacional del Cuy" como una forma de promover su crianza y consumo.

Generalidades de la especie

Referido a los cuyes tenemos que son animales cuyos antecesores silvestres son Cavia tschudii and Cavia aperea según los estudios filogenéticos. Aunque hay mucha controversia respecto a su origen, considerando el lugar de donde provengan se pueden postular cruces entre los cuyes primigenios. ([3]). Otro punto interesante viene a ser las posibles clasificaciones de los cuyes en Fenotipos, razas, líneas, etc. Es sabido que nuestros cuyes pertenecen a tres razas Perú, Andina e Inti, y en los últimos años se obtuvo una raza sintética conocida como Raza Kuri. Para esto, en Perú, múltiples investigaciones realizadas en INIA o Universidades como la UNALM o la UNMSM han tratado de esclarecer estos problemas y fortalecer a los criadores de cuyes mediante trabajos en muchos aspectos de la crianza de esta especie. Por lo general se consideran las razas en base a la conformación del cuerpo, largo y color del pelaje, sin dejar de lado su prolificidad, precocidad y producción cárnica ([4]).

La *American Cavy breeders Association* ([5]) considera las siguientes razas: *Cuy Abisinio, Cuy Abisinio satinado, Cuy Americano, Cuy Americano Satinado, Cuy Coronet, Cuy Peruano, Cuy peruano satinado, Cuy Silkie, Cuy Silkie Satinado, Cuy Teddy, Cuy Teddy satinado, Cuy Texel y cuy White Crested*

El cuy como animal de producción cárnica

La carne de cuy según las Tablas peruanas de composición de los alimentos de 2017 ([6]) contiene 96 Kcal de energía (402 kJ) 78.1 g de agua, 19 g de proteínas, 1.6 g de grasa, 0.1 de carbohidratos totales y 0 de fibra dietaria. Lo cual conlleva a que sea una fuente alimenticia muy sana. Y versátil para la preparación de distintos platos.

En Perú ([7]) la producción de animales menores incluye a la producción avícola, porcícola, cavícola y cunícola, distribuida entre pequeños y medianos productores y los grandes productores. Para el caso de los criadores pequeños y medianos de cuyes se asume que para los años 2015 y 2016 fue de 16520 y 17168 (en miles de animales) respectivamente cuya localización está centrada principalmente en la sierra. Siendo el mayor porcentaje los pequeños y medianos productores.

Es interesante el saber que la crianza comercial del cuy como animal cárnico ha sido llevada a países en otros continentes, es el caso de países africanos como Senegal, Sub-Sahara, Camerún, etc., donde la crianza es muy similar a las crianzas familiares pequeñas de hace unas décadas del Perú y ha servido para la Seguridad alimentaria y una importante actividad de apoyo a la economía familiar (8)

Gastronomía

Siendo principalmente un animal de abasto para el poblador peruano, hace que sea parte de su gran culinaria, conocida mundialmente y que hace al Perú uno de los principales destinos gastronómicos del mundo. Teniendo diversas preparaciones típicas como "Cuy chactado", "Picante de Cuy", "Cuy frito", "adobo de cuy", "Cuy a la brasa", etc. Los cuales son muy requeridos por los comensales nacionales y extranjeros, y no solo por su sabor, sino también por su gran valor nutricional y propiedades curativas que se le atribuyen como antineoplásico, reconstituyente frente a enfermedades muy demandantes como lo es el COVID 19, siendo principalmente su preparación como caldo de cuy.

Investigación

Es innegable el gran aporte que los cuyes han dado a la ciencia, siendo considerado por excelencia como uno de los principales animales de laboratorio de las décadas pasada. Pero actualmente se vienen realizando en muchos ámbitos trabajos que están

orientados a las ciencias básicas, para verificar su morfología y fisiología, como en ciencia aplicada, ya sea en reproducción, nutrición y en las enfermedades mas frecuentes en ellos, en especial en animales de producción. Lo cual es muy promisorio para mejorar las condiciones de bioseguridad en las crianzas comerciales.

Mascotas

En otras latitudes, los cuyes, también conocidos como "Cobayos", son una mascota muy popular, y para los médicos veterinarios de exóticos puede representar un porcentaje alto de su clientela. Siendo actualmente en Perú aun una especie que no ha entrado como una mascota del todo, como si lo han hecho los Conejos, hurones y erizos, cuya frecuencia en consulta no ha sido aun calculada oficialmente, pero si constituye un gran porcentaje. Esto posiblemente debido a que culturalmente el peruano tiene la percepción del cuy como un animal cárnico.

El cuy en rituales andinos

También es sabido que los cuyes en muchos países como el nuestro son utilizados dentro de rituales, este es el caso del conocido "jubeo" que es un término que se relaciona a "sanar a un enfermo frotándole el cuerpo con un cuy" según lo que indica la Asociación de academias de la lengua española, realizado por los chamanes o curanderos en muchas partes del Perú y Latinoamérica y al que se le atribuyen curaciones espontáneas de las personas que son sometidas a ese procedimiento ritual. Y que se acompaña del sacrificio del animal y "necropsia".

Principales enfermedades: Infecciosas, parasitarias, reproductivas, otras.

Los cuyes de producción tienen en Perú debido a las aun no del todo establecidas medidas de bioseguridad, en especial en granjas medianas y grandes problemas sanitarios infecciosos como lo es la Salmonelosis, Colibacilosis, Linfadenitis cervical, etc. Parasitarios como coccidiosis, nematodiasis, piojeras, pulgosis, acarosis y sarna. Dermatomicosis y otras dermatitis de causas variadas. Además de presentar problemas derivados de la falta de asesoramiento en la crianza y reproducción, de ahí que sean frecuentes las distocias, torsiones gástricas, etc. ([9])

Conclusiones

Los cuyes en Perú participan en múltiples ámbitos, desde su crianza comercial para mantener la seguridad alimentaria de los pobladores (en especial los pequeños y medianos criadores altoandinos), además que ya está expandiéndose su crianza con este fin a otros continentes. Es una especie que ya nos viene acompañando desde muchos años como animal doméstico, y por sus características intrínsecas ha sido también utilizado en la investigación biomédica, en medicina tradicional, rituales por curanderos y como mascotas.

Para países de Latinoamérica como el Perú el crecimiento de su tenencia como mascotas va en aumento, para lo cual debemos estar preparados explorando aun más la medicina veterinaria en esta especie.

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HEMATOLOGICAL CHANGES ASSOCIATED WITH TICK-BORNE DISEASES

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ABSTRACT BODY:

Rocky Mountain Spotted Fever

Rocky Mountain Spotted Fever is caused by *Rickettsia rickettsii*, an obligate intra-cellular bacterial organism. The organism is transmitted by *Dermacentor variabilis*, *D. andersoni*, *Rhipicephalus sanguineus*, and *Amblyomma cajennense*. Within the body the organism invades and replicates in the endothelial cells of small arteries and venules, resulting in a necrotic vasculitis with subsequent activation of platelets and the coagulation system. A mild-to-moderate normocytic normochromic anemia occurs with the disease. During the early course of the disease mild leukopenia develops that is followed by moderate left shift neutrophilic leukocytosis. Toxic neutrophils may also be present. The most consistent hematological change is severe thrombocytopenia with megathromocytes often evident. The most common coagulation abnormality is prolongation of the ACT, however, severe cases may show DIC with prolongation of PT, PTT, TT, and elevated FDP and D-dimer. The diagnosis requires identification of antigen by direct immunofluorescent in tissues, serologic testing, or PCR.

Feline Hemoplasmosis

Hemoplasmosis is caused by *Mycoplasma*, of which currently there are 3 species, namely *M. haemofelis*, *M. haemominutum*, and *M. turicensis*. The disease was previously referred to as hemobartonellosis. *Rhipicephalus sanguineus* ticks transmit the organisms.Cats infected with *M. hemofelis* typically show regenerative anemia, whereas those infected with *M. hemominutum* may show no hematological changes. Leukocyte changes with *M. hemofelis* are inconsistent. Thrombocytopenia occurs with *M. hemofelis* infection but not with *M. hemominutum*. During the acute phase of the disease, the cat may become anemic (hematocrit < 20% and even < 10%). The anemia is usually associated with a febrile reaction and often preceded by a short-lived increase in leukocyte count. Most cats will show a positive Coombs' positive test and often, there is macroscopically detectable autoagglutination (occasionally shown to be associated with cold agglutinins) and usually a microscopically detectable autoagglutination. The decrease in the erythrocyte size has been attributed to increased osmotic fragility and decreased erythrocyte life span. Erythrophagocytosis can be evident in peripheral blood, bone marrow and spleen. *Mycoplasmosis* can morphologically be diagnosed on Romanowski-stained blood smears. The organisms occur as blue staining, small rings (0,5mm), cocci or rods attached to the erythrocyte membrane. The organisms may occur singly, in clusters and in chains. In recent years, PCR techniques have been become the diagnostic "gold standard" for the diagnosis of *Mycoplasmosis*.

Canine Mycoplasmosis

Mycoplasma hemocanis is an opportunistic pathogen that can cause a mild anemia in dogs that are either splenectomized or immune-suppressed. The anemia can vary from mild to severe and the disease is usually detected when an animal is examined for other concurrent disease. The anemia is typically regenerative and often of a hemolytic nature. In some dogs, spherocytosis and a positive Coombs' test may be evident. Leukocyte and thrombocyte changes are inconsistent.

Canine Monocytic Ehrlichiosis

Ehrlichia canis morulae are found in monocytes and macrophages as a "micro-colony" of bacteria surrounded by a membranous vacuole. Morulae may contain 100 or more ehrlichial organisms resembling elementary bodies of chlamydiae. *Ehrlichia canis* is transmitted by *Rhipicephalus sanguineus*.

The typical hematological changes include anemia, leucopenia, and thrombocytopenia. Infection may result in either a regenerative or non-regenerative anemia. In the acute disease although the anemia is due to destruction of progenitor and proliferative cells in the bone marrow, bone marrow is usually hypercellular suggestive that there is also cell destruction in the peripheral blood. As the erythrocyte lifespan is long, the anemia is usually mild or absent. The organism can also trigger a secondary immune mediated hemolytic anemia. In the chronic phase of the disease pancytopenia is evident as a result of stem cell injury with an associated hypocellular bone marrow, which appears to be more common in the German shepherd dog. Lymphocytosis has been reported to occur with ehrlichiosis and may mimic lymphocytic leukemia, especially if there is also an associated monoclonal gammopathy on serum protein electrophoresis. The

thrombocytopenia and thrombocytopathy that occurs in the acute phase of the disease is associated with both immunologic and inflammatory mechanisms which results in increased platelet consumption and reduced platelet life span. Reported immunologic mechanisms are platelet associated antibodies and platelet migration-inhibition factor.

Canine Granulocytic Ehrlichiosis

The causative organism of canine granulocytic ehrlichiosis is *E. ewingii*, which is responsible for the disease in people. Transmission is by *Amblyomma americanum*.

Hematological changes are include nonregenerative anaemia, thrombocytopenia, lymphopenia, and eosinopenia.

Human Monocytic Ehrlichiosis

Although *E. chaffeensis* is the causative agent of human ehrlichiosis it can also infect dogs. Transmission is by *Amblyomma americanum*. The majority of cases have been with experimentally infected animals, with thrombocytopenia being documented.

Canine Granulocytic Anaplasmosis

Canine granulocytic anaplasmosis is caused by *A. phagocytophilum*, which results in non-regenerative mild to moderate normocytic normochromic anemia, occasionally regenerative anemia, lymphopenia, neutropenia, occasional left-shift neutrophilia, and thrombocytopenia.

Canine Thrombocytotrophic Anaplasmosis

Anaplasma platys infects canine thrombocytes and results in cyclic, moderate to severe thrombocytopenia. Transient leukopenia and mild to moderate normocytic normochromic non-regenerative anemia anemia may also be associated with the disease.

Babesiosis

In the dog babesiosis is caused by *Babesia canis*, *B. vogeli*, *B. rossi*, and *B. gibsoni* (North American and Asiatic stain); whereas in the cat multiple species of *Babesia* organisms have been documented. Other *Babesia* species affecting both cats and dogs have been reported. Infection typical results in a regenerative anemia due primarily to erythrocyte destruction by the parasite itself or as a result of a secondary immune mediated hemolytic anemia. *B. canis* is transmitted by *Dermacentor reticulatus; B. vogeli* and *B. gibsoni* by *Rhipicephalus sanguineus; and B. rossi* by *Haemophysalis elliptica*. *B. gibsoni* can also be transmitted by bites. The primary hematological abnormalities in canine babesiosis are anemia, thrombocytopenia, and leukocytosis. Hematological profiles, however, vary in different parts of the world: mild anemia and thrombocytopenia has been reported in France; in the Philippines, only mild anemia; and in Nigeria in peracute cases severe anemia and leukocytosis and mild-to-moderate anemia in the acute and chronic cases. In South Africa severe anemia, left shift neutrophilia, monocytosis, eosinopenia, and thrombocytopenia are the typical hematological changes evident with canine babesiosis. These variations in the hematological profile is most likely due to various strains of *Babesia canis*. The anemia in canine babesiosis is initially mild, normocytic and normochromic that then becomes macrocytic, hypochromic, and regenerative as the disease progresses. The reticulocytosis is proportional to the severity of the anemia. Leukocyte abnormalities are inconsistently observed but may include leukocytosis, neutrophilia, neutropenia, lymphocytosis, and eosinophilia. In-saline agglutination of erythrocytes is a common finding and a positive Coombs' test occurs in the majority of cases, making a diagnosis of immune mediated hemolytic anemia difficult. Moderate to severe thrombocytopenia is common. Uncommonly reported hematological changes are a relative polycythemia with normal plasma protein concentration and a leukemoid response. In feline babesiosis the anemia is typically macrocytic, hypochromic, and regenerative. There is no characteristic change in total or differential leukocyte counts and the thrombocyte count is usually normal with thrombocytopenia an inconsistent finding. The in-saline agglutination test may also be positive. The diagnosis is made by demonstrating *Babesia* organisms within infected erythrocytes on a blood smear stained with a Romanowski-type stain. Large (2x5mm) pear-shaped organisms usually present in pairs are indicative of *B. canis* infection, whereas smaller (1x3mm), singular, round to oval organisms are *B. gibsoni*. *Babesia felis* parasites are approximately 1/3 of the size of *B. canis* and various shapes can be seen. PCR has become the gold standard for detecting occult parasitemias and characterizing the parasites.

Cytauxzoonosis

An infrequent but usually fatal, disease of domestic cats caused by the theileria-like protozoan *Cytauxzoon felis*. The vector of the disease is thought to *Dermacentor variabilis*. Infection results in mild to moderate non-regenerative anemia as a result of peracute erythrocyte destruction. Differentiation needs to be made between *C. felis* and the smaller feline parasites: *Babesia felis* and *Mycoplasma hemofelis*. The primary hematological abnormalities are mild to moderate normocytic normochromic non-regenerative anemia, mild to marked leukopenia with neutropenia and band neutrophilia, and marked thrombocytopenia. The diagnosis of *C. felis* is made by demonstrating

organisms within infected erythrocytes on a blood smear, histological identification of schizonts in tissues, or PCR.

Hepatozoonosis

Hepatozoonosis is a protozoal parasite that only infects dogs once they have ingesting ticks that contain *Hepatozoon* sporozoites. Once ingested they penetrate the intestinal wall and form macromerozoites, which then penetrate into skeletal muscle, myocardium, lungs, lymphoid tissue, and liver. From here micromerozoites are formed that then enter the leukocytes. There are two types of the disease, the one caused by *Hepatozoon americanum* (transmitted by *Amblyomma maculatum*) and the other by *H. canis* (transmitted by *Rhipicephalus sanguineus*). The former is a more virulent form of the disease. Hematological changes that occur with *H. americanum* infection are left shift neutrophilic leukocytosis, eosinophilia, thrombocytosis, and mild-to-moderate regenerative anemia. Approximately 1% of peripheral leukocytes contain gametocytes. Hematological changes that occur with *H. canis* infection are variable neutrophilic leukocytosis and mild regenerative anemia. A large percentage of peripheral leukocytes contain gametocytes (60-90%). Diagnosis is based on the identification of gamonts in neutrophils or monocytes on Romanowski-stained blood smears or by the demonstration of organisms in muscle biopsy sections.

HOW TO DEAL WITH BAD BIOPSIES DURING SURGERY

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ABSTRACT BODY:

Surgical oncology

Incisional and excisional biopsy techniques

Larger amounts of tissue can be obtained by incisional and excisional biopsy techniques. These techniques will allow better evaluation of tumour characteristics, including margins, but require general anaesthesia. Excisional biopsies are performed in areas where the tumour can be easily removed using adequate margins or in case of benign lesions. Incisional biopsy techniques are best used for large tumours, or for tumours located in places where wide excision is not possible. The location of the biopsy incision is of extreme importance because it needs to be excised during later surgical procedures

Surgical planning

The extent of surgery depends on the type and stage of the tumour. A pre-resection biopsy is often necessary to anticipate sufficient margins of resection and behaviour of the primary tumour. Staging the tumour will prevent treatment failures caused by early metastasis and will help in deciding the type of resection and use of adjuvant treatment modalities. For some tumour types, histologic grading may be valuable to anticipate the prognosis after surgery. Diagnosis of intercurrent diseases often will alter surgical treatment options and should be evaluated beforehand to assess the risk versus benefit of surgical intervention. The surgical field should be prepared carefully to allow changes in the extent of resection based on new information obtained during the operation.

Surgical technique

Oncologic surgery often combines many surgical specialties. Knowledge of general, orthopaedic, reconstructive, and oncologic surgical techniques will need to be combined with a solid anatomical knowledge base to prevent unpleasant and unnecessary surprises. Dissection of tumours should be clean, and exposure to the tumour should be prevented. Tumour cell contamination (seeding) is minimized by avoiding tumour incisions, shielding normal tissue with protective covering, and using proper electrosurgical techniques. In case of contamination, contaminated sites should be resected (where possible), the wound should be lavaged, and contaminated gloves, drapes, and instruments replaced. Adjunctive therapy often is indicated in these cases. Tissue handling should be according to Halstead's principles, and the tumour itself should be handled as little as possible. Neovascularisation is often prominent and should be dealt with properly by electrocoagulation or suture ligation. Tissues macroscopically invaded by the primary tumour should be excised 'en bloc'.

Surgery for cure

Resection for a cure must be aggressive and at an early stage of the disease. The most important theme for oncologic surgical management is that the first time has the best chance of curing the patient. Margins should not be compromised because it is better to leave a wound open to heal by second intention than to leave tumour cells behind. Recurrent tumours are always more difficult to treat than primary ones. Familiarity with tissue grafts, skin flaps, and other reconstructive techniques will allow wider resections, minimize patient morbidity (and in the end, patient mortality) and decrease the temptation to compromise the resection and thus recurrence.

Surgery for palliation

The goal of palliative surgery is to improve the quality of the patient's life without the guarantee that it will increase the survival time. This type of surgery demands very careful consideration of the risk versus benefits but will be beneficial for certain patients (e.g., splenectomy for an acutely bleeding hemangiosarcoma of the spleen).

Surgery for cytoreduction

Incomplete removal of the tumour is only beneficial in cases in which total resection is not possible, in which adjunctive therapy may benefit from the procedure, and in cases of severe morbidity caused by the primary tumour. Cytoreduction is, however, rarely indicated and should be avoided.

Surgical margins

The necessary surgical margins depend on the tumour type, grade, and anatomical location of the tumour. The most rational approach is to think of biologic rather than geometric margins and to combine this information with the expected growth behaviour of the given tumour type. Tumours with a high probability

of local recurrence (e.g. mast cell tumours, feline mammary tumours) should have 2 to 3 cm margins removed three-dimensionally. Collagen/matrix-rich and poorly vascularised tissues are the least vulnerable to tumour invasion and may be used for margin determination. All previously performed biopsy tracts should be removed in continuity with the primary tumour to prevent tumour seeding. With a proper resection, the tumour is never visualized.

Postoperative care

Supportive care is provided as necessary for vital organ function, nutritional support, and wound healing. Appropriate pain management is essential and often neglected. Resected tumour specimens should be sent for histologic examination to evaluate tumour type, grade, and surgical margins. Questionable results are discussed with the pathologist. Long-term monitoring includes regular examinations to facilitate early detection of tumour recurrence.

Conclusion

Surgical therapy of companion animal cancer is an important treatment modality. Correct and aggressive surgical resection after a solid diagnosis in an early stage of the disease will be associated with the best results.

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ACTUALIZACIÓN SOBRE LOS PROBLEMAS RELACIONADOS CON LA SEPARACIÓN: MÁS ALLÁ DEL HIPERAPEGO

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ABSTRACT BODY:

INTRODUCCIÓN

Los problemas de comportamiento se definen como todas aquellas conductas que molestan a los cuidadores del animal. Muchas de estas conductas afectan al bienestar del animal y de las personas con las que convive. Los problemas relacionados con la separación (PRS) hacen referencia a todos aquellos problemas de conducta que se dan única o mayoritariamente en ausencia de los tutores.

Los signos clínicos más comunes de los PRS son las conductas destructivas, las vocalizaciones (aullidos, ladridos...) y las conductas de eliminación (orinar y defecar) que se producen única o mayoritariamente cuando el perro está solo en casa.

Clásicamente se ha etiquetado a los PRS como "ansiedad por separación" (APS), como si ambas "entidades" fueran sinónimas. Además, se ha asociado el problema al exceso de apego del perro (hiperapego) hacia los cuidadores (personas) como una de las principales causas del problema. De hecho, se clasificaban los diferentes tipos de problemas relacionados con la separación en función de cómo se había formado, supuestamente, el hiperapego. Es fácil encontrar en la literatura científica clasificaciones de ansiedad por separación en relación a si el animal tenía un problema de hiperapego primario o secundario. Sin embargo, el abordaje a los PRS ha cambiado radicalmente en los últimos años.

El objetivo de esta charla es mostrar los factores que deben tenerse en cuenta para entender los problemas de comportamiento relacionados con la separación, dar las claves para el correcto diagnóstico del problema y presentar las principales estrategias de tratamiento para los principales diagnósticos.

¿POR QUÉ ES IMPORTANTE DIAGNOSTICAR Y TRATAR LOS PRS?

Los PRS afectan al bienestar de la familia y al del animal por varios motivos:

- Algunos de los PRS se deben directamente a la expresión de emociones negativas (miedo, ansiedad o frustración –ver más adelante-) y, por lo tanto, la presencia de estas emociones es, en sí mismo, un indicador de falta de bienestar.
- Los signos clínicos más comunes pueden generar una situación estresante para las personas (problemas con el vecindario, denuncias a la policía, etc.). Como consecuencia es habitual que pongan en marcha medidas de tratamiento que afectan al bienestar del animal (collares de descargas eléctricas) o, incluso, que se planteen el abandono o eutanasia del animal.

DIAGNÓSTICOS DE PRS, POR QUÉ NO SE DEBE LLAMARLO APS

Los signos clínicos más comunes de los PRS (vocalizaciones, destructividad y eliminación inadecuada) no son específicos de un solo diagnóstico. Estos pueden empezar a manifestarse incluso antes de que los cuidadores abandonen la casa (esto es especialmente importante en los casos de PRS asociados a emociones negativas).

No todos los perros que presentan estos signos clínicos en ausencia de los tutores lo hacen por un problema de ansiedad, miedo o estrés.

PRS RELACIONADOS CON EMOCIONES POSITIVAS

Con frecuencia se tiende a relacionar los PRS con emociones negativas (miedo, ansiedad o frustración), sin embargo, existen PRS que se correlacionan con emociones placenteras.

- PRS relacionados con el **juego**: Es frecuente en perros jóvenes. Existen comportamientos comunes de los cachorros que son molestos para los tutores. La conducta exploratoria y el juego están muy exacerbados en los primeros meses de vida. Tanto para el juego como para la exploración del entorno, es normal que los perros utilicen la boca (mordisqueando) o las patas delanteras. Esto conlleva que se puedan presentar conductas destructivas. Con frecuencia los tutores castigan estas conductas. Este hace que el perro aprenda rápidamente a realizar la conducta solo en su ausencia. Si, además, se le suma que durante el juego es habitual que haya vocalizaciones (con la misma respuesta de los cuidadores) y que todavía no han aprendido los hábitos higiénicos, es muy fácil que se confunda este problema, con una APS "clásica". Es importante, por lo tanto, hacer un

correcto diagnóstico (ver más adelante) para evitar tratamientos incorrectos.

- **Conducta sexual (lujuria)**: Algunos perros muestran conductas de vocalización y de escapismo, que se asocia a destructividad hacia las puertas de salida, con la finalidad de tener acceso a una monta. Es típico de machos y de hembras en celo.

PRS RELACIONADOS CON EMOCIONES NEGATIVAS

- PRS relacionados con el **miedo/fobia**: Perros que muestran miedo o fobia específica a estímulos ambientales (como, por ejemplo, a fuegos artificiales o tormentas) y experimentan un estado fóbico estando solos en casa, pueden desarrollar miedo a quedarse solos en casa (especialmente en el entorno en el que han experimentado la situación fóbica). Estos perros muestran signos de ansiedad o estrés cuando van a quedarse solos, y cuando están solos, en ese entorno, incluso en ausencia de los estímulos fóbicos. Por este motivo, si se sospecha en la historia clínica de esta posibilidad, es importante hacer grabaciones del perro en diferentes entornos (interior de casa, jardín, otras casas…).
- PRS relacionados con la **frustración**: Al quedarse solos en casa existen muchas conductas que se ven frustradas. Como salida normal a la frustración pueden aparecer signos clínicos de ansiedad o estrés. Es importante determinar para el tratamiento si la frustración juega un papel importante. Por ejemplo, es probable que las medidas ambientales puedan ser más efectivas que el uso de psicofármacos en algunos de estos casos.
- **Falta de habituación** a quedarse solo en casa: Con frecuencia no se le ha enseñado correctamente a quedarse, de forma progresiva, solo en casa. Como animal social, el perro es una especie que necesita estar acompañado y el hecho de estar solo es un proceso que debe aprenderse de forma progresiva. El papel del apego en estos casos es fundamental para que este aprendizaje sea consistente y duradero en el tiempo.

Es importante señalar que un mismo animal pueden coexistir varias de las motivaciones que se acaban de mencionar.

PAPEL DEL APEGO EN LOS PRS

El apego es un sistema comportamental que regula una relación afectiva entre dos individuos, en la que uno trata de mantenerse cerca del otro, al que necesita como referencia (base segura) para su correcto desarrollo. Se ha observado que el sistema de apego que se estable entre un perro y su cuidador es igual que el que se establece entre un niño y sus progenitores o cuidadores.

El apego juega un papel fundamente en el desarrollo de los PRS (especialmente los relacionados con emociones negativas). Aunque clásicamente se hablaba de hiperapego, hoy en día se sabe que lo importante en los PRS es el tipo o calidad de apego que perro establece.

Existen dos grandes grupos de apego, seguro e inseguro (y varios tipos de apego inseguro). Los perros que tienen un apego seguro con sus tutores tienden a aceptar mejor la soledad en la edad adulta, lo contrario que ocurre con la mayoría de apegos inseguros. Durante la charla se abordará con mucho detalle el papel del apego en el desarrollo de los PRS.

DIAGNÓSTICO Y TRATAMIENTO DE LOS PRS

Siempre deberá hacer un abordaje amplio que incluya tanto aspectos del comportamiento del animal, como aspectos de su salud física. Además, será necesario hacer varias grabaciones de video cuando el animal esté solo en casa.

El tratamiento debe incluir varios aspectos importantes:

- **Manejo del entorno**. Incrementar la previsibilidad de las salidas ayudará a reducir tanto el estrés como sus posibles consecuencias negativas. Se utilizarán una serie de señales ambientales para anticipar al perro que va a quedarse solo.
- **Habituación** a quedarse solo en casa. Se realizará una desensibilización y contracondicionamiento a quedarse solo en casa. Para ello será mejor utilizar una zona de la casa en la que no se quede solo habitualmente.
- **Utilización de psicofármacos y feromonas**. La feromona apaciguadora del perro se utiliza tanto para reducir el estrés como para la señalización de la zona de seguridad del animal. En muchos casos, además, deben utilizarse combinación de varios psicofármacos ansiolíticos (a corto y largo plazo) para controlar los signos clínicos mientras se implementan las otras medidas de tratamiento.
- **Manejo de la rutina del animal**. Con frecuencia, muchos otros problemas de conducta que se den de forma concomitante podrían afectar al desarrollo del problema RS.

CONCLUSIONES

La comprensión, diagnóstico y tratamiento de los PRS ha cambiado radicalmente en los últimos años. El papel del apego es crucial, y potenciar un apego seguro será necesario para el tratamiento de la mayoría de problemas. El tratamiento será multifactorial, teniendo en cuenta todos los elementos que intervienen en el problema.

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QUE HEMOS APRENDIDO EN 30 AÑOS DE PROFESIÓN DE: PATOLOGÍA DE CADERA

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ABSTRACT BODY:

Que hemos aprendido en 30 años de profesión de: Patología de cadera

La principal causa de osteortrosis (OA) coxo-femoral en el perro es la Displasia de Cadera (DC) que consiste en una formación anómala de la articulación de la cadera con laxitud articular. Descrita en el perro en el año 1935, es la patología ortopédica más diagnosticada en el perro y sigue constituyendo un reto tanto para veterinarios como para criadores.

La etiopatogenia se debe a factores ambientales: alimenticios, crecimiento rápido, exceso de ejercicio, esterilización precoz, etc…, unidos a un importante componente hereditario en el que intervienen muchos genes. El resultado es una falta de congruencia entre el acetábulo y la cabeza femoral que provoca laxitud coxo-femoral e inestabilidad articular, con el consiguiente desarrollo de OA. El hecho de que sea una patología poligenética complica su erradicación.

Ha sido descrita en más de 70 razas de perros afectando con mayor incidencia a las razas grandes y gigantes con prevalencias del 60-70% en algunas razas.

El diagnóstico clínico se basa en la forma de andar del animal, dolor a la manipulación de la cadera y sobretodo en evidenciar la laxitud articular mediante el test de Ortolani, con el animal bajo sedación o anestesia.

No existe correlación entre los signos radiológicos y los síntomas clínicos: algunos animales con displasia grave pueden mostrarse asintomáticos en tanto que otros con displasia menos severa pueden presentar síntomas graves e incluso invalidantes.

La técnica radiológica estandarizada y aceptada universalmente requiere la sedación o anestesia del animal, colocándolo en posición de decúbito dorsal con las extremidades extendidas, paralelas y sometidas a rotación interna, de modo que las rótulas se sitúen sobre la tróclea femoral. La simetría debe ser perfecta, de lo contrario la imagen radiológica puede inducir a error.

Con el fin de objetivar y cuantificar la lectura de la radiografía, se utilizan diferentes medidas coxométricas. Las más utilizadas son : el ángulo de Norberg y el recubrimiento acetabular. En el primero, una línea une los dos centros de las cabezas femorales y otra línea va del centro femoral tangente al borde acetabular real. Cualquier ángulo menor de 105º indica subluxación . En cuanto al recubrimiento acetabular, éste debe ser como mínimo del 50%. La clasificación de los grados de displasia varía en función de los países. La clasificación aceptada en nuestro país es la propuesta por la FCI (Federación Canina Internacional) con una escala de 5 puntos , en tanto que la OFA (Orthopedic Foundation for Animals) considera una escala de 7 puntos.

Un método de diagnóstico precoz que identifica y cuantifica la laxitud articular, ha sido puesto a punto por la Universidad de Pennsylvania (U.S.A) : PennHIP (Pennsylvania Hip Improvement Program).Se puede hacer a partir de los 4 meses de edad. El grado de desplazamiento femoral (laxitud articular) se cuantifica con el Indice de Distracción (ID), de manera que un fémur totalmente luxado tendría un índice de laxitud de 1 , en tanto que una cadera perfecta, sin laxitud , daría un índice de 0. El inconveniente más importante del método es que no es tan fácilmente estandarizable como la técnica convencional, y que el índice de laxitud “fisiológico” varía en función de las razas

El tratamiento conservador es multimodal y persigue el alivio del dolor y ralentizar la lesión cartilaginosa articular.Los AINES son el medicamento de elección para tratar la inflamación y el dolor asociado a la DC mediante la inhibición de enzimas COX. Existen en el mercado un gran número de AINES y se aconseja un control clínico periódico para controlar posibles efectos secundarios (toxicidad hepática y renal). Más recientemente disponemos de medicamentos que no inhiben la COX, sino que actúan por bloqueo selectivo de los mediadores del dolor y la inflamación (grapiprant), y también medicamentos a base de anticuerpos monoclonales que actúan sobre los mediadores del dolor (bedinvetmab)

Los analgésicos se pueden combinar con AINES y poder disminuir la dosis de los mismos .Son efectivos el tramadol, gabapentina, amantadina, entre otros.El uso de corticoides se debe aconsejar solo en casos refractarios a otros tratamientos.

La utilización de los condroprotectores es controvertida y la respuesta clínica difícil de valorar. Los más utilizados son los glicosaminoglicanos-polisulfatos ,la combinación de condrotinsulfato - glicosamina y

omega 3, entre otros.

Clásicamente se ha aconsejado ejercicio moderado y control del peso en animales displásicos. La natación es una excelente alternativa, ya que ayuda a mantener el tono muscular del animal, liberando la articulación de las cargas biomecánicas

Las técnicas quirúrgicas en el tratamiento de la D. C. están perfectamente estandarizadas. En animales jóvenes haremos técnicas orientadas a restablecer la congruencia articular para que el animal no desarrolle OA: la sinfisiodesis púbica en animales jóvenes (hasta los 5 meses de edad) osteotomía doble o triple de cadera (hasta los 8 meses de edad) y la osteotomía femoral en casos de displasia femoral En animales adultos el objetivo será suprimir el dolor debido a la OA mediante la artroplastia y la prótesis de cadera

La sinfisiodes consiste en cauterizar el tercio craneal de la sínfisis pubiana con el objetivo de que durante el crecimiento del animal se produzca una lateralización del cótilo con el fin de aumentar la congruencia articular. Indicada en caso de leve o moderada laxitud articular con un ID <0.6

La Osteotomía Triple de cadera consiste en la osteotomía de los tres huesos del coxal : pubis, isquion e ilion para liberar la parte acetabular de forma que se pueda reorientar y fijar con una placa de osteosíntesis rotada 20º- 25º,para lograr una mayor congruencia de la misma con la cabeza femoral. El uso de placas bloqueadas permite ahorrar la osteotomía del isquion: Osteotomía doble de cadera. El candidato ideal es animal joven, hasta los 8 meses , con displasia acetabular, que presente laxitud articular , sin subluxación y sin signos radiológicos de OA

La artroplastia consiste en la exéresis del cuello y de la cabeza femoral y solo debe utilizarse como último recurso. El fin que persigue es suprimir el dolor, eliminando la articulación. La recuperación funcional no es del todo satisfactoria en la mayoría de los casos y debe ir acompañada de buena rehabilitación física post-operatoria.

La sustitución de la articulación de la cadera en animales con osteoartosis por una prótesis es una técnica cada día más empleada. Generalmente se utilizan prótesis no cementadas, cementadas o hibridas que mediante un sistema modular permite elegir la longitud de cuello femoral más idónea para cada caso en particular. Una correcta selección del paciente y una técnica cuidadosa proporcionan un alto porcentaje de éxitos (90 %) en perros adultos con disfunción por displasia de cadera.

La pectinectomia es una intervención fácil que consigue un efecto antiálgico a corto plazo, pero que no modifica en absoluto la progresión de los signos de osteoartrosis. La mayoría de los pacientes vuelven a mostrar signos clínicos en un futuro más o menos próximo y no se puede considerar como una alternativa a las osteotomías correctivas en animales jóvenes.

POPULATION CONTROL OF DOGS AND CATS

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ABSTRACT BODY:

Population control

Pet overpopulation leading to animal suffering is one of the largest global animal welfare challenges.

Pet "overpopulation" results from several factors:

Allowing cats and dogs to reproduce unchecked knowing there are not enough suitable homes for the offspring i.e., the population of dogs and cats exceeds the capacity to responsibly care for them.

Relinquishment of dogs and cats by owners who can no longer keep their animals (e.g., financial or housing reasons), or who no longer want them.

Stray dogs and cats (these may be referred to as "community" or "feral" animals).

For decades the "holy grail" of animal welfare has been to find a single injection or medication that will permanently and safely sterilize dogs and cats.[A] Although there is a lot of ongoing research in this area, we have not yet found the best way to achieve this. Therefore, until a non-surgical method is available, controlling cat and dog populations depends primarily on surgical procedures (i.e., castration, ovariohysterectomy or ovariectomy) which is time consuming when performed on the scale needed, and requires anesthesia and surgical expertise.

Anesthesia, anesthesia and analgesia for surgical sterilization (neutering)

Castration and ovariohysterectomy/ovariectomy performed in dogs and cats are associated with pain of varying severity which correlates with the degree of surgical trauma. For this reason, surgery should be performed with careful (gentle and minimal) tissue handling and adherence to good surgical principles. In most cases surgical procedures are performed under general anaesthesia and preventive and multimodal analgesia techniques are employed. The overall approach should be integrative, using both pharmacologic and non-pharmacologic techniques to ensure patient comfort.

Anesthetic protocols

WSAVA is a Global Veterinary Community and the Global Pain Council (GPC) recognize that not all analgesic drugs or drug categories are available to all members.[B] For this reason the GPC has given careful thought to providing suitable anesthetic and analgesic care to dogs and cats regardless of their geographical location.

Protocols

Protocols for male and female dogs and cats have been created by the GPC for three scenarios: 1. No restrictions to drug access, 2. Where not all controlled drugs are available and 3. When there is limited access to analgesic drugs. Protocols for castration of dogs and for ovariohysterectomy or ovariectomy in cats are shown in **Table 1 and 2**. Other protocols are in the GPC's Guidelines for the Recognition, Assessment and Treatment of Pain, which has been updated and will be launched in 2022.

Local anesthetic blocks

Local anesthetic drugs are widely available and are not controlled drugs. They should be used whenever possible. Intratesticular and incisional blocks in males and intraperitoneal administration and incisional blocks in females are easy to administer and are part of a multimodal approach. The value of intraperitoneal administration of local anesthetics in dogs and cats has been reviewed[1].

Cold therapy

Cold therapy is a valuable non-drug therapy for acute pain[2]. Cold therapy works in many ways including but not limited to decreasing activation of tissue nociceptors, slowing conduction velocity along peripheral nerves, reducing swelling secondary to vasoconstriction and activation of specific channels e.g., those of the Transient Receptor Potential (TRP) cation channel family[2].

Nursing care

The term "Tender Loving Care" is used in nursing of humans and animals. Providing clean, dry and comfortable bedding, maintaining a warm environment, decreasing stressors and using gentle handling all contribute to the comfort of a dog or cat after surgery.

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WSAVA RESOURCES

https://wsava.org/committees/global-pain-council

- *WSAVA Global Pain Council's Guidelines for the Recognition, Assessment and Treatment of Pain (updates to be published in October 2022)*
- *Pain assessment tools*
- *Local anesthetic blocks – video links*
- *Campaigns – ketamine as an essential medicine*

Table 1. Suggested protocol for canine castration

		Protocol with controlled drugs	Protocol without controlled drugs	Protocol with limited availability of analgesic drugs
Preoperative		Opioid ± acepromazine or benzodiazepines ± alpha$_2$-adrenoceptor agonist	NSAID + alpha$_2$-adrenoceptor agonist	Same as protocol without controlled drugs
Induction of anesthesia	IV†	Choose one of the following: Propofol Ketamine + diazepam or midazolam Alfaxalone	Choose one of the following: Propofol Alfaxalone	Any available injectable agent
	IM	Opioid + alpha$_2$-adrenoceptor agonist + ketamine or tiletamine/zolazepam	Alpha$_2$-adrenoceptor agonist + tiletamine/zolazepam	
Maintenance of anesthesia‡		Choose one of the following: Inhalation anesthesia Ketamine Propofol Alfaxalone	Choose one of the following: Inhalation anesthesia or Propofol Alfaxalone	Any available injectable or inhalant agent
Local anesthetic techniques		Intratesticular ± incisional block	Same as protocol with controlled drugs	Same as protocol with controlled drugs
Postoperative analgesia		NSAID	Same as protocol with controlled drugs	Same as protocol with controlled drugs

IV intravenous, IM intramuscular, NSAID non-steroidal anti-inflammatory drug

†Note that premedication reduces IV anesthetic requirements; thus, induction doses should be titrated to effect

‡Injectable drugs are administered IV to effect (⅓ or ½ of initial dose)

Table 2. Suggested protocol for feline ovariohysterectomy/ovariectomy

		Protocol with controlled drugs	Protocol without controlled drugs	Protocol with limited availability of analgesic drugs
Preoperative		Opioid ± acepromazine or alpha$_2$-adrenoceptor agonist ± ketamine	NSAID + alpha$_2$-adrenoceptor agonist	Same as protocol without controlled drugs
Induction of anesthesia	IV†	Choose one of the following: Propofol Ketamine + diazepam or midazolam Alfaxalone	Choose one of the following: Propofol Alfaxalone	Any available injectable agent
	IM	Opioid + alpha2-adrenoceptor agonist + ketamine or tiletamine/zolazepam	Alpha$_2$-adrenoceptor agonist + tiletamine/zolazepam	
Maintenance of anesthesia‡		Choose one of the following: Inhalation anesthesia Ketamine Propofol Alfaxalone	Choose one of the following: Inhalation anesthesia Propofol Alfaxalone	Any available injectable or inhalant agent
Local anesthetic techniques		Incisional ± intraperitoneal block	Same as protocol with controlled drugs	Same as protocol with controlled drugs
Postoperative analgesia		NSAID	Same as protocol with controlled drugs	Same as protocol with controlled drugs

IV intravenous, IM intramuscular, NSAID non-steroidal anti-inflammatory drug

†Note that premedication reduces IV anesthetic requirements; thus, induction doses should be titrated to effect

‡Injectable drugs are administered IV to effect (⅓ or ½ of initial dose)

ANTIMICROBIAL USE GUIDELINES IN CATS: WHICH DRUG, WHAT DOSE AND FOR HOW LONG?

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ABSTRACT BODY:

Guidelines for treatment of urinary tract disease in dogs and cats (recently updated), respiratory infections in dogs and cats, and superficial pyoderma in dogs have been published by the International Society for Companion Animal Infectious Diseases (www.iscaid.org). Recommendations are based on available data, whenever present, along with expert opinion, considering principles of infectious diseases, antimicrobial treatment, antimicrobial resistance, pharmacology, and internal medicine. Concerns about poor antimicrobial stewardship in companion animals has led to increased attention from legislative bodies such as the United States Food & Drug Administration to prescribing practices, and several studies are underway to better understand these practices and optimal regimens for treatment of different disorders.

Because of the increased prevalence of antimicrobial drug resistance, the need to properly document the presence of an infection before initiating antimicrobial drug treatment is more important than ever. In veterinary medicine, this may be at odds with client financial resources. However, inappropriate use of antimicrobial drugs is wasteful of client resources when an infection is not present or a multidrug resistant pathogen is present, and risks selection for antimicrobial resistant bacteria that may be harmful to the pet, other animals, and also humans that are in contact with the animal.

Some basic recommendations within the urinary and respiratory guidelines are summarized below. Doses of specific antimicrobial drugs are listed in the guidelines. New point-of-care diagnostic tests (such as RapidBac Vet, Silver Lake Technologies) that can rule out bacteriuria in animals with lower urinary tract signs are strongly recommended in order to reduce overprescribing, especially because the incidence of lower urinary tract infections in cats is low.

RECOMMENDATIONS FOR URINARY TRACT DISEASE

Sporadic Cystitis

Definition: Sporadic bacterial infection of the bladder in an otherwise healthy cat with normal urinary tract anatomy and function.

- The presence of urinary tract infection implies the presence of dysuria, pollakiuria, and/or stranguria. However, diagnosis of UTI cannot be made on the basis of clinical signs alone.
- Sediment analysis alone is not adequate for diagnosis.
- Complete urinalysis and quantitative aerobic C&S testing should be performed for all cases. Free-catch samples should not be used.
- For cystocentesis specimens, counts $\geq 10^3$ CFU/mL is supportive of a diagnosis of UTI in association with signs of lower urinary tract disease. For catheterized specimens, counts $\geq 10^4$ in males and $\geq 10^5$ CFU/mL in females are significant.
- Bacterial isolation should only be attempted in clinics with appropriate laboratory facilities, proper biosafety containment and waste management, and adequately trained individuals. A recent study showed that in-house “urine paddles” may be useful to rule out the presence of infection but these do not reliably identify bacteria and can generate false negative results.[3]
- Treatment is indicated to relieve patient discomfort while awaiting C&S test results. Recommendations for initial treatment are amoxicillin (11 – 15 mg/kg PO q12h) or trimethoprim-sulfonamide (15 mg/kg PO q12h).
- Veterinarians are encouraged to document and monitor resistance patterns among isolates from their hospital.
- If C&S testing reveals a resistant isolate and there is a lack of clinical response, treatment should be changed to an appropriate antimicrobial drug.
- Although treatment has been recommended in the past for 7 to 14 days, recent research suggests 3-5 days may be more appropriate.[4,5]
- There is no evidence that intra- or post-treatment urinalysis or urine culture is indicated in the absence of ongoing clinical signs of UTI.

Recurrent UTI

Definition: the presence of 3 or more episodes of UTI during a 12-month period.

- The same general principles as for sporadic cystitis apply
- Efforts should be made to identify the underlying cause; consider referral

- Treatment should be based on the results of C&S testing
- Although 4 weeks has been recommended for treatment, shorter durations are recommended, with a focus on clinical cure rather than microbiological cure.
- There is insufficient evidence to recommend "pulse" or chronic low-dose treatment, urinary antiseptics, and nutritional supplements such as cranberry juice extract for prevention of UTIs.

Subclinical Bacteriuria

Definition: presence of bacteria in the urine as determined by positive bacterial culture, in the absence of clinical signs of UTI.

- Treatment may not be necessary, but could be considered if there is a high risk of ascending or systemic infection (eg. patients with underlying renal disease)
- Diagnosis and management of the underlying cause is critical

Urinary Catheters

- Clinical signs of UTI absent: no culture or treatment indicated.
- Removal of urinary catheters: urine culture is reasonable if the risk and implications of a UTI are high, but in general it is not recommended. There is no indication for routine use of prophylactic antimicrobials.
- Clinical signs of UTI present: perform a culture after replacement of the urinary catheter with a new catheter. Several mL of urine should be removed to clear the catheter before a specimen is obtained for culture. Alternatively, remove the catheter and perform a cystocentesis. Culture from the collection bag, and culture of the catheter tip after removal are not recommended. Treatment should follow the guidelines for complicated and uncomplicated UTIs, and is more likely to be successful after catheter removal.

Pyelonephritis

- C&S testing should always be performed.
- Treatment should be initiated while awaiting culture results, using antimicrobials effective against Gram-negative Enterobacterales. A fluoroquinolone is a reasonable first choice, after which treatment should be based on C&S results. If combination treatment was used initially and C&S results indicate that both drugs are not required, the spectrum should be narrowed.
- Treatment for 4 to 6 weeks is recommended until further information becomes available.
- Culture is recommended 1 week after starting treatment and 1 week after treatment is discontinued.

RECOMMENDATIONS FOR RESPIRATORY DISEASE

Acute Upper Respiratory Tract Disease (URTD)

- Consider an observation period of up to 10 days without antimicrobial treatment for cats with acute URTD. Antimicrobial therapy should be considered if a mucopurulent nasal discharge is accompanied by fever, lethargy or anorexia. In the latter case, appropriate empiric therapy would be doxycycline (first choice) followed by amoxicillin (the latter is not active against *Mycoplasma* spp.). The duration should be 7-10 days.
- Avoid performing C&S on nasal discharge from cats with acute URTD
- If empiric antimicrobial therapy is ineffective, a diagnostic work-up is indicated.

Chronic Upper Respiratory Tract Disease in Cats

- A diagnostic work-up is recommended. If treatable causes of nasal discharge are not identified, then nasal lavage or brushings could be submitted for C&S testing, and a nasal biopsy could be submitted for histopathology. Treatment should be based on these results.
- Should nasal discharge recur, the previously effective antimicrobial drug should be used for a minimum of 48 hours; if this is ineffective, then switch to a different class should be considered, provided a diagnostic work-up to rule out other causes of nasal discharge (tumors, fungal infection, foreign bodies etc) has been performed.

Bacterial Bronchitis in Cats

- Airway lavage with cytologic examination and C&S testing is indicated if bacterial bronchitis is suspected
- While awaiting results of the above tests, empiric treatment is recommended with doxycycline for 7 to 10 days. If this results in clinical improvement, treatment should be continued for 1 week past resolution of clinical signs.

Pneumonia

- Antimicrobial therapy for pneumonia should be initiated as soon as possible and within 1-2 hours if signs of sepsis exist
- Antimicrobial therapy should be parenteral while patients with pneumonia are hospitalized
- If there is no evidence of systemic sepsis, parenteral administration of a beta-lactam is recommended for empiric therapy; if signs of sepsis are present, then a combination of a fluoroquinolone and a drug

that targets gram-positive bacteria and anaerobes (e.g., ampicillin or clindamycin) is recommended pending the results of C&S if possible. Animals should be re-evaluated for possible discontinuation of antimicrobials no later than 10 to 14 days after starting treatment.

Pyothorax

- Pyothorax should be treated with IV fluids and drainage of pus after placement of chest tubes. Surgical debridement may be required for some animals.
- Empiric antimicrobial therapy pending the results of C&S should be with a parenteral combination of a fluoroquinolone and a penicillin or clindamycin
- It has been recommended that treatment continue for at least 3 weeks and ideally 4-6 weeks, but the optimum duration is unknown. Animals should be re-evaluated 10 to 14 days after starting treatment.

CASE-BASED NUTRITIONAL MANAGEMENT OF CANINE PROTEIN-LOSING ENTEROPATHY

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CASE-BASED NUTRITIONAL MANAGEMENT OF CANINE PROTEIN-LOSING ENTEROPATHY

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Protein-losing enteropathy

Protein-losing enteropathy (PLE) refers to a gastrointestinal condition that results in a net loss of protein from the body through a leakage into gut lumen. The leakage of albumin is often marked, leading to severe hypoalbuminemia. [1]

PLE is not a specific disease but a syndrome. PLE may develop due to various aetiologies, and it often involves the lymphatic system, such as lymphangiectasia and lymphangitis in dogs.

Causes of PLE in dogs:

- Mucosal injury due to e.g., chronic inflammation, parvovirus, NSAID-induced damage, neoplasia
- Parasite infection
- Lymphatic disease e.g., intestinal lymphangiectasia (IL) [1]

Most dogs with PLE are diagnosed with IL, which is believed to be secondary to lymphocytic-plasmacytic enteritis.[2]

Clinical presentation

Dogs with PLE are commonly presented with diarrhoea, vomiting, weight loss, depression, and poor appetite. Severe hypoalbuminemia may lead to ascites, peripheral swelling and/or pleural effusion.

PLE may affect any breed (including mixed breed) and gender, however there is predilection for the syndrome in the Yorkshire Terrier, Border Collie, German Shepherd, and Rottweiler.[2]

Marked hypoalbuminemia (< 20 g/l) is the hallmark of canine PLE. Other laboratory findings include lymphopenia, hypocholesterolemia, hypoglobulinemia, hypocalcemia, and hypomagnesemia. Hypocobalamenia is also common and is associated with poor prognosis. [3]

Diagnostic work-up

The workup of dogs suspected to have PLE is like that for dogs with chronic small intestinal diarrhoea, including complete blood count, serum biochemistry panel (with cobalamin assay) and urinalysis. Pancreatic diseases and hypoadrenocorticism should be ruled out.

As the prognosis of PLE is always guarded and appropriate therapy needs to be started as soon as possible, intestinal biopsies should be obtained promptly. Intestinal lymphoma and lymphangiectasia can sometimes be found in ileum only so both duodenal and ileal biopsies are indicated in these patients.[2]

Enteric mucosal lesions associated with IL in dogs may be observed during endoscopic examination. There may be white patchy appearance to the small intestinal mucosa, and white tipped intestinal villi. However, the presence of visible mucosal changes is of low specificity and sensitivity, thus good quality biopsies are always needed for more precise diagnosis.[2]

Therapy

Therapy for PLE consists of medical therapies and dietary intervention.

Immunosuppressants are the cornerstone of PLE medical therapy. Most dogs are started with prednisolone 2-3 mg/kg/day, dose is tapered down with approximately 2 weeks intervals. In most cases prednisolone is combined with another immunosuppressant (e.g., azathioprine, chlorambucil, methotrexate, or mycophenolate) either because of poor response to glucocorticoid therapy alone, or to lessen prednisolone-related adverse effects. [1,3]

PLE dogs have a higher risk of thrombosis, so an antithrombotic medication (e.g., clopidogrel or low-dose aspirin) can be prescribed.[1]

For dogs diagnosed with hypocobalananemia, supplementation with vitamin B12 is needed. Cobalamin may be supplemented either orally or parenterally.[4]

Dietary therapy

Dietary therapy has a vital role in the management of canine chronic enteropathies (CE). Many dogs with CE respond well to hydrolysed protein food and need no other therapy besides a strict diet.

Also, dogs with PLE can be managed with dietary treatment alone. Positive outcome of dietary

intervention has been demonstrated across various diet types (both homemade and commercial), and controlling nutrient ratios (e.g., fat-restricted, limited, or novel antigen). Dogs with PLE may be seriously malnourished and anorectic, so placing a feeding tube when the dog is anesthetized for endoscopy should be considered to support adequate nutrition.[1]

In the management of IL, a low-fat diet is traditionally advocated. There is no consensus for the ideal amount of fat in the diet, but nutritionists generally believe less than 20% fat on a metabolizable energy (ME) basis to be suitable. Ultra–low-fat diets are generally less than 15% fat on an ME basis.

Supplementation with medium-chain triglycerides (MCTs) has been advocated to increase caloric intake in dogs with PLE and IL. In humans, MCTs are commonly used in IL patients as they are absorbed directly into the portal vein. In dogs, however, MCTs are absorbed into the lymphatic system and thus may not help reduce lymph flow. On the other hand, MCTs may have anti-inflammatory effects in canine IL as they modulate intestinal inflammation. More studies are needed to evaluate the role of MCTs in canine IL therapy.[4]

There are numerous commercial low-fat diets available, but a home-cooked diet may be more suitable in the management of PLE, especially when IL is present. Home-cooked diet can be designed ultra-low-fat with good digestibility, and possibly with a novel protein. However, consultation with a board-certified veterinary nutritionist is recommended to ensure that the diet is complete and balanced.[4]

Prognosis

Unfortunately, PLE has very high mortality, and survival rates are less than 50 %. Therefore, a prompt diagnosis followed with an aggressive and multimodal treatment plan, combining dietary modification, and medication (mostly immunosuppressives) is imperative.

Immunosuppressive treatment in PLE has been scrutinized because of adverse effects of the medications. Currently, there have been encouraging reports of good clinical response to various dietary interventions alone in dogs with PLE. In addition, dietary management of PLE, compared with immunosuppressive medication, is associated with a more positive outcome.[3]

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ARTIFICIAL INSEMINATION: INTRAVAGINAL, TRANSCERVICAL OR SURGICAL?

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ARTIFICIAL INSEMINATION - INTRAVAGINAL, TRANSCERVICAL OR SURGICAL?

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1. Introduction

The first artificial insemination of fresh semen was performed by Spallanzani in 1780 and with frozen-thawed semen and subsequent pregnancy by Seager in 1969 (1). Since then, many studies were performed to improve the insemination technique and the pregnancy rates, especially with frozen semen; however, also with fresh and cooled canine semen.

Transfer of fresh semen is advantageous in cases, where the male dog is not able to mate, because of orthopaedic problems or advanced age, and in cases of beginning deterioration of semen quality due to testicular or other health problems. In many countries, artificial insemination (AI) of fresh semen is performed as a routine method, to be able to examine semen quality before the insemination and to be able to improve the quality, for example in case of a prostate gland disease. Intravaginal deposition of the semen is the preferred technique.

The advantage of cooled and deep frozen semen is obvious: semen of valuable breeding dogs can be stored and sent all over the world. Cooled canine semen is easy to produce; however, good fertilization results can only be obtained, when the initial semen quality is good, the storage temperature is constant, the handling is good and the technique and time of insemination are well chosen. Cooled canine semen of good quality is mostly deposited intravaginally.

Cryopreserved dog spermatozoa, when used for artificial insemination, usually produce lower conception rates than fresh spermatozoa, particularly when inseminated intravaginally (2); the preferred technique is transcervical intrauterine insemination (TCI).

Successful fertilization of bitches with fresh, cooled or frozen-thawed semen requires knowledge about semen handling, preservation, oestrus detection in the bitch; the insemination techniques is only the final step; however, has to be chosen carefully.

2. Insemination Technique & Pregnancy Rates

For the transfer of fresh semen, the semen should be collected and inseminated within 30 to 60 minutes into the vagina or uterus to avoid semen quality deterioration. The number of acrosome defects increases significantly after 2h of storage at room temperature, the insemination technique will therefore mostly be intravaginal. Provided the quality is good and the stage of oestrus at the time of insemination is correct, good pregnancy rates > 80% can be achieved (3, 4); only in case of bad semen quality, intrauterine insemination is recommended since higher pregnancy rates may be the result (5). For intravaginal insemination, different methods exist. In most cases, simple infusion pipettes or special catheters without or with an inflatable cuff, like the Osiris catheter (IMV technologies, Normandy, France), are used. The cuff successfully prevents backflow of semen but is not required to improve pregnancy rates and is not useful for large breeds (6, 5). It is not proven, whether instillation of prostate gland secretion after insemination with the second fraction is beneficial or not, some authors describe a positive effect on pregnancy rates (7). However, the prostate secretions should be examined carefully for ph and admixtures, especially in older dogs, and should not be used for insemination when unphysiologically changed. In the case of prostate gland disease, fractionated collection is advantageous to be able to discard the changed prostatic secretion; centrifugation and addition of a diluent before insemination can be beneficial.

Otherwise not only the insemination technique, but in addition the semen quality, especially the number of progressively motile spermatozoa, the semen handling, the insemination time, and the age, weight and fertility of the bitch significantly influence subsequent pregnancy rates (3).

Concerning cooled canine semen, dependant on the initial semen quality, the diluent and the storage temperature, good quality concerning motility and viability was still found after 7-10 days of cooled storage. The obtained pregnancy rates after AI can be similar to those after natural mating (8). Semen usually may be inseminated intravaginally. No controlled study

is available proving the advantage of intrauterine semen deposition; only in case of bad semen quality, TCI might be advantageous, as has been stated for fresh semen (5).

Frozen-thawed dog spermatozoa usually produce lower conception rates than fresh spermatozoa (3) even though exceptions have been described (9). Intrauterine deposition of semen is strongly recommended when using frozen semen (8). Using the Norwegian Catheter for TCI, pregnancy rates between 51 and 75% have been achieved (4). Intravaginal deposition of frozen-thawed semen by using an Osiris catheter and instillation of prostatic secretions after insemination resulted in significantly lower pregnancy rates (6). With endoscopical TCI, results varied considerably, probably because of inaccurate timing of oestrus and different numbers of progressively motile spermatozoa; the latter should be > 150 x 10^6 (5).When data were corrected for insemination time and semen quality, results of > 80 % were obtained (8). Surgical intrauterine insemination did not improve pregnancy rate in comparison to TCI (10). Own experiences show that the endoscopic technique allows secure deposition of semen inside the uterus without stress and results in good pregnancy rates of 60% to >80% dependant on semen quality, timing of AI and fertility of the bitch.

2. Insemination timing

Plasma *progesterone measurements*, *vaginal smears* and *vaginal inspections* contribute to the detection of the optimal time for insemination and pregnancy rates. A progesterone concentration of 5 ng/ml plasma and above indicates ovulations. Insemination should take place 1-2 days later. Frozen-thawed dog spermatozoa retain fertility for 24 hours and because after thawing the percentage of spermatozoa with capacitation-like reactions increases, intrauterine insemination should be performed close to the end of ova maturation. Fresh and cooled canine semen can be inseminated earlier since the transferred spermatozoa retain fertilization capability in the female genital tract for on average 4 days. The bitch should show a predominance of cornified epithelial cells in a vaginal smear and the vaginal mucus membranes should show the typical oestrus appearance.

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ENTRE HABITACIONES, UNA APROXIMACIÓN A LA TCI (TARGED CONTROLED INFUSION)

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La farmacología es sin lugar a dudas parte fundamental del ejercicio anestésico, convencionalmente estamos familiarizados con la interpretación farmacodinámica en pacientes anestesiados; sin embargo, poco a poco nos vemos en la necesidad de desmenuzar conceptos que nos ayuden a entender la farmacocinética de las drogas que usamos convencionalmente.

En esta oportunidad buscaremos entender la compartimentalización de las drogas anestésicas, a fin de perfeccionar nuestros protocolos de Anestesia Total Intravenosa o TIVA por sus siglas en inglés (Total Intravenous Anaesthesia) y en la medida de lo posible realizarla por objetivos o TIVA - TCI.

Para ello deseo brindar algunas definiciones tomadas de los libros Farmacología aplicada a la anestesiología y Farmacología Básica y Clínica:

- **Concentración plasmática** (Cp) Es la cantidad o masa de fármaco disuelta en plasma, rutinariamente obtenemos la Cp según la dosis del fármaco administrada; en anestesia aplicamos fármacos en bolo único o múltiple, y en infusión continua a ritmo constante (IRCO). Para mantener una concentración plasmática estable es necesario administrar dosis ajustadas en el tiempo, las cuales se titulan acorde al efecto que se desea obtener, entiéndase que no siempre son iguales; por lo tanto, son dosis cambiantes. La Cp dependerá de la dosis administrada, de la forma como se administre, del volumen de distribución y de la depuración. (1)
- **Volumen de distribución** (Vd) Es el volumen aparente en el cual se distribuirá el fármaco a fin de alcanzar una determinada concentración plasmática, según sus características fisicoquímicas y la forma en que lo maneje el organismo. Varía dependiendo de diferentes factores, entre ellos de su unión a proteínas plasmáticas (pH), su liposolubilidad (Ionizado o no) y la edad, sexo y composición corporal del paciente. Su cálculo se hace de la siguiente forma: Vd = dosis/Cp (1)
- **Modelos de compartimentos** Existen tres tipos de modelos farmacocinéticos: 1. Modelos de compartimentos. 2. Modelos fisiológicos. 3. Modelos híbridos, siendo el modelo de compartimento el que mejor explica el comportamiento de los anestésicos intravenosos. En este modelo el organismo está representado matemáticamente en uno o varios compartimentos que agrupan a los diferentes tejidos dentro del organismo, y el paso del medicamento de uno a otro está representado por la distribución del fármaco que se hace por medio del flujo sanguíneo. Estos modelos pueden ser de un compartimento o de múltiples compartimentos. (1)
- **Biofase o sitio de efecto** Se refiere al tejido en donde se ubica la célula diana. (1)
- **Ke0** Es el enlace entre farmacocinética y farmacodinamia, en esencia es la constante de equilibrio de eliminación del sitio de efecto. (1)
- **Tiempo de vida media con Ke0 (T1/2 Ke0)** Se refiere al tiempo en el cual la biofase y el plasma o compartimiento llegan al equilibrio de la concentración del fármaco, entiéndase como periodo de latencia desde el enfoque de la farmacodinamia. (1, 2)
- **Depuración** Se define como la salida o eliminación de un fármaco de un volumen determinado, entiendáse clearence, aclaramiento o purificación. (1, 2)
- **Ventana terapéutica (VT)** es el rango en el cual se puede utilizar un fármaco sin provocar efectos nocivos, tóxicos o incluso letales en el organismo, y dentro del cual obtenemos el efecto deseado. (1, 2)

Hablar de compartimentalización es una manera simple de referirnos a la distribución de un fármaco dentro del organismo de nuestro paciente, para ello se ha planteado agrupar los diversos tejidos de un ser vivo en compartimentos de alta, media y baja vascularidad, A través de su conceptualización podemos extrapolar lo que sucede con nuestro paciente durante un evento anestésico y de alguna manera limitar los efectos colaterales de los mismos. Entender que nuestro fármaco se distribuye dependiendo de la dosis y

tiempo de administración es farmacocinética, en otras palabras, que le hace el fármaco al organismo. Debemos tener presente que la dosis se refleja en un efecto, el mismo que se puede evaluar clínicamente, entiéndase lo que le hace el fármaco al cuerpo, esto es farmacodinamia. (2)

Para entender la compartimentalización debemos tener presente que los fármacos deben llegar a una concentración plasmática determinada que asegure la disponibilidad de la molécula activa farmacológicamente en la célula que cumplirá el efecto que buscamos; efecto que dependerá, de la activación o no, de los receptores moleculares de la célula diana o del pasaje del fármaco a través de la membrana celular. (2)

Para explicar el modelo por compartimentos se han descrito modelos monocompartimentales, bicompartimentales y tricompartimentales, siendo los mismos muy útiles para entender la cinética de fármacos anestésicos de uso rutinario y correlacionarlos con sus efectos clínicos (1); por ejemplo algunos trabajos sugieren un comportamiento bicompartimental para el Fentanilo y tricompartimental para el Propofol.

Para explicar los conceptos farmacocinéticos descritos, tomaré de ejemplo la cinética del Propofol, podemos conceptualizar que la distribución del fármaco en tres compartimentos: alta, medianamente y pobremente vascularizados. Donde el compartimento central o V1 esta conformado por los tejidos de mayor perfusión y permeabilidad al fármaco, y contiene a la biofase (Cerebro), el compartimento periférico rápido o V2 contiene los tejidos medianamente vascularizados, y el compartimento periférico lento o V3 contiene a los pobremente vascularizados (Grasa). Durante el evento anestésico procuramos mantener la Cp dentro de la VT, teniendo presente la relevancia del Vd en cada uno de los compartimentos para entender su comportamiento farmacocinético (1); pero, debemos tener presente que las características químicas de cada fármaco tienen efecto directo sobre su comportamiento farmacocinético, por ejemplo, la fijación a proteínas y/o afinidad a las grasas (2).

Entonces para administrar Propofol podemos conceptualizar su distribución en tres compartimentos y sus características químicas le dan una alta afinidad a la biofase, dentro del V1, y al V3; sin embargo, su acumulo dentro del V3 se da en proporcionalidad al tiempo de infusión del mismo. Al inicio de la infusión la dosis administrada de Propofol tendrá que compensar la distribución en los tres compartimentos, la Ke0 y la depuración del mismo; con el paso del tiempo se dará la saturación de los compartimentos y solo se deberá compensar la Ke0 y depuración del fármaco. Esto justifica la idea tradicional de administrar una dosis inicial alta y reducirla paulatinamente con el paso del tiempo, la misma que debe ser administrada considerando el efecto clínico deseado. Por lo tanto, la dosis o bolo inicial busca compensar la distribución en los tres compartimentos, la Ke0 y la depuración; luego de reduce en la medida que se satura el V1, V2 y V3 respectivamente, quedando compensar la depuración y la Ke0 (1). Al aplicar el bolo inicial de Propofol debemos considerar la T1/2 Ke0 para la velocidad de infusión y no exceder lo conceptualizado en la VT, entiéndase que bolos rápidos pueden generar la aparición de efectos no deseados al no titular adecuadamente la dosis acorde al efecto deseado, excediendo el rango superior de la VT (1, 2).

Finalmente, el mantenimiento en el tiempo de los efectos buscados con el fármaco elegido se realizarán con bolos repetidos, o de preferencia con una IRCO cuya velocidad de infusión, entiéndase goteo por unidad de tiempo, debe reducirse gradualmente acorde al tiempo que dure la infusión, a fin de limitar efectos colaterales al exceder el limite superior de la VT.

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LEPTOSPIROSIS: PRICE PAID FOR A PREVENTABLE DISEASE

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Leptospirosis is caused by infection with various serovars of *Leptospira interrogans* sensu lato. Organisms are transmitted by direct contact with infected urine, bite wounds or ingestion of infected tissues, or indirectly, through contact with infected water, soil, food or bedding. Survival of leptospires is promoted by stagnant warm water and temperatures between 0 and 25°C, although the spirochete can survive outside these conditions, and even withstand freezing. The seasonality of the disease is variable depending on local climatic conditions. However, some pathogenic strains of *Leptospira* are transmitted primarily through animal to animal interactions because they survive poorly in the environment.

There are over 200 pathogenic serovars, which are grouped into antigenically-related serogroups. Serovars known to infect and cause disease in dogs include Canicola, Icterohaemorrhagiae, Grippotyphosa, Portlandvere, Copenhageni, Hebdomadis, Dadas, Pomona, Ballum, Bratislava, Autumnalis, Bataviae, Australis, and Hardjo; others are also being recognized. In addition, classification of leptospires is gradually moving from predominantly serovar-based classification to that based on genetic typing (genotype-based classification). Each serovar (and more accurately, each genotype) is adapted to a one or more mammalian host species (maintenance hosts). Other hosts act as incidental hosts. Disease in incidental hosts tends to be more severe and the duration of shedding is generally shorter. Maintenance hosts include dogs (Canicola); rats (Icterohaemorrhagiae); small wildlife mammalian species such as voles, skunks, and raccoons (Grippotyphosa); cattle and pigs (Pomona); pigs (Bratislava); cattle (Hardjo); and mice (Ballum). However, the separation between maintenance and incidental host may not be clearly defined, because factors such as the host immune response and degree of exposure may influence whether disease or subclinical infection occur.

Pathogenic leptospires penetrate abraded skin or mucus membranes and multiply rapidly in the bloodstream and tissues, causing renal failure, hepatic injury (usually not hepatic failure) and vasculitis. The disease may also involve the pancreas (pancreatitis), gastrointestinal tract (gastroenteritis), eye (uveitis) and lungs (leptospiral pulmonary hemorrhage syndrome, or LPHS). In humans and likely also in dogs, *Leptospira* can also cause meningitis, which in humans is most commonly manifest as a severe headache.

CLINICAL MANIFESTATIONS

Most infections are subclinical. Disease can occur in any dog breed and at any age; dogs that live in cities may become infected as a result of exposure to rodent reservoir hosts. Disease can occur in dogs as young as 4-6 months of age.

Lethargy, anorexia, vomiting, pyrexia, dehydration, abdominal pain and increased thirst and urination are common signs of acute leptospirosis. Reluctance to move due to myositis, icterus, and uveitis may be noted. Respiratory difficulty may result from pulmonary hemorrhage.

LABORATORY FINDINGS

Leukocytosis, thrombocytopenia, azotemia, hypoalbuminemia and mild to moderately elevated liver enzyme activities are common. Although hyperkalemia has been reported, normokalemia or hypokalemia are more common because of the effect of *Leptospira* on the renal medullary thick ascending limb tubular Na+-K+-Cl- cotransporter. Urinalysis may reveal isosthenuria, proteinuria, glucosuria and casts. Proteinuria is typically low-level (urine protein:creatinine ratio < 5), which helps differentiate leptospirosis (interstitial nephritis) from Lyme nephritis (which involves the glomerulus). Thoracic radiography may reveal a focal or diffuse interstitial to bronchointerstitial pattern; alveolar patterns may represent pulmonary hemorrhage. Occasionally mild pleural effusion is evident. Hepatomegaly, splenomegaly, renomegaly and/or peritoneal effusion may be evident from abdominal radiography. Hyperechoic renal cortices and mild renal pelvis dilation are occasionally seen with abdominal ultrasound.

DIAGNOSIS

Identification of leptospirosis requires a high clinical suspicion for the disease based on knowledge of the range of clinical presentations that suggest leptospirosis. Currently available diagnostic tests include PCR, serology using the microscopic agglutination test (MAT), and in-clinic serologic

assays that detect IgG/IgM (e.g., SNAP Lepto, IDEXX Laboratories), or IgM (e.g., WITNESS Lepto, Zoetis).

In the MAT, respective titers are provided for each of several different serovars in order to increase the chance of antibody detection. Studies in humans and dogs have shown that the serovar with the highest titer can vary over time and that paradoxical cross-reactivity to multiple serovars occurs after exposure to a single serovar. Thus, the MAT does not reliably predict the infecting serovar, and therefore should not be used for this purpose. Titers with any serologic test may be negative in the first week of illness because of the short incubation period and delay in antibody production. Low positive or negative titers after at least one week of illness suggest leptospirosis is not present. Overdiagnosis results from misinterpretation of positive serologic test results. Positive titers early in the course of an illness may reflect residual post-vaccination titers or prior subclinical infection, and are not diagnostic for the disease. Demonstration of a fourfold rise in titer is required over a 1-2 week interval. In acutely ill dogs (< 1 week of illness), it is the author's opinion that leptospirosis serology should only be performed in a paired fashion or not at all, because of the limited utility of a single positive titer, regardless of its magnitude. Postvaccinal titers against Icterohaemorrhagiae, Canicola, Grippotyphosa and Pomona occasionally rise as high as 1:6400 for a few months after vaccination, and these can interfere with interpretation. The results can also vary dramatically between laboratories. Use of a laboratory with a high level of quality control is recommended, or a laboratory that participates in the International Leptospirosis Society's proficiency testing scheme.

In-clinic serologic assays yield qualitative (positive or negative) results, and are useful for screening dogs for the presence or absence of antibodies. Should these kits yield negative results, then the clinician should consider whether it may be too early for the animal to have developed antibodies (as can occur with the MAT). Should these kits yield positive results, then the clinician should consider whether a positive result may reflect recent previous vaccination for leptospirosis or previous exposure without clinical disease. Clinicians should consider reflex testing with MAT in order to obtain a quantitative titer if positive results occur using in-clinic serologic tests, followed by convalescent serology 1-2 weeks later in order to document a change in titer. Additional clinical validation of these assays in different regions of the United States would be helpful to confirm their sensitivity and specificity.

Culture is difficult because of the fastidious growth requirements of leptospires and the need for specialized media and darkfield microscopy, but there have been recent advancements in methods that accelerate growth of leptospires and may overcome these hurdles. PCR assays do not provide information about the infecting serovar, although they have been used to provide information on genotype. The sensitivity and specificity of PCR may vary geographically depending on the serovars present and shedding patterns that occur for those serovars. The sensitivity may also be higher very early in the course of illness and in dogs that have not received any treatment with antimicrobials. Positive results have been detected in the urine of some healthy dogs, especially those that are free-roaming, from kennels, or shelters (acting as reservoir hosts). PCR assays are best performed on blood AND urine concurrently because urinary shedding begins 10 days after the onset of infection.

TREATMENT

Specific treatment involves initial use of parenteral penicillin derivatives for leptospiremia. Ampicillin is recommended (20 mg/kg IV q6-8h) for as long as the patient is vomiting or appears nauseated. Treatment should then be changed to doxycycline (5 mg/kg PO q12h) for 2 weeks, in order to optimize elimination of the organism from renal tubules. Supportive therapy is also indicated for acute kidney injury. Approximately 50% of the patients with leptospirosis at the author's institution are dialyzed. Recovery of renal function can continue for weeks to months after discharge from hospital, even in the absence of ongoing dialysis.

PREVENTION

Leptospira vaccines are generally safe and efficacious and studies suggest they provide a minimum of a 1-year duration of immunity. Although it was prevalent when the two-way (Canicola and Icterohaemorrhagiae) vaccines were in widespread use, vaccine failure appears to be uncommon with the current 4-serovar vaccines. Reaction rates of newer vaccines appear to be approaching those of distemper-hepatitis-parvovirus vaccines, even in small breed dogs. Vaccination against pathogenic leptospires is strongly recommended, even for small breed dogs that are confined to urban backyards, because of the possibility of infection as a result of direct or indirect rodent exposure. There have been recent outbreaks of leptospirosis in dog populations from dog daycare environments and urban concrete backyards. Minimizing access to potential maintenance hosts also should help to prevent infection.

PUBLIC HEALTH RISK

Leptospirosis remains an important zoonosis, although most documented human leptospirosis results from

exposure to rodents or other reservoir hosts like cattle, or recreational activities that involve water, rather than contact with dogs. Human leptospirosis is typically a 'flu-like illness', but in some cases may be associated with vomiting, diarrhea, shock, jaundice, renal failure, pneumonia, meningitis, or abortion. Any animal with acute kidney injury should be treated as a suspect. Warnings should be placed on cages, gloves should be worn while handling these dogs and bleach or iodine-based disinfectants should be used to clean areas soiled with urine. Contact precautions can be reasonably lifted after 48-72 hours of specific antimicrobial therapy.

MAMMARY CANCER HISTORY AND DIAGNOSIS IN DOGS AND CATS

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ABSTRACT BODY:

Questions about mammary gland tumors dogs.

Mammary tumors are the most common neoplasm in intact bitches, so pet owners invariably have questions and doubts. In our lecture, we will answer the most frequently asked questions about mammary tumors in dogs and cats. Answers to the feline questions are in a separate abstract by Dr. Ann Hohenhaus.

Does my pet have a risk of developing a mammary tumor?

Three main risk factors:

1. Age Breast tumors are rare in dogs under 5 years of age. Much more common in dogs aged 7-8 years. Malignant tumors are much more common in older animals than in younger animals.
2. hormonal status (not neutered animals)
3. breed (predisposed, spaniels, setters, German shepherds, poodles, terriers; in the publications of Japanese researchers, small breed dogs were more prone to mammary tumors than large breed dogs, but tumors are more often benign.

Additional risk factors: Overweight before puberty

Can mammary tumors occur in a male dog?

Yes, but neoplasms of the mammary glands in males are very rare, more often they are benign, they make up only 1% of animals with neoplasia of the mammary gland.

How can I identify a mammary gland tumor in my dog?

Most often, the tumor is detected while stroking or combing the dog.

Are swellings in the mammary gland area away a tumor of the mammary gland?

In the differential diagnosis of swelling in the mammary gland include bi mastitis (if the bitch is lactating); skin tumors: mast cell tumors, melanoma or subcutaneous tissue (lipoma); masses not associated with mammary gland tissues: lymphadenomegaly of inguinal lymph nodes of various nature (inflammatory, systemic or metastatic tumor process),or an inguinal hernia, Physiological increase in mammary tissue occurs in different phases of the estrus cycle, including during psuedopregnancy.

How are mammary tumors in dogs treated?

The main treatment for mammary tumors in dogs is surgical excision.

Can I wait and see if the tumor gets large before performing surgical removal of the tumor?

It is better to excise the tumor immediately after detection if its size is 5 mm or more. Various studies have shown that the larger the mammary tumor, the more likely it is to be malignant and the more likely it is to potentially metastasize. Neoplasms up to 1 cm in diameter ae benign in 2/3 of cases. Neoplasms more than 2 cm in diameter will be malignant in 2/3 of cases. Over time, benign tumors can become malignant if they are not removed.

Will a fine needle aspiration (FNA) cytology alone, without surgery, determine whether or not my dog has cancer in the mammary gland?

FNA of a mass in the mammary gland area is performed primarily to rule out other tumors (lipomas, mast cell tumors enlarged lymph nodes). Reliable differentiation between benign and malignant tumors is difficult based on a FNA cytology. This requires a histological examination.

What preoperative investigations are required before the operation? Will blood tests be helpful?

Routine blood tests (biochemical profile and complete blood count) are done to assess the general condition of the dog before surgery, including anesthetic risks, but do not confirm or rule out cancer. Rarely, paraneoplastic syndromes have been described in dogs with mammary tumors: hypercalcemia, hypereosinophilia, and hypoglycemia. A thoracic radiograph in at least 2 views and abdominal ultrasonography may be recommended for staging to rule out distant tumor metastasis prior to surgery. Only 10% of dogs that do not have distant chest metastases may have metastases in other organs (liver, central nervous system, skin, kidneys, bones).

What other paraneoplastic syndromes can be observed in patients with mammary tumors?

Hypertrophic osteopathy in dogs with lungs metastasis.

Do mammary tumors in dogs have tumor markers detectable in a blood test?

The results of studies regarding CEA and Cancer Antigen 15.3 (CA15.3) in serum are inconsistent: in one study, dogs with mammary malignancies had higher levels of CEA and Cancer Antigen 15.3 (CA15.3), in another study these data were not confirmed. In another study, CA15.3 and LDH levels were significantly higher in canine patients with breast tumors and metastases. However, for the routine diagnosis of tumor malignancy or the presence of metastases, these studies are still insufficient.

How much surgery is needed?

Small neoplasms about 5 mm in size are removed by nodulectomy. The volume of each operation is individual for each animal and tumor type. Simple or radical mastectomy may be performed.

Is a histological examination of the excised tumor necessary if cytology was performed before the operation?

Histological examination of the neoplasm and excised l lymph nodes is mandatory for the final determination of the type of tumor and the exclusion of regional metastasis. This helps to determine the need for further therapy and prognosis.

What factors are unfavorable for prognosis in a patient with malignant mammary tumors?

Unfavorable prognostic factors are:

-large size of the tumor (more than 5 cm)

-ulceration of the tumor surface

- regional or distant metastasis

- histological types: osteosarcoma, solid, anaplastic, micropapillary-invasive carcinoma

- inflammatory carcinoma

- morphological criteria for high tumor aggressiveness: invasive growth involving blood and lymphatic vessels, low tumor differentiation, high proliferative indices, absence of progesterone receptors

-incomplete surgical removal

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MAMMARY CANCER HISTORY AND DIAGNOSIS IN DOGS AND CATS

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Abstract Body:

Pet owners often don't realize the lump they have found while giving a tummy rub to their cat or dog could be cancer. They often arrive in the veterinarian's office with many questions about how their pet got this tumor, if it is malignant, what tests are used to confirm the diagnosis and what the treatment will be. Paramount in their minds is how long their cat will live if a malignancy is the diagnosis. This presentation will answer questions commonly asked by pet owners about the history and diagnosis of mammary gland tumors (MGT). Answers to the canine questions are in a separate handout by Dr. Natalia Ignatenko.

Feline questions

I found a lump on my cat's tummy when I was petting her. What could it be?

While MGT comprises 10-20% of all feline tumors, other types of tumors can be found in the mammary area.[1] Data from the Small Animal Veterinary Surveillance Network based in the United Kingdom indicates squamous cell carcinoma is the most common skin tumor in the cat.[2] Other malignant tumors found in the skin include soft tissue sarcoma, mast cell tumors, lymphoma and other carcinomas. Benign skin tumors such as, adenomas and lipomas were also reported.

How did my cat get this tumor?

A number of risk factors have been identified for feline MGT. Most cats that develop MGT are older; the mean age is between 10-12 years.[3] MGT are most common in female cats, but male cats can develop MGT.[4] Cats receiving progestin therapy and female cats spayed after 1 year of age have an increased risk. Several studies have reported Siamese cats have a increased risk of developing MGT.

Could we just leave the lump there and see if it gets bigger?

The longer the tumor is present the larger the tumor becomes, the worse the prognosis is. In one study, cats with MGT > 3 cm had a survival time between 4-12 months where cats with MGT less than 3 cm had a survival time of 21 months.[5,6] Larger size is associated with shorter survival in male cats with MGT. Larger tumors are more likely to be ulcerated and ulceration may indicate a poor prognosis. The longer a tumor is present, the greater the chance the tumor will metastasize.

Could the tumor have spread elsewhere?

The majority of cats with MGT will develop metastatic disease. Before performing a mastectomy to remove the tumor, clinical staging should be performed. The goal of clinical staging is to determine the extent of tumor. Cats with MGT that has already spread to the lungs, liver or pleura, may not be a good surgical candidate and may not benefit from surgery.

	Location of metastatic mammary gland tumors	
	Weijer[6]	Moulton[7]
Lungs	85%	83%
Lymph nodes		83%
Pleura	76%	42%
Adrenal glands	19%	
Pleura	18%	
Spleen	10%	
Kidneys, ovaries, other organs	≤8%	24% liver

Is there something else we can do besides surgery?

Veterinarians recommend surgery for feline mammary gland cancer for two reasons. First removal of the tumor provides material for diagnostic histopathology. Second, surgery is the most important treatment for feline mammary gland cancer. If confirmation of malignancy before surgery is important, a fine needle aspiration cytology can be submitted. This test will help determine if the lump is benign or malignant and this information will help direct our treatment approach.

Do you need to submit the lump for a biopsy after you remove it?

Yes! A biopsy is critical for diagnosis since many different types of tumors can occur in the mammary gland area. If the tumor is a MGT, the pathologist will assign a grade to the mammary gland tumor which helps the veterinarian to give a prognosis and directs treatment.

My cat had a mammary gland tumor removed last year and now is coughing.

My cat with a mammary gland tumor removed last year is breathing funny.

My cat had a mammary gland tumor removed last year and she seems painful and reluctant to walk.

All three of these scenarios require diagnostic imaging to search for metastatic lesions causing these clinical signs. The most common sites of metastatic disease are lymph nodes, lungs, liver and pleura.

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COGNICIÓN CANINA: ESTAMOS ANTE UN CAMBIO DE PARADIGMA?

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ABSTRACT BODY:

INTRODUCCIÓN

La cognición se define como la capacidad de los seres vivos de procesar la información a partir de la percepción (estímulos que nos llegan del mundo exterior a través de los sentidos), el conocimiento adquirido con la experiencia y las características subjetivas que permiten integrar toda esta información para valorar e interpretar el mundo.

La capacidad cognitiva de cada especie viene determinada por su historia evolutiva, ya que cada especie ha necesitado adaptarse a las condiciones en las que ha evolucionado. La capacidad cognitiva se define mediante 6 procesos:

- Percepción (o UMWELT).
- Atención.
- Memoria.
- Lenguaje.
- Aprendizaje.
- Pensamiento.

El objetivo de esta charla será comprender la capacidad y las características cognitivas del perro y, paralelamente, mostrar algunas aplicaciones en la clínica diaria de medicina del comportamiento. En los últimos años se ha avanzado enormemente en el conocimiento de la cognición canina, especialmente gracias al trabajo de múltiples grupos de investigación alrededor del mundo. En definitiva, se desmentirán algunas de las ideas preconcebidas de los perros que afirmaban cosas como que solo actúan en función de las consecuencias de sus actos, o que no tienen previsión de futuro y viven el presente.

PERCEPCIÓN CANINA (UMWELT)

El *umwelt* de una especie hace referencia a cómo percibe su entorno. Para entender el *umwelt* de una especie se deben definir las características de sus órganos de los sentidos. Aunque para definir correctamente el *umwelt* del perro se deberían describir todos los sentidos, en esta charla se abordarán únicamente la vista, el oído y el olfato.

Vista

Los ojos de los perros se diferencian a los de los humanos porque son mucho más sensibles a la luz, pero perciben las imágenes con menor nitidez. Además, los perros son bicrómicros (ven en combinaciones de dos colores), a diferencia de las personas que vemos en combinaciones de tres colores. El color que no perciben los perros es el rojo y sus combinaciones, por lo tanto, también son percibidas de forma diferente.

Olfato

El olfato canino tiene dos particularidades que deben destacarse:

- Su sensibilidad olfativa es, en términos generales, muy superior a la del ser humano en la mayoría de las sustancias analizadas.
- Presencia del órgano vomeronasal (VNO). El VNO, que no está presente o es vestigial en las personas adultas, juega un papel fundamental en la percepción de las feromonas que, a su vez, tienen una función muy importante en su comportamiento.

Oído

Para definir la capacidad auditiva de una especie se debe caracterizar la frecuencia y la intensidad del sonido que es capaz de percibir. En el caso de los perros la intensidad de sonidos que percibe es muy similar a la de los seres humanos, sin embargo, el rango de intensidad es superior, ya que pueden perciben ultrasonidos que pasan desapercibidos para el oído humano.

ATENCIÓN

A diferencia de otros mamíferos domésticos, se sabe que los perros focalizan la atención en los seres humanos y lo que están haciendo e, incluso, como se verá más tarde, tratan de imitar los comportamientos que hacen. Además, no solo centran su atención en lo que están haciendo, sino que también se focalizan en lo que los humanos están centrados, aunque no estén haciendo ninguna acción concreta. Finalmente, pero no menos importante, se sabe que centran mucho más su atención hacia los humanos con los que tiene una relación estrecha (vínculo), con los que tienen más confianza.

Muy probablemente esta predilección de los perros hacia las personas y sus intereses, se deba a la historia evolutiva conjunta y, de hecho, haya supuesto una ventaja evolutiva a lo largo de la convivencia entre perros y personas.

Finalmente, también es importante destacar que el estrés juega un importante papel sobre la capacidad de

atención de los perros (y de los animales en general). En resumen, se sabe que la atención aumenta a medida que aumenta el estrés del animal, hasta que llega a un punto óptima de atención. A partir de este, si se continúa aumentado el estrés, la atención decrece. Desde un punto de vista práctica es tremendamente importante para entender las dinámicas de las sesiones de modificación de conducta en un servicio de medicina del comportamiento e, incluso, para entender la capacidad de aprendizaje en cualquier sesión de entrenamiento canino.

MEMORIA

Igual que ocurre en muchas otras especies, el aprendizaje depende directamente de la capacidad de poder almacenar los recuerdos y las experiencias. Existen diferentes tipos de memoria y se en los perros se ha demostrado la presencia de la memoria a corto y a largo plazo, y más recientemente la memoria episódica.

De hecho, la memoria episódica tiene un especial interés ya que se utiliza en los seres humanos como una forma de explicar la "autoconciencia", el ser consciente de uno mismo. Se ha demostrado que los perros son capaces de "ponerse en el lugar en el que estaban" hace unos días para recordar algún suceso que había tenido lugar alrededor de ese recuerdo. Esta capacidad es más fiable para demostrar autoconciencia que las pruebas de autoreconocimiento en un espejo.

LENGUAJE

Los perros se comunican entre ellos mediante de muchas maneras, a través de las vocalizaciones (aullidos, ladridos, gruñidos…), a través de la vista (posturas corporales, faciales, etc…), del olfato (feromonas, por ejemplo) y a través de su comportamiento. A través de esta comunicación expresan sus estados emocionales y sus intenciones con otros animales de su misma especie o, incluso, de otras (por ejemplo, los humanos). Esta comunicación les permite mantener las relaciones con otros animales.

Es especialmente destacable en el caso del perro que su capacidad comunicativa con el hombre. Se ha demostrado que el perro es capaz de entender las señales comunicativas de las personas sin que nadie se las haya enseñado antes o, incluso, que tiene una intención comunicativa con las personas. De hecho, no solo eso, sino que es capaz de ponerse en el lugar de la persona para tomar decisiones y, también, de adaptar su conducta en función de las limitaciones comunicativas que tenga el "interlocutor".

APRENDIZAJE

La capacidad de aprendizaje del perro es más compleja de lo que se la ha atribuido durante mucho tiempo. Más allá de la capacidad de aprender a través de procesos no asociativos (habituación y sensibilización) y asociativos (condicionamiento clásico y operante), en las últimas décadas se ha demostrado que también pueden aprender a través de procesos de descarte (*fast mapping*) y de imitación, no solo de otros perros, sino que también imitando a las personas.

Estos tipos de aprendizajes más complejos, permiten abrir las puertas a nuevas técnicas de aprendizaje canino y de modificación de conducta.

PENSAMIENTO

Quizás a día de hoy sea el componente menos estudiado de la cognición canina debido a su complejidad. Pero las respuestas de conducta que ofrece el perro teniendo en cuenta todos los aspectos que se acaban de mencionar hasta ahora son el reflejo de este pensamiento del animal. Lo que queda claro, es que su capacidad cognitiva va mucho más allá de ser meros autómatas que responden en función de unas asociaciones hechas con diferentes estímulos.

OTROS PUNTOS IMPORTANTES

La capacidad cognitiva de los perros ha evolucionado conjuntamente con su vida al lado del ser humano. Aunque su capacidad en algunos aspectos es muy superior a la de su ancestro, el lobo, u otros animales, lo cierto es que el perro no es mejor que otros animales en todas las pruebas cognitivas que se han realizado. De hecho, son mucho peores que los lobos, por ejemplo, en la resolución de laberintos o en entender ciertos "principios de física básica". Durante la charla se discutirán todos estos puntos con detenimiento.

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CUÁLES SON LAS COMPETENCIAS PARA EL MÉDICO VETERINARIO DEL FUTURO?

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las competencias al veterinario del futuro son basicamente entender de las técnicas de diagnostico y tratamiento, y basicamente de negocios, actuar como empresario del SIclo XXI. No basta saber la medicina veterinaria, sino tambiem como ser líder, manejar os indicadores de salud del negocio, las mejores prácticas.

Como lidear equipos de jóvenes, com ideias distintas de la generación de los mayores

Que tengo que aprender ademas de la técnica de tratamientos

Como abodar equipos en conflictos

Como manejar los nuevos tiempos de M&A (merger and acquisition), compras y ventas de clínicas

Cuales son las metodologias de gestión que puedo aplicar

y mucho más.

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ACUTE AND CHRONIC USE OF ANTI-INFLAMMATORIES

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ACUTE AND CHRONIC USE OF ANTI-INFLAMMATORIES

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Non-steroidal anti-inflammatory drugs (NSAIDs) are the most widely used analgesics in veterinary medicine. They are crucial for the treatment of acute and chronic pain in dogs and cats. In the acute pain setting, as in the perioperative period, NSAIDs are usually part of a multimodal treatment in combination with local anesthetic blocks and opioids (i.e. concomitant use of two or more classes of analgesics, which results in reduction of doses, synergistic effects, as well as reduced morbidity and mortality). In the chronic pain setting, as in osteoarthritis, NSAIDs are the first line of pharmacological treatment, and are usually associated with other analgesics or non-pharmacological treatments such as weight control, physical activity, and environmental changes, among others (Lascelles et al. 2007; Monteiro & Steagall 2019).

The anti-inflammatory and analgesic effects of NSAIDs are related to the inhibition of the expression of cyclooxygenase (COX) enzymes in cell membranes. In a simple approach, it is well accepted that COX-1 enzymes are generally related to constitutive functions of cells which maintain homeostasis, whereas COX-2 enzymes are generally induced after tissue injury in order to produce inflammatory mediators that amplify nociceptive input (pain) to the spinal cord. Therefore, most of NSAIDs´ analgesic effects are related to the depression of COX-2 induced-effects, and most of the adverse-effects are related to the depression of COX-1 cytoprotective effects (Lascelles et al. 2005a; Mathews et al. 2014).

NSAIDs can have different effects in different organ systems based on COX inhibition which can potentially cause adverse effects including the gastrointestinal tract, the hematopoietic system and the kidneys. There is also hepatic adverse-effects which are not related to the inhibition of COX enzymes, but rather, are considered as an idiosyncratic effect of the drug (i.e., are unpredictable, are not dose-dependent and are not consistently inducible).

The true incidence of NSAIDs-related adverse-effects remains unknown. The most common adverse-effects of NSAIDs are gastrointestinal signs (vomiting, diarrhoea and anorexia). These are generally self-limiting or require minimal supportive treatment. A systematic review of studies in which dogs were administered NSAIDs concluded that the overall incidence of adverse drug experiences cannot be calculated from the data in the literature. Nevertheless, estimates for the most serious adverse effects suggest that they occur at a very low frequency, and are usually related with erroneous administration of these drugs (Monteiro-Steagall et al. 2013). Furthermore, a retrospective study looking at gastrointestinal perforation following NSAID treatment also revealed that 90% of the cases had some error in the administration (Lascelles et al. 2005b). Risk factors for the development of serious adverse-effects may include the administration “off-label,” at higher doses than recommended, for prolonged periods of time, or in combination with corticosteroids or a 2nd NSAID.

Guidelines to prevent the development of serious adverse-effects following NSAID administration are available (Lascelles et al. 2005a). Patients should be carefully selected including screening for pre-existing diseases, current medications and possible drug interactions. Good client communication is imperative when NSAIDs are administered for long-term. In the acute pain setting, fluidtherapy and monitoring of blood pressure should be performed during surgical procedures in which NSAIDs were administered pre-emptively (i.e., before the painful stimulus). A wash-out period is recommended when switching NSAIDs.

Only veterinary-approved NSAIDs should be used in animals and label recommendations should be respected. Ibuprofen has a narrow margin of safety and is the most common drug involved in drug exposure to dogs and cats that is reported to the American Society for Prevention of Cruelty to Animals (Jones et al. 1992). Paracetamol (or acetaminophen) is toxic in cats. Veterinarians are encouraged to report adverse-effects to the pharmacovigilance service of drug companies. This will reinforce post-marketing and safety data as well as provide evidence for the development of safer NSAIDs.

Cats have some particularities due to limited hepatic glucuronidation and potential greater incidence of adverse-effects (Sparks et al. 2010). However, NSAIDs that are labelled for use in cats such as meloxicam and robenacoxib have good efficacy and safety even for long term administration. Additionally, although NSAIDs would be normally contra-indicated in renal patients, recent research is indicating otherwise (Monteiro et al. 2019). Based on available evidence, it seems that meloxicam and robenacoxib can be safely administered to cats with concomitant chronic painful conditions and chronic kidney disease.

In this lecture, I will briefly review the pharmacology of anti-inflammatories including NSAIDs, present the most up to date scientific evidence on the efficacy and safety of these drugs, and discuss their clinical use for acute and chronic pain management in dogs and cats.

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ACUTE PAIN IN CATS: MORE THAN MEDICATION

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ABSTRACT BODY:

The International Association for the Study of Pain (www.iasp-pain.org) have run several global campaigns related to acute pain. In their 2010, campaign they introduced the advice to "Anticipate, Assess, Alleviate", or the "three As" of pain management. This emphasizes three key approaches to acute pain management. They also emphasized that management of acute pain is not a specialist's job and is a condition that medical professionals encounter every day. It is important to prevent or alleviate acute pain to decrease the distress it causes but also to enhance nursing care, permit sleep and rest, prevent ileus, decrease the stress response, mitigate catabolism, and prevent poor wound healing.

Clinically, there are two phases associated with surgery; the first is the sensory input arising directly from the surgery itself and the second is from the resultant, more prolonged inflammatory response. Acute pain, now termed adaptive pain, begins with thermal, chemical or mechanical stimulation (nociceptive stimulation) of the peripheral nervous system which is amplified by inflammation (transduction). Adaptive pain is protective and if treated correctly is reversible. Signaling from peripheral sites (transmission) causes central plasticity (sensitization) which can occur quickly as a result of changes in N-methyl-D-aspartate (NMDA) receptors in the dorsal horn of the spinal cord. If this goes unchecked, maladaptive pain, which we have termed chronic pain in the past can occur. Maladaptive pain serves no biological function.

Preventive analgesia The term pre-emptive has been used to emphasize that administration of analgesics prior to surgery is beneficial. However, for many surgical procedures, one treatment is not sufficient therefore the term "preventive analgesia" is preferred to describe analgesic treatment before, during and after surgery. It is now understood that an effective and appropriate level of analgesia must be maintained throughout the duration of the inflammatory response. If analgesic therapy is withdrawn too early, "re-initiation" of pain is possible1. To prevent prolonged or persistent post-operative pain, analgesic therapy should be started prior to surgery (or a soon as possible after trauma), maintained during surgery, be robust in the immediate post-operative period and not withdrawn until the inflammatory response has subsided. Duration of treatment will depend on the degree of surgical trauma and resultant inflammation.

Prevention and alleviation of pain – pharmacologic techniques The major analgesic drug classes employed for acute pain management are:

Opioids

Non-steroidal anti-inflammatory drugs (NSAIDs)

Local anesthetics

NMDA antagonists (e.g., ketamine)

Alpha2–adrenergic agonists

Other drugs that are not analgesics can play a role in patient comfort. Nausea and vomiting are detrimental ad also unpleasant therefore maropitant and ondansetron may be an important component of a perioperative plan. In the face of acute pain, gabapentin is unlikely to act like a traditional analgesic but it's calming, and anxiolytic effects are valuable, and enhance nursing care.

Multimodal analgesia

This term describes the use of more than one drug for the different classes of analgesic used at the same time to provide more robust analgesia because they work at different parts of the pain pathway. For acute pain the most common combination is an opioid plus an NSAID. However, the addition of a local anesthetic is ideal.

Medication challenges

Cats are notoriously difficult for owners to medicate. Regardless of how appropriate your choice of drug is, low compliance and treatment failure is common. The results of an online owner survey highlighted the struggles owners have. The survey contained thirty-five questions and asked about experiences administering different forms of medications, preferences for how drugs were formulated, support provided from veterinary practices, the cat's behavioral responses, and impact on the owner's relationship with their cat because of the need to medicate them. The authors analyzed 2,507 responses from 57 countries2. Here are some of the key findings from the study:

35% of owners failed to complete a course of medication

50.7% of cat owners reported they were "sometimes" or "never" given information or advice on how to

administer medication by their veterinary team

Tablets were the most difficult to administer, with many owners preferring liquids

51.6% of owners reported that medicating their cat had changed their relationship with them; this is a breakdown of the cat-owner bond, and we must find ways to avoid this.

The most frequently reported challenges included the cat spitting out tablets (78.7%), trying to bite or scratch the owner (77.0 %), refusing medication in food (71.7%), and running away from the owner (52.7%).

Great thought should be given to how post-operative drugs will be administered both in the clinic and by the caregiver at home.

The appropriate use of different analgesic drugs are discussed in detail in other WSAVA sessions. In addition, extensive information is available in the 2022 ISFM Consensus

Guidelines on the Management of Acute Pain in Cats3. Non-pharmacologic treatments are valuable but often overlooked and this is what we will discuss in more depth (Figure 1).

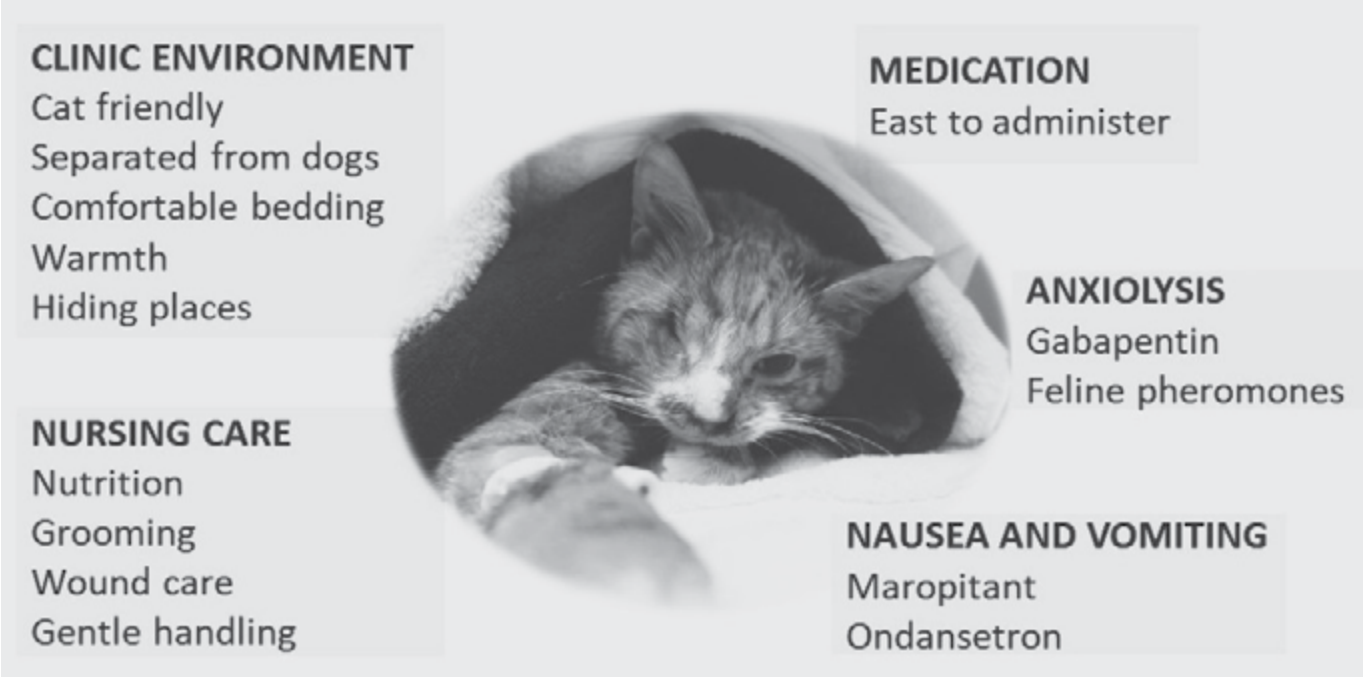

Figure 1. The many ways that comfort can be improved in cats

Non-pharmacologic techniques

Multimodal analgesia is not restricted to using several drugs that act at different sites in the “pain pathway”. It includes non-pharmacologic therapies such as icing, acupuncture, massage, laser therapy and addressing stress and anxiety, which means we are practicing integrative pain management. In humans addressing stress and anxiety is often part of a treatment plan for both acute and long-term pain. Research on neural pathways suggests there is substantial overlap between how the brain processes emotional and physical pain4,5.

Good nursing care (warm, dry, comfortable bedding), non-restrictive bandages and feline friendly handling can have a positive effect on a patient’s recovery. The environment where a cat recovers is also important, and it is essential to reduce visual, olfactory and auditory stimulation from dogs. All cats must have a place to hide in their cage – hiding is an important coping mechanism for cats when they are stressed or anxious.

Cold therapy

Cold therapy is a valuable non-drug therapy for acute pain(6). Cold therapy works in many ways including but not limited to decreasing activation of tissue nociceptors, slowing conduction velocity along peripheral nerves, reducing swelling secondary to vasoconstriction and activation of specific channels e.g., those of the Transient Receptor Potential (TRP) cation channel family6.

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059 / #59

NUTRITIONAL MANAGEMENT OF CANINE AND FELINE UROLITHIASIS

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ABSTRACT BODY:

NUTRITIONAL MANAGEMENT OF CANINE AND FELINE UROLITHIASIS

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This topic covers uroliths that range from those that have extensive published information on pathogenesis and proven clinically effective interventions to those where only incomplete information is still available to guide our decisions. The six most common urolith types will be discussed though it is noted that individual urolith prevalence varies with species, breed, age, sex, geographic location, anatomic location, urine pH, urinary tract infections, anatomic or metabolic abnormalities and diet. It is critical to send uroliths for mineral analysis wherever possible, as specific therapy often depends on urolith composition, though the addition of water to the diet or addition of salt to drive voluntary water intake almost always helps to reduce recurrence. Uroliths generally form secondary to one or more underlying abnormalities, so investigation into possible influences and appropriate management if confirmed can be beneficial in management.

Magnesium Ammonium Phosphate (struvite) uroliths in dogs form most often secondary to a urinary tract infection. They can potentially be dissolved with specific diet and antimicrobial therapy, but there are some risks and complications with this approach. Generally, control of recurrent urinary tract infection in dogs is sufficient to reduce the risk of struvite urolith recurrence. Cats generally form struvite uroliths in sterile urine, so long-term feeding of diets formulated to reduce struvite urolith recurrence is warranted. These diets tend to be higher in moisture or salt, lower in mineral precursors and are formulated to maintain urine acidity in very tight ranges to assist with maintaining solubility of struvite precursors.

Calcium Oxalate urolithiasis now occurs in both dogs and cats with a frequency roughly approximating struvite uroliths, but there are marked variations over time and geographically. Due to difficulties in creating a reproducible model of this disease, there is limited prospective study data and our current understanding of its pathogenesis is incomplete, despite considerable effort being expended. It is generally accepted that there are dietary and genetic influences involved in the formation of these uroliths. Dissolution of calcium oxalate uroliths is not possible and efforts to reduce recurrence tend to focus on increasing water intake, lowering oxalate (or precursor) intake, moderating protein and calcium intake, supplementation of potassium citrate or potassium chloride, and weight management. It would also to be prudent to avoid calcium and/or vitamin D supplements, vitamin C supplements, treats with high oxalate content, treats with high collagen content, strongly acidifying diets or drugs that promote urinary calcium excretion. New studies show some promise for supplementation of omega-3 polyunsaturated fatty acid supplementation of diets and management of hypertriglyceridemia when present. Close monitoring of individual response to medical interventions is advised at this time. It is recognized that recommendations are likely to be adjusted as more is discovered about this condition.

Ammonium Urate uroliths are more common in dogs than cats. In cats they are thought to occur due to genetic renal tubular defects or portosystemic shunts. In non-dalmatian dogs they are most commonly associated with portovascular abnormalities. Dalmatian dogs have reduced or absent ability to convert uric acid to allantoin, so form urate uroliths more often than other breeds, with males being particularly at risk. Along with exploring possible underlying causes, controlling recurrence tends to focus on nutritional management with increasing water consumption and reducing dietary purine intake.

Calcium Phosphate uroliths are uncommon and are usually related to hypercalcuria. Calcium Phosphate is more commonly encountered as a minor component or a shell around other uroliths, likely related to urine alkalinity. They cannot be dissolved and must be surgically removed for identification. Prevention strategies are aimed at identifying and removing any underlying medical contributors, and then increasing water intake while controlling levels of precursors in the diet and avoiding urinary alkalization.

Cystine uroliths are increasing in frequency in the USA, but are still more common in Europe. They are

caused by an inherited error of metabolism that affects reabsorption of amino acids in the renal tubules. Cystine stones occur more often in male dogs and can be androgen responsive. Cystine stones occur with equal frequency in male and female cats. Nutritional strategies for dissolution and prevention encapsulate increasing water intake, reducing dietary protein and eliminating aciduria.

Silica uroliths are rare in all countries except Mexico, where their prevalence is believed to be related to high water silica levels. They occur more frequency in male dogs and dietary silica content is also thought to be a risk factor. They are not able to be medically dissolved and nutritional prevention strategies include increasing water intake, reducing water and diet silica content, and avoiding urinary acidification.

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060 / #58

PREGNANCY, PARTURITION AND PUERPERIUM IN SMALL ANIMALS

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ABSTRACT BODY:

Introduction

Pregnancy, parturition and the puerperium places the animal under extraordinary stresses and complications may arise. A chronological landmark from which all events are timed is important. Cats are mainly, but not exclusively, induced ovulators (1). Mating dates are assumed to coincide within a 24 hour period after multiple matings during oestrus. In the bitch the chronologic landmark is the LH surge and is generally estimated by the concomitant preovulatory rise in plasma progesterone concentration (6.4 nmol/L) (2).

Pregnancy

Pregnancy commences at the point of fertilisation and practitioners should be mindful of medications administered to pregnant queens and dams that may be harmful to their offspring (3).

Pregnancy diagnosis by ultrasound examination is routinely encouraged to diagnose causes of early pregnancy loss and distinguish resorption from non-conception. Canine brucellosis and canine herpes virus remain a constant threat and challenge to dog breeding and may cause failure to conceive, resorption, abortion, mummification, stillbirths and neonatal death. Habitual abortion may be very frustrating in queens and definitive diagnosis and prevention is problematic. Monitoring of progesterone curves during pregnancy may elucidate cases of hypoluteodism in both the queen and the bitch (3). Short acting gestagen supplementation maintains pregnancy in such cases but should be terminated timeously to allow for onset of spontaneous parturition. Any vaginal discharge during pregnancy, besides a scant mucosal discharge (greyish white) originating from the cervical slime-plug, is considered abnormal in the bitch and queen and should be viewed with suspicion.

Implantation in the bitch and queen takes place at around 14-21 days after the LH surge and stressors during this crucial period may cause embryonal death. Serial ultrasound examinations are invaluable in monitoring pregnancy but caution should be exercised in using ultrasound data to decide when to perform caesarean sections in isolation of other signs of impending parturition. Significant physiological adaptations to pregnancy occur in the second term of pregnancy and these may be inadequate in some breeds carrying large litters or when pathological conditions such as hydramnios are present. Metabolic disturbances such as hypoglycaemia in mainly small and toy breeds may be insidious and fatal if not identified and corrected timeously. Uterine rupture is an uncommon complication of pregnancy and may occur spontaneously during dystocia, or it can inadvertently be induced through the injudicious administration of ecbolics. Other rare complications of pregnancy include a gravid hysterocoele, cervical incompetence, uterine torsion and prolapse. Radiography in the last week of pregnancy is advised as it gives a fair assessment of the number of foetuses that need to be delivered. This is helpful in identifying singleton and two-pup litters in large breeds as well indicating the number of offspring that the owners should be looking out for during natural unassisted labour and parturition observation.

Various methods of gestational aging exist with varying precision, and these may generally give a helpful estimate of the expected date of parturition. The LH surge and the first day of cytological dioestrus are the most valuable predictors of the day of parturition in the bitch. Knowledge of the expected day of delivery is helpful in the management of parturition. Knowledge of normal spontaneous delivery is required to distinguish uncomplicated spontaneous whelp form dystocia.

Parturition

Parturition is believed to be initiated by the foetuses once they have developed adequately to survive extra uterine live. Central to this survival is lung maturity, surfactant production and subsequent ability of the neonate to breath properly soon after birth. Delivery by means of induced parturition or caesarean section should therefore be as close as possible to the onset of spontaneous parturition. The safe period of intervention by CS in the dog is defined as that period before the onset of spontaneous parturition wherein foetuses may be delivered by CS and have survival rates comparable to those of puppies delivered by parturient CS. The safe period of intervention also known as “term” in the bitch is believed to be around 48 hours based on research on preparturient delivery and induction protocols (4).

Dystocia is the inability to pass a foetus through the pelvic canal. The origin can be maternal or foetal. Foetal anatomy and changes in the foetus’ orientation account for most cases of obstructive dystocia,

followed by oversized puppies. The most common maternal cause is uterine inertia characterised by the inability to expel a foetus from the uterus in absence of obstructions. Uterine inertia may be primary or secondary, both ultimately leading to delays in the whelping process and leading to stillbirths, emergency caesarean sections and poor puppy survival. Although dystocia has many causes and can befall any breed, several dog breeds are known to suffer from an increased risk for dystocia. The method of delivery and question of elective caesarean section is a matter that should be discussed between the practitioner and the animal owner. Factors such as breed, obstetric history and value of the offspring are considerations in the risk benefit assessment between the decision to perform a caesarean section or attempt natural birth. Some owners wish to avoid the risk of foetal loss that may result from trial of labour and prefer elective CS. Against the background of CS-rates approaching 100% in some breeds as well as planned elective CSs ending up in emergency CSs and foetal compromise, the concept of preparturient CS has arisen as preferred method of delivery for some workers. Preparturient progesterone concentration drop may aid in the decision to establish readiness for CS in this regard.

There is an increasing sentiment against performing CSs in the absence of medical necessity. Some registering authorities have imposed restrictions of registrations of puppies delivered after a second CS and others have placed bans on certain breeds to mitigate the problem of health concerns associated with extreme features. The veterinary profession and more specifically, veterinary obstetricians have a role to play and delivery by natural whelp should be encouraged in all species.

Singleton pregnancies and two pup litters in large breeds present a unique problem for the canine obstetrician. The exact mechanism by which parturition fails to occur in some singleton pregnancies and not in others, is unknown and managing the parturition of singleton pregnancies remains problematic.

Puerperium

The puerperium is the time after delivery of the offspring when maternal physiologic changes related to pregnancy return to the nonpregnant state. Increase in energy requirement is moderate in late pregnancy (around 10%) but is formidable during lactation (200-250%). Supplementation by folic acid may be beneficial in some breeds to prevent prevalence of split palates. It is important for both the breeder and the veterinary obstetrician to confirm that parturition has been completed and that no retained foetuses are present. Retained foetuses surrounded by gas may evade ultrasound detection and only be detected by radiographs. Placentas not expelled within 12 hours after the birth of the last puppy are deemed to be retained placentas.

Metritis is common after parturition due to ascending bacterial infections and may be a medical emergency requiring intensive therapy and in exceptional cases require ovariohysterectomy. Toxic milk syndrome may also be a sequel. Maintaining good hygiene and routine parasite control is crucial in the puerperium as periparturient stress is known to promote giardiasis, coccidiosis and verminosis.

Excessive haemorrhaging after natural whelp and caesarean section may occur and should be monitored and a haematocrit of below 27% with continued blood loss may be an indication for transfusion. Subinvolution of the placental sites is a less serious cause of haemorrhaging and may persist for up to 8 weeks in particularly younger primiparous bitches.

Colostrum intake is should be achieved within 8 hours in puppies (5). Milk production is best assessed by monitoring neonatal weight gain. Acepromazine and sulpiride may be used to stimulate milk production and help relax bitches. Mastitis is a common complication of lactation and may progress to abscessation, septicaemia and toxaemia. Feline mammary fibroepithelial hyperplasia represents a unique challenge in pregnant queens.

Hypocalcaemia is caused by the inability to timeously mobilise adequate free calcium ion concentrations in the blood and is common in small breed dogs but any dog breed may be affected (6). Hypocalcaemia usually coincides with peak milk production and is characterised by hyperventilation, fever, salivation and tremors. Treatment includes correcting the hypocalcaemia. In rare cases, hypocalcaemia may occur just before labour in the bitch and queen and present more as a paretic syndrome rather than classical tetany.

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061 / #241

ENFERMEDAD MIXOMATOSA VALVULAR MITRAL ¿QUÉ DEBE SABER EL CLÍNICO?

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ABSTRACT BODY:

Enfermedad mixomatosa valvular mitral ¿Qué debe saber el clínico?

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La enfermedad degenerativa mixomatosa valvular mitral es la enfermedad cardiaca más frecuente en los perros. Afecta principalmente pacientes de edad avanzada (arriba de 7 años) y de raza pequeña, pero se puede ver en pacientes más jóvenes en las razas King Charles Spaniel y Dachshund. También algunas razas grandes pueden padecerla, sobre todo los labradores y pastores alemanes.

La enfermedad afecta la matriz del aparato valvular, induciendo alteraciones estructurales tanto de las valvas, como de las cuerdas tendíneas, que induce un déficit de cierre o insuficiencia valvular.

Esta insuficiencia valvular produce una regurgitación de sangre desde el ventrículo izquierdo hacia el atrio izquierdo durante la sístole, y dependiendo del volumen regurgitante, se producirá una sobre carga de volumen para el atrio, que se remodelará adaptándose a esta sobrecarga, incrementando su volumen y luego durante la diástole incrementando el volumen de llenado ventricular izquierdo, induciendo también el crecimiento de la cámara ventricular izquierda. Luego conforme avanza la lesión valvular y la insuficiencia la fracción regurgitante se incrementa, disminuyendo el gasto cardiaco, e induciendo la actividad del sistema Renina/angiotensina/Aldosterona (RAAS) que producirá un incremento de la retención de sodio y cloro a nivel renal, produciendo un incremento de la precarga e incrementando las presiones de llenado dentro de las cámaras cardiacas, además de la vasoconstricción periférica que produce incremento de la resistencia periférica y por lo tanto incremento de la post carga, lo cual empeora la fracción regurgitante, incrementándose cada vez más la presión atrial izquierda, y consecuentemente la presión de venas y vénulas pulmonares que terminará en edema pulmonar cardiogénico, con el respectivo riesgo para la vida del paciente, o en algunos casos hipertensión arterial pulmonar, con el mismo riesgo de muerte.

En el 2019 el colegio americano de medicina interna (ACVIM) publico las guías de manejo y clasificación de la enfermedad degenerativa mixomatosa mitral, (1) estadiándola en 5 clases, recomendando el manejo de acuerdo a la fase de la enfermedad:

Estadío A: Pacientes con riesgo de padecer enfermedad mixomatosa mitral, pero aún sin regurgitación.

Estadío B1: Pacientes con soplo audible debido a regurgitación presente, pero que no presentan crecimiento significativo del atrio y ventrículo izquierdos

Estadío B2: Pacientes con soplo, y con crecimiento significativo de las cámaras izquierdas. Pero asintomáticos, sin insuficiencia cardiaca congestiva.

Estadío C: Pacientes que ya se encuentran en insuficiencia cardiaca congestiva

Estadío D: Pacientes en tratamiento, descompensados

Para evitar esto es importante el reconocimiento de la enfermedad, su correcta evaluación, tratamiento y monitorización, anticipándose al desastre.

El médico general, de la mano del cardiólogo, debe trabajar de forma conjunta para el correcto manejo del paciente, reconociendo la enfermedad y estadiándola, incrementando el tiempo de sobrevida, la calidad de vida y bienestar de la mascota y el propietario.

Los médicos generales deben tener en cuenta varios conceptos y estrategias que les permitan evaluar al paciente.

- **Primero deben reconocer al paciente en estado pre clínico (estadío B1 y B2 de la enfermedad según las guías del ACVIM (1)) El comienzo de la terapia indicada durante el estadío B2 ha demostrado retrasar la aparición del estadìo C en 15 meses(2). Para es necesario familiarizarse con la auscultación del paciente y reconocer el soplo típico holosistólico en meseta, con punto de máxima intensidad entre la 4 y 6 costilla del hemitórax izquierdo. Así no exista sintomatología al paciente se le debe realizar un estudio ecocardiográfico, o al menos radiográfico para identificar el estadío.**
- **Los paciente scon insuficiencia cardiaca (estadío C) debe ser identificados, estabilizados de ser necesario en caso de padecer de edema pulmonar para luego profundizar en más estudios. El uso de técnicas de emergencia como el BLUE® o la radiografía torácica pueden ser de utilidad para reconocer al paciente con edema pulmonar.**

- **Los pacientes en estadío C estables deben pasar por un estudio ecocardiográfico minucioso para identificar gravedad y riesgo de edema. Debe ser controlado periódicamente y educar al propietario en métodos de monitorización caseros, como cálculo de la FR.**
- **Familiarizarse con patrones radiográficos siguiendo pautas por zonas afectadas**
- **Familiarizarse con los informes ecocardiográficos, evaluando ciertos parámetros claves y su relevancia para el paciente:**
- **Diámetro VI diastólico normalizado**
- **Relación AI:Ao en 2D**
- **Velocidad de onda E del flujo transmitral**
- **Relación E/A**
- **Tiempo de relajación isovolúmico (TRIV)**
- **Relación E/TRIV**
- **Identificación probable de hipertensión arterial pulmonar**
- **Función sistólica**
- Conocer la terapéutica adecuada para la patología según el estadío y el manejo nutricional del paciente.

Existen nuevas formas de evaluar la gravedad de un paciente con enfermedad mixomatosa mitral, que, si bien no son catalogadas como guías, proviene de estudios científicos basados en evidencia científica como e MINE score (3)

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062 / #177

ADVANCES IN DIAGNOSIS AND TREATMENT OF ENDEMIC MYCOSES

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ABSTRACT BODY:

Introduction

In recent years, fungal infections have been recognized more frequently in both human and veterinary patients, and the use of antifungal drugs has increased, especially with the use of more potent immunosuppressive drug treatments for longer periods of time. Efforts have focused on the development of new, less toxic and more efficacious antifungal drugs, and antifungal drugs with novel mechanisms of action, and early diagnosis for more effective treatment.

Diagnosis of Fungal Infections

Although different fungal species vary in their organ tropism, fungal infections should be highly suspected in dogs with fever, nodular, cavitary, or lobar pulmonary lesions, osteolytic bone lesions, ocular inflammation such as uveitis or chorioretinitis, diskospondylitis, enlarged lymph nodes, or cutaneous nodules. A lack of response to antibacterial drugs may also suggest underlying fungal disease.

Major progress has been made in diagnostic testing for fungal disease. Cytology, histopathology and culture are useful but sometimes organisms are undetectable with cytology and histopathology, and culture may be dangerous to laboratory personnel. A history of antifungal drug use may render these assays falsely negative. In addition, these techniques may require invasive specimen collection techniques. Previously, with the exception of cryptococcosis, the only alternative tests available were assays that detect antibodies. These assays are largely only useful for diagnosis of coccidioidomycosis and nasal aspergillosis, provided they are performed and interpreted correctly by the laboratory that performs the assays. A positive agar gel immunodiffusion assay titer in a dog suspected to have nasal aspergillosis is strongly suggestive of the disease, but a negative titer does not rule out the possibility of nasal aspergillosis. Commercially-available assays that detect antibodies to *Histoplasma capsulatum* and *Blastomyces dermatitidis* have also not shown to be useful for diagnosis, because of low sensitivity and specificity, but recent research suggests that assays that detect antibodies to the BAD-1 antigen may be more useful.

Over the last 5-10 years, additional assays that detect fungal antigens have been used more widely in veterinary patients. They are useful for diagnosis of histoplasmosis, blastomycosis, and systemic (not sinonasal) aspergillosis. Cross-reactivity between different fungal antigens can occur, especially between *Blastomyces* and *Histoplasma* antigens, and among mold pathogen antigens. Antigen tests for *Blastomyces* are most sensitive when used on urine rather than serum. Serial monitoring of urine *Blastomyces* antigen can also be useful to detect clinical remission and when there is suspicion for relapse, although urine antigen can become negative when clinically detectable disease is present during treatment in some dogs, and clinical relapse can occur before urine antigen tests become positive. *Histoplasma* antigen testing is sensitive in cats when performed on urine specimens. In humans with histoplasmosis, the Infectious Diseases Society of America recommends monitoring urine antigen concentration during treatment and for at least 12 months after discontinuing treatment. Point-of-care (POC) assays for cryptococcal antigen have recently become available that are highly sensitive tests for in-practice diagnosis of cryptococcosis (e.g., IMMY line immunoassay). Positive results using these tests should be followed up with a cryptococcal latex agglutination test. Other POC assays that detect *Coccidioides* antibody or *Aspergillus* antigen are under study at the time of writing, and have potential to improve diagnosis of coccidioidomycosis and disseminated aspergillosis in dogs.

Treatment of Fungal Infections

Cure with antifungal drug treatment is most likely to be achieved in dogs with localized pulmonary infections, or in dogs with mold infections that have occurred secondary to immunosuppressive drug treatment and the immunosuppression can be reversed. Prognosis is poorest for dogs with disseminated mycoses that are predisposed breeds or that lack a clear underlying cause for their disease. The inflammatory response that follows organism lysis with treatment may lead to significant worsening of clinical signs in the first few days of treatment. Use of NSAIDs or, when the brain is involved, a short period of anti-inflammatory doses of glucocorticoids, may be required until the signs begin to resolve.

Some fungal infections respond to treatment with single agent fluconazole or itraconazole. Molds such

as *Aspergillus* have intrinsic resistance to fluconazole, so fluconazole should not be used to treat mold infections. Fluconazole penetrates the brain and the urinary tract, whereas itraconazole may not, unless significant inflammation is present. Many compounded formulations of fluconazole and itraconazole lack efficacy. When treatment failure occurs, serum drug levels can be performed. Itraconazole levels should be performed at least 2 weeks after initiating treatment.

For dogs with disseminated disease, treatment with amphotericin B (AMB) should be considered, although this can be expensive, especially when lipid formulations are used. AMB irreversibly binds sterols in fungal cell membranes, forming pores with subsequent leakage of ions. AMB is virtually unabsorbed from the gastrointestinal tract, so it is formulated for IV infusion (FungizoneÒ, AMB-D) as a complex with the bile salt deoxycholate. Penetration of the CSF and vitreous humor is poor, but we have still had clinical successes using AMB intravenously for treatment of CNS infections.

The major adverse effect of AMB-D is nephrotoxicity. Loading with IV 0.9% NaCl for 1 hour before the infusion decreases nephrotoxicity. Slow administration in a large volume of fluid also decreases nephrotoxicity. Fever, inappetence and vomiting also appear to occur in some dogs treated with AMB-D. Treatment with nonsteroidal anti-inflammatory drugs can be used to decrease pyrexia during therapy.

New Triazoles

Newer triazoles include voriconazole and posaconazole. Voriconazole has been used for treatment of refractory invasive mold infections in dogs. It penetrates the CNS, and can sometimes cause neurologic signs in dogs, as well as hepatopathy. Cats are very predisposed to neurologic adverse effects and cardiac arrhythmias at the doses used in dogs and humans and so its use at those doses is contraindicated in cats. Posaconazole, an itraconazole analog, is a safer option for both dogs and cats - it is an itraconazole analog and has also shown efficacy for treatment of refractory fungal infections in dogs and cats. Absorption of the oral suspension has been shown to be variable in dogs, whereas use of the delayed-release tablet formulation has improved absorption and a longer half-life. A dose of 5 mg/kg q48h is recommended. Posaconazole suspension has also been used successfully to treat life-threatening fungal infections in cats. The pharmacokinetics of posaconazole in cats has been reported. Two treatment regimens using the oral suspension appeared to maintain targeted trough concentrations of 0.5-0.7 µg/mL; either 30 mg/kg PO loading dose followed by 15 mg/kg q48h, or 15 mg/kg PO loading dose followed by 7.5 mg/kg q24h.

Pneumocandins/Echinocandins

The echinocandins inhibit formation of beta(1,3)-D-glucans in the fungal cell wall. The prototype drug is *caspofungin acetate*. Other drugs in this class are micafungin and anidulafungin. Caspofungin is given once daily as a slow IV infusion. Caspofungin is effective against resistant *Candida albicans*, and it also has efficacy against *Aspergillus*. It is ineffective against *Cryptococcus*. Cost is slightly less than for lipid formulations of AMB, but nevertheless the need for daily IV infusion together with the cost means it is rarely used in veterinary medicine at this time. In addition, the optimum dose for dogs and cats and its adverse effects in these species are not known.

Terbinafine

Terbinafine (LamasilÒ) inhibits fungal squalene epoxidase, blocking fungal ergosterol synthesis. In veterinary medicine, it has been most commonly used to treat dermatophytosis, and it is well tolerated. Its efficacy for invasive fungal infections has not been well investigated, although there are a few published and some anecdotal reports of its use to successfully treat some deep mycoses, especially when used in combination with other drugs.

Commonly used drugs for treatment of deep mycoses in dogs and cats

Drug	Dose Range	Frequency	Route
Deoxycholate amphotericin B	Dogs: 0.5 mg/kg in 1 L D5W Cats: 0.25 mg/kg in 250 mL D5W	Mon-Wed-Fri until a total dose of 4-6 mg/kg or azotemia occurs*	IV over 4-6 h
AbelcetÒ (amphotericin B lipid complex)	Dogs: 2-3 mg/kg Cats: 1 mg/kg	Mon-Wed-Fri for 9-12 treatments*	IV; dilute in D5W to 1 mg/kg; give over 1-2 h
Itraconazole	Cats: 3 mg/kg Dogs: 5 mg/kg	q24h (suspension) q12-24h	PO
Fluconazole	Cats: 50 mg/cat/d Dogs: 5-10 mg/kg/d	q24h; divide high doses and give q8-12 h	PO
Terbinafine	Cats: 10-30 mg/kg Dogs: 10 mg/kg	q24h	PO

- Check BUN and creatinine prior to each administration. Discontinue if azotemia occurs.

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REDEFINING CANINE MAMMARY TUMORS AND INFLAMMATORY CARCINOMA MANAGEMENT

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ABSTRACT BODY:

The therapeutic approach to canine mammary tumours is a challenge in daily clinical practice. There is no universally accepted standard therapy according to the histological type of the tumor and/or its respective clinical status, leading to difficulties in choosing the correct approach in each case.

One of the obstacles to choosing the best therapeutic option is the correct interpretation by the veterinarian of the pathologist's histopathology report. Being able to identify which tumor characteristics (clinical and histopathological) are important is vital in order to prescribe the most appropriate treatment.

In the female, the clinical features that indicate malignancy are tumor size, along with rapid and infiltrative growth, with adherence to surrounding tissues. Tumors greater than 5 cm in diameter have a high probability of being malignant. Cutaneous ulceration is another feature of concern. It is important to emphasize that definitive diagnosis is based exclusively on the histopathological result, in almost every case obtained after resection of the tumor. Fine needle aspirates are often inconclusive due to the heterogeneous nature of most mammary tumors in the dog.

Regarding the histopathological characteristics of tumors, special attention should be paid to nuclear grade; mitotic index; histological degree of malignancy, presence of necrosis, all of which can have important prognostic implications, and should be considered in any therapeutic decision (medical and/or surgical).

Notwithstanding the above, the cornerstone for most mammary tumors in the dog is surgery. It not always curative however, and in some cases is not even advised, and so a medical approach centred around systemic chemotherapy is often required

The extent of surgery required depends on the number of tumors and their location in the mammary chain, the clinical characteristics of the tumor and sometimes the surgeon's preference. In the case of multiple tumors that affect several glands of the same chain, radical or modified radical mastectomy should be considered. We are learning that long-term survival of animals after mammary surgery does not depend on the surgical technique used, but on the complete removal of all tumors, with adequate surgical margins for each tumor.

The use of adjuvant chemotherapy after surgery has shown some efficacy, but there are no clinical studies that demonstrate the best drug or the best therapeutic protocol. The most used drugs are Doxorubicin-30 mg/m2, Mitoxantrone-6mg/m2 or Carboplatin 280-300mg/m2). In recent years, the expression of cyclooxygenase-2 (Cox-2) in breast tumors in bitches has been described, mainly in carcinomas of high histological grade of malignancy and in inflammatory carcinomas. The use of cox-2 inhibitors may be indicated in conjunction with conventional chemotherapy. The scientific evidence at our disposal supports its use in conjunction with conventional chemotherapy in cases of malignant tumors with ganglionic invasion (regional clinical stage) and in non-operable tumors (inflammatory carcinoma, largely ulcerated tumors).

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064 / #66

AGRESIVIDAD CANINA: QUÉ PUEDO HACER COMO VETERINARIO GENERALISTA?

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ABSTRACT BODY:

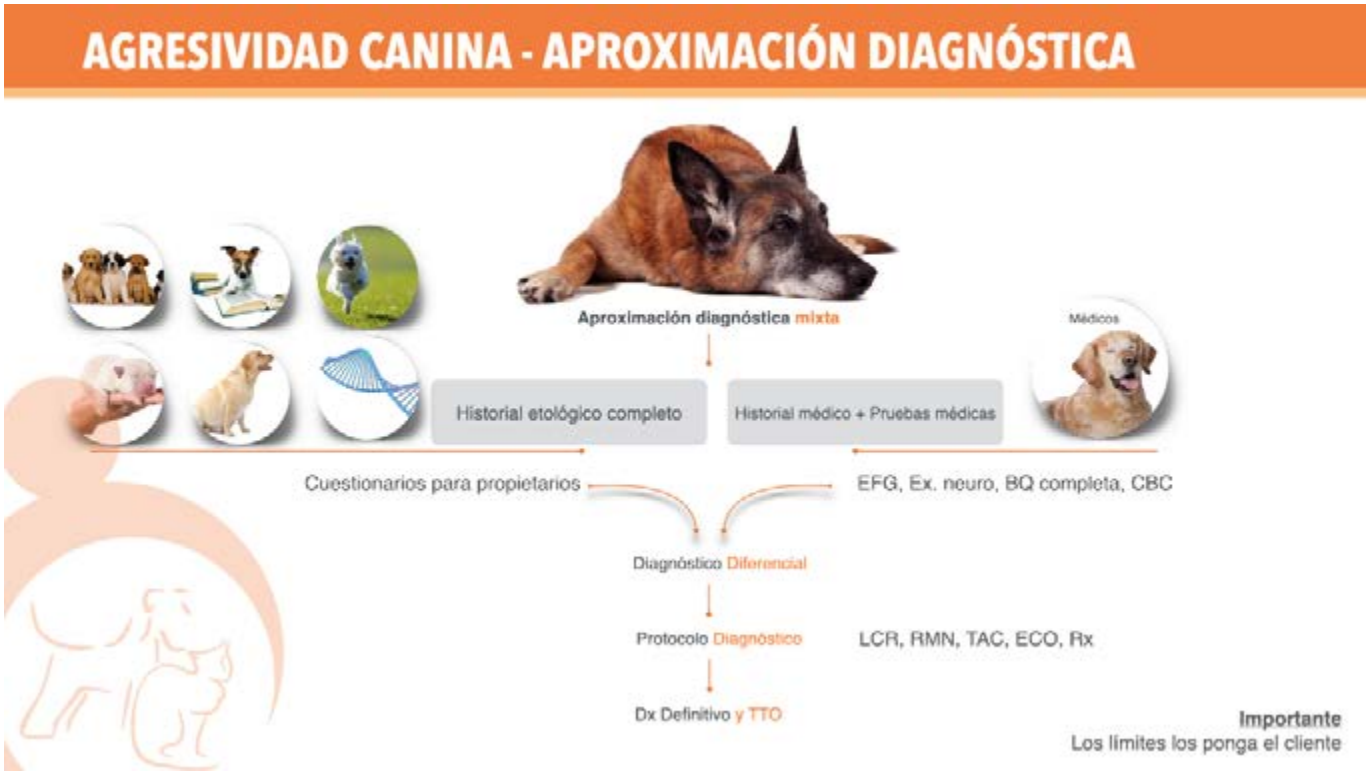
Los problemas de comportamiento se definen como aquellas conductas que suponen una molestia para los tutores. La agresividad es uno de los principales problemas de comportamiento en los perros (y en los gatos). A parte de ser el problema de comportamiento más común, representa un riesgo para la salud pública de las personas o para otros perros o animales. Finalmente, los perros con problemas de conducta, y especialmente los agresivos, tienen más probabilidades de ser eutanasiados o abandonados.

Esta charla tiene como objetivo establecer las bases para entender la agresividad canina y desmentir algunos conceptos clásicos que son perjudiciales y peligrosos para el animal y las personas. En segundo lugar, enumerar y justificar las principales recomendaciones que deberían darse ante los problemas de agresividad canina como centro de veterinaria generalista.

EL PERRO AGRESIVO: UN RETO DIAGNÓSTICO

La agresividad no es un diagnóstico. Esta afirmación, que puede parecer tajante, es uno de los mensajes más importantes de la charla. La agresividad es un signo clínico. Como tal, no se debería establecer un tratamiento sin conocer la causa de dicha agresividad. Es crucial que ante cualquier caso de agresividad y, de hecho, ante cualquier problema de comportamiento, se realice un protocolo de diagnóstico que incluya tanto aspectos del comportamiento del animal como de su salud.

El protocolo diagnóstico (Figura 1) siempre incluirá un historial etológico completo, un historial médico y, finalmente, un examen del estado de salud actual del perro.



El historial etológico debe recoger toda la información sobre los principales aspectos que afectan a la conducta del animal. Se realizarán preguntas acerca de las condiciones de cría durante el desarrollo del cachorro, del entorno del animal, del manejo general y particular de las situaciones de conflicto, entre muchas otras cosas.

Las pruebas médicas para conocer el estado actual de salud del animal deberán incluir, como mínimo, un examen físico general, un examen neurológico, una bioquímica completa que, en el caso de la agresividad, siempre debe incluir una valoración del perfil tiroideo y de la funcionalidad hepática, y un hemograma.

Con toda la información se establecerá diagnóstico

diferencial que determinará el protocolo diagnóstico más adecuado. Solo bajo un diagnóstico definitivo o presuntivo justificado, se deberían establecer el tratamiento.

¿PUEDO DAR ALGÚN CONSEJO ANTES DE TENER EL DIAGNÓSTICO DEFINITIVO?

En la mayoría de las ocasiones, tanto en centros generalistas como de especialidad, cuando se realiza el primer contacto entre la familia del perro y el veterinario (o ATV) debemos dar una serie de consejos que tienen por objetivo:

- Evitar el empeoramiento del problema.
- Garantizar el bienestar y la seguridad de las personas.
- Maximizar el bienestar del perro o de los perros.

En ningún caso deben darse consejos con el objetivo de tratar el problema, ya que todavía no se ha realizado ningún diagnóstico. En la Tabla 1 se resumen los consejos que deben darse y las medidas que nunca deberían aconsejarse a las familias de un perro agresivo antes de tener un diagnóstico definitivo.

Tabla 1. Recomendaciones aconsejables y desaconsejables para perros agresivos antes de tener un diagnóstico definitivo.

RECOMENDACIONES ACONSEJADAS	RECOMENDACIONES DESACONSEJADAS
Agresividad hacia personas	No recomendar imponerse al animal
Evitar las situaciones de conflicto	No recomendar collares que inducen dolor o miedo (collares de castigo, cualquier que sea) para controlar al perro en la calle.
No castigar bajo ningún concepto	Uso de feromonas
Intentar potenciar las emociones positivas	Uso de terapias alternativas
Agresividad entre perros que conviven	Uso de psicofármacos con objetivo terapéutico a largo plazo
Si es posible, no separar a los perros	Castración
Si se tiene que separar a los perros, mantener actividades placenteras juntos (paseos con distancia de seguridad, juego, etc…)	
No castigar bajo ningún concepto	
Si hay una pelea, no separarlos físicamente con nuestras manos	
Agresividad hacia otros perros desconocidos	
No forzar el contacto en la calle con perros desconocidos	
Si hay perros con los que se lleva bien, mantener el contacto y las actividades placenteras	
No castigar bajo ningún concepto	
Si hay una pelea, no separarlos físicamente con nuestras manos	

La castración no debería recomendarse en ningún caso sin tener un diagnóstico definitivo del problema ya que, en la mayoría de los casos, podría empeorar el problema de agresividad. Desgraciadamente es una recomendación que todavía se sigue utilizando como primera herramienta para intentar controlar cualquier problema de agresividad, sin embargo, los estudios más recientes apuntarían a que, tanto en machos como en hembras, la agresividad (el miedo y la ansiedad) se verían potenciados por la castración. Además, este efecto parece ser una relación de causa-efecto, ya que cuánto más pronto se castre al perro o perra, la probabilidad de que aparezcan problemas de miedo, ansiedad o agresividad será mayor.

POR QUÉ MUERDEN LOS PERROS

Una vez que se hayan instaurado las primeras medidas de seguridad que se mencionan en la Tabla 1, se debe realizar un correcto diagnóstico. El objetivo de esta charla no es el de enumerar todos los diagnósticos clínicos de agresividad del perro, pero sí que se analizarán cuáles son las principales motivaciones de la agresividad canina.

- Miedo. La agresividad es una estrategia de conducta habitual para controlar las situaciones que provocan miedo. Dicho en otras palabras, cuando el perro tiene que enfrentarse a un estímulo que le produce miedo, o bien intentar evitarlo (huyendo, por ejemplo), o enfrentarse a él para conseguir que se aleje (mostrándose agresivo), entre otras

estrategias.

- Frustración. Ante una situación que genere frustración (no se cumplen las expectativas del animal) el animal puede responder, básicamente, de cuatro formas distintas: (1) aceptar la situación (resignarse), (2) Intentar encontrar otra estrategia para conseguir su objetivo, (3) realizar conductas de desplazamiento para aliviar la situación de estrés derivada (por ejemplo, perseguirse la cola en perros) o, finalmente, (4) puede mostrarse agresivo. Que la agresividad sea una salida de conducta común en los perros (y otros animales) no significa que sea aceptable. Dicho en otras palabras, aunque sea un comportamiento que tiene una justificación, se deberá enseñar al perro a gestionar de otra forma las situaciones que le generen frustración.
- Defender un recurso valioso para el animal.
- Por enfermedad.
- Porque defienda su territorio (su casa, por ejemplo) de la entrada de animales o personas que no sea de su "grupo familiar".
- En el caso de los machos, porque compita con otros perros del mismo sexo (agresividad intrasexual).
- En el caso particular de las perras, porque quieran defender a sus crías (agresividad maternal).

Para determinar cuál es la motivación subyacente del problema de agresividad que muestra un paciente en concreto, se deberá realizar la aproximación diagnóstica que se ha mencionado anteriormente. Es cierto que para realizar la entrevista etológica se requiere mucho tiempo, y muchos centros generalistas prefieren derivar esta parte a un centro veterinario o veterinario especializado de referencia de su confianza. Sin embargo, técnicamente todas las partes que incluye la aproximación diagnóstica planteada pueden realizarse en un centro veterinario generalista. Sin embargo, la aplicación de algunas de las medidas terapéuticas es difícil en los centros convencionales, ya que se requieren de amplias zonas destinadas especialmente a este fin (durante la charla se expondrá algún caso para ilustrarlo).

No sucede lo mismo, sin embargo, con las medidas que se pueden aconsejar para la prevención de los problemas de agresividad en el perro.

PAPEL DEL VETERINARIO EN LA PREVENCIÓN DE LA AGRESIVIDAD CANINA

Es mucho más efectivo prevenir que tratar los problemas de agresividad canina. Los centros veterinarios generalistas juegan un papel crucial en la prevención de estos problemas. Las principales medidas que pueden adoptarse son:

- Asegurar una correcta socialización. Durante la primovacunación serán fundamentales los consejos que las familias reciban de su veterinario de cabecera. Incluso en los centros pueden realizarse sesiones de socialización para cachorros con la finalidad de socializar al perro y educador a los tutores sobre las medidas adecuadas de educación del cachorro en casa.
- Valorar individualmente la necesidad de castración temprana y valorar otras posibilidades de esterilización como, por ejemplo, la vasectomía.
- Asegurar un correcto estado de salud del animal con chequeos rutinarios de los perros.
- Recomendar un manejo amable del perro en el día a día de su convivencia con los tutores.

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065 / #67

POR QUÉ A LOS VETERINARIOS NO LES GUSTA LA GESTIÓN?

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ABSTRACT BODY:

Veterinarios tienen cierta molestia a manejar el tema Gestión por cuenta que nunca ou casi nunca lo estudiaron en la faculdad. A los veterinaraio le gustan los numeros de la parte clínica, diagnostico, tratamiento. La vocación está en las mascotas, no en los número. Sin embargo, hay que se saber los KPIs, Key Performance Indicator, los indocadores básicos de la gestión, pues además der sermos veterinarios, tenemos que axctuar también en la administración de nuestros negocios. Con la experiencia que tenemos em 38 años en el mundo vet, enseñando emprendedorismo y administración, percebi que muchos vets psan a se encantar con la gestión cuando la aprenden de hecho, pus saberán utilizar la BI, business inteligence, o sea, usar los datos como informacion que traiga estrategia, acciones práticas que nos lleven a crescer como empresa, sabiendo manejar los números con el equipe. La participación de todos es fundamental al crescimiento del negocio. SAber cuando es tiempo de tener gerentes administrativos y otro clínico, cuando contractar, etc.

AN EVIDENCE BASED APPROACH TO THE DYSPNEIC CAT

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ABSTRACT BODY:

Evidence-based veterinary medical practice aims to integrate the best clinical expertise and judgment with the best available scientific evidence. Professional ethics claim that veterinary practitioners and clinicians have to choose optimal diagnostics, effective and up-to-date treatments and to provide comprehensive advice to animal owners. The aim of evidence-based veterinary medicine (EBVM) is to base clinical decisions on recent, valid, clinically relevant research data.

Evaluation and first approach to the dyspneic cat is often considered one of the most difficult tasks of the feline practitioner. Prompt recognition and localization of the source of respiratory distress are fundamental to determining the appropriate diagnostic and therapeutic maneuvers. As they may decompensate rapidly, it is essential a feline friendly environment and interactions.

In this session, we will work through the how apply EBVM principles, with focus on scientific literature involving information that can be applied to how approach dyspneic cats. Oxygen supplementation, tranquilization and a gentle/minimal restrain are fundamental in stabilization process, and keep as pillars of first approach, even not discussed in literature cited here.

Most common causes of dyspnea

The most common causes of dyspnea in cats fall into one of four main categories: cardiac, respiratory, neoplasia or traumatic/other. In two different retrospective studies evaluating etiology of respiratory distress in cats, cardiovascular and respiratory causes comprehend more than 70% of the cases (Swift et al., 2009; Dickson et al., 2018).

Evaluating respiratory patterns and physical examination findings

During stabilization and oxygen supplementation, obtain a concise history of owner and evaluate respiratory pattern can help to distinguish the dyspnea origin. In conjunction with some physical examination findings (more specifically, auscultation), those points are important tolls to better approach cats with respiratory distress.

Cats with inspiratory effort, specially associated with respiratory noises, are more associated with extra-thoracic airway disease. Common causes include nasopharyngeal polyps, upper respiratory infection and foreign bodies. In patients with stridor, laryngeal disease (laryngitis, neoplasia, paralysis) is more commonly found. Cats with nasopharyngeal disease tend to have decreased nasal air flow associated with stertors (Sigrist et al., 2011; Domínguez-Ruiz et al., 2021).

Expiratory effort (usually associated with wheeze in a cat with history of cough) is related to lower airway diseases, with asthma and chronic bronchitis as predominant diseases (Sigrist et al., 2011; 2012; Domínguez-Ruiz et al., 2021).

Paradoxical breathing (or asynchronous/inverse breathing) is moderately to strongly associated with pleural space disease, usually with pulmonary/cardiac sounds attenuation (Sigrist et al., 2011; Le Boedec et al., 2012; Domínguez-Ruiz et al., 2021).

Pleural effusion as cause of dyspnea

Most common cause of respiratory distress secondary to pleural space disease is pleural effusion. There are some interesting retrospective studies about most common causes of pleural effusion in cats, with more than thousand cats with diagnosed cause of chest fluid. More than 90% of pleural effusion cases in cats fall into one of four categories: cardiogenic (most common cause), neoplasia, infectious (pyothorax, FIP) and idiopathic chylous effusion. History, fluid complete analysis/cytology, as so imaging (radiography/echo – only in stable patients!) are recommended for definitive diagnosis (Ruiz et al., 2018; Konig et al., 2019; Hung et al., 2022).

Dyspneic cat – heart or lungs?

Differentiating congestive heart failure (CHF) from other causes of respiratory disease (pleural space or lungs) can be difficult in the unstable cat. Some data in history and physical examination findings may help increase the index of suspicion for congestive heart failure (CHF). Usually, cats presenting with CHF are more likely to be hypothermic compared to non-cardiac causes of dyspnea (Dickson et al. 2018; König et al. 2019). An algorithm (RAPID CAT) comprised of the presence of a gallop rhythm, heart rate >200 bpm, respiratory rate >80 bpm, temperature <37.5 °C is a potentially useful tool for differentiating cardiac from non-cardiac causes of dyspnea (Dickson et al. 2018).

Point-of-care-ultrasound (POCUS) and measurement of cardiac biomarkers may help support a diagnosis

of CHF. Measurement and evaluation of an enlarged left atrium to aorta ratio on POCUS is highly accurate predictor of CHF as the cause of dyspnea (Ward et al. 2018; Janson et al. 2020). Additional POCUS findings supportive of a diagnosis of CHF include the presence of a pericardial effusion and the presence of three or more B-lines (lung rockets) per site on lung ultrasound. Combining lung ultrasound with measurement of the cardiac biomarker NT-ProBNP increases the accuracy of the diagnosis of CHF (Ward et al. 2018). Since restraining and blood collection can be fatal in some unstable cats, measure NT-ProBNP using blood of IV catheter or in pleural fluid is recommended.

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HEALTHY START: NUTRITION BASICS FOR PUPPIES AND KITTENS

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INTRODUCTION

Adult size and growth potential is primarily regulated by genetics while diet is within the owner's direct control each day and can have a more immediate influence on the health of puppies and kittens. Pet owners are inundated with pet food marketing messages that may or may not be rooted in nutritional science. Growth is a nutritionally challenging time for young dogs and cats and rates of development can be impacted by under or over-feeding, and essential nutrient deficiencies or excesses can cause life-long and potentially fatal health complications. Owners will turn to veterinarians for guidance when making decisions about puppy and kitten diets and it is important for veterinary professionals (both veterinarians and support staff) to understand not only nutrition concepts but the benefits and limitations of various diet options to avoid improper feeding practices that can harm these young patients.

THE BASICS

Essential Nutrients

Dogs and cats have similar requirements for specific essential amino acids, fatty acids, macrominerals, trace minerals, water-soluble vitamins, and fat-soluble vitamins, with a few notable variations such as differences in amounts required between the species and key nutrients such as taurine, vitamin A, and arachidonic acid (AA) that are dietary requirements for cats but not dogs.[1] Irrespective of individual species' requirements these essential nutrients are largely required at higher levels for growth than during adult maintenance. The selected growth diet should also contain higher levels of the long-chain omega-3 fatty acid docosahexaenoic acid (DHA); this conditionally-essential nutrient is found in high concentrations in neural tissue and is required for optimal visual acuity and brain development in puppies and kittens.[2] Home-prepared diets rarely meet the essential nutrient needs of growing animals[3-7] and the feeding of a species- and life-stage-specific commercial pet food should be used preferentially for at least the first year of life before transitioning to a home-prepared diet developed by a Veterinary Nutrition Specialist.

Skeletal Growth

Normal skeletal growth can be broken into three distinct phases: endochondral ossification (growth, maturation, and apoptosis of chondrocytes with replacement of cartilage with bone); periosteal growth (new bone formation); and remodeling (adaptation of the newly formed bone).[8] These steps are controlled by the interrelationship between parathyroid hormone (PTH), which stimulate osteoclastic activity to break down bone, and calcitonin, which promotes osteoblastic activity to form new bone. These hormones are regulated by dietary intake of calcium and vitamin D and whole body levels of these essential nutrients as well as total energy intake. Immature animals have limited control of calcium absorption from the intestinal tract, with 45-50% of ingested calcium absorbed regardless of the quantity of calcium consumed.[9]

Excess consumption of calcium, vitamin D, and total energy during the first 12 months of life (rapid growth period) can adversely affect skeletal and joint development and lead to osteochondrosis (OCD) lesions and angular limb deformities with large and giant breed dogs. Over-nutrition, whether through excess energy intake or excess calcium and vitamin D supplementation, can also increase the risk of elbow and hip dysplasia in at-risk dog breeds.[10,11] Inadequate calcium or vitamin D intake, or both, can lead to nutritional secondary hyperparathyroidism and the development of pathological fractures in any puppy or kitten.

For cats and small to medium breed dogs, the period of maximal skeletal growth is relatively short (2-3 months) and an individual will typically reach 80% of their adult size by 6 months of age; large and giant breed dogs in contrast will continue to have maximal growth rates through 4-5 months of age and will reach 80% of their adult size by 8-10 months of age.[8] Given this prolonged growth period in larger breed dogs, the timing of nutrient excess can also influence orthopedic disease seen.

Energy

Feeding guidelines are required on the package of all over-the-counter pet foods and are based on energy calculations from the National Research Council (NRC).[1] The specific mathematical formula used will vary somewhat by pet food manufacturers but often begins with a calculated resting energy requirement (RER) of $70*Bw_{kg}^{0.75}$ with a multiplier added for growth.

For puppies, during the first 2-4 months of life daily energy requirements can be 2-3*RER and will slow to 1.8-2*RER once the individual is close to their adult size. This RER calculation may overestimate energy requirements for cats and kittens and the NRC recommended the use of an alternative calculation for cats and kittens ($100*Bw_{kg}^{0.67}$) with the caveat that kittens, at least for the first 6 months of life, should be allowed *ad libitum* access to food.

Adult dogs and cats can vary up to 50% above or below these calculated RER values but the individual variability in puppies and kittens is not known. Neonatal feeding can influence future energy requirements one study showed that kittens allowed to nurse for 7 weeks are less likely to become obese as adults.[12] In growing animals, obesity (being more than 20% over ideal weight) can result in an overall increase in the number of adipocytes rather than enlargement of existing adipocytes, which in turn may increase the risk of obesity in adulthood. Obesity in adult dogs and cats can contribute to the development of peripheral insulin resistance, increases the risk of developing orthopedic disease (or causes progression of pre-existing orthopedic disease), and has been shown to decrease longevity.[13]

Food as Environmental Enrichment

For dogs and cats that spend much of their lives indoors with humans, early life experiences can influence future feeding preferences. Studies have also shown that early pre- and post-natal flavor exposure through the maternal diet can influence subsequent food preferences by kittens and cats.[14] Puppies can be less predictable with some showing a clear preference for flavor exposure during the pre-natal period while other may seek novel flavors and textures.[15] Owner-feeding behaviors such as treating and use of food-dispensing enrichment toys that mimic food-seeking behaviors in the wild such as hunting and scavenging can also reduce the risk of obesity and avoid more fixed flavor and texture preferences that can occur when diet type is help static from a young age,[16,17] but care should be taken to avoid conditioning what may be seen as "finicky" food preference by rotating diets too frequently and giving excessive treats outside of meal times.

SUMMARY

The time between weaning and reaching adult size is a period of active and rapid growth for puppies and kittens. Irrespective of breed or species, growing animals should be fed a diet that has been developed to meet either the Association of American Feed Control Officials (AAFCO) or European Pet Food Industry Federation (FEDIAF) recommended nutrient intakes for either “all life-stages” or “growth”. Ideally, this diet will have been evaluated in a controlled setting using feeding trials (fed to groups of healthy puppies to prove suitability for growth), but diets produced by commercial companies that work with trained Food Scientists and Animal Nutrition experts (PhDs or Veterinary Nutrition Diplomates) can also meet these needs. Feeding unbalanced diets to growing animals can result in potentially fatal developmental diseases and should be avoided.

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068 / #63

CLINICAL APPROACH TO INFERTILITY IN MALE DOGS AND CATS

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ABSTRACT BODY:

Infertility in male dogs – Poor semen quality is the most common cause of low male fertility. The most important alterations of semen quality are *azoospermia* (absence of spermatozoa in the ejaculate), *oligozoospermia* (low number of spermatozoa in the ejaculate), *asthenozoospermia* (presence of a high percentage of spermatozoa which do not show a normal progressive motility) or *teratozoospermia* (presence of a high percentage of spermatozoa with morphological defects). Poor semen quality can be due to poor quality of spermatozoa, abnormal composition of seminal plasma, congenital defects (testicular hypoplasia, the immotile cilia syndrome or chromosomal abnormalities such as XXY syndrome or XX males), mono- or bilateral cryptorchidism, anomalies of the duct system (cysts or other developmental anomalies of the epididymis, the vas deferens or the rete testis).

Azoospermia means ejaculation of seminal fluid devoid of spermatozoa. Dogs may fail to ejaculate the second, sperm-rich fraction if they feel uneasy or apprehensive at the time of semen collection and/or if there is no bitch in heat present. Semen collection should be repeated (in a trained dog) at least 3-4 times over several days to confirm a diagnosis of azoospermia. Whenever an azoospermic sample is collected, alkaline phosphatase (AP) should be measured on seminal plasma of the second, sperm-rich fraction. Normal dogs with semen coming from the testicles have AP values > 5.000, while when an incomplete sample is collected AP is < 5000 U/L but often even < 2000 U/L. An azoospermic semen sample with high AP comes from the epidydimis (and therefore likely from the testicles), while an azoospermic sample with low AP may come only from the prostate (indicating incomplete ejaculation) or its source is however posterior to the epididymis (indicating bilateral epidydimal duct outflow blockage). Azoospermic dogs may have sired one or more litters previously. Heritability is suspected. Azoospermia may be due to pre-testicular, testicular or post-testicular factors. Pre-testicular factors include endocrine conditions such as hypopituitarism, hypothyroidism, steroid excess (Cushing syndrome or exogenous steroid administration), treatment with antineoplastic drugs, inguinal or scrotal hernia. Testicular causes of azoospermia include intersex, germinal cell aplasia, bilateral cryptorchidism, testicular injury due to trauma, irradiation, thermal insult, chronic orchitis, autoimmune testicular disorders (such as spermatogenic arrest) and testicular cancer (4). Germinal cell aplasia occurs in about 10% of azoospermic dog and is characterized by presence of only Sertoli cells. Orchitis and/or epididymitis may determine fibrotic changes of the duct system causing stenosis which results in oligozoospermia and frequently evolves in azoospermia despite antibiotic treatment. Post-testicular diseases responsible for azoospermia are those which cause outflow obstruction such as spermatocele, sperm granuloma or segmental aplasia of the epididymis. Incidence of post-testicular causes of azoospermia in humans is ≤1% while it is unknown in dogs.

Ejaculation failure - The canine vas deferens, epididymis, prostate and bladder neck are primarily under sympathetic nervous system as demonstrated by the fact that following administration of sympathomimetic drugs the canine epididymal and prostatic tissue display phases of rhythmic contractions. The bladder neck plays an important role during ejaculation preventing a retrograde flow of semen into the urinary bladder. During the canine ejaculatory process, the bladder neck shows periods of intense contractile activity followed by phases of relaxation: the duration of each relaxation phase decreases gradually until a continuous contractile momentum of the dorsal segment of the bladder neck occurs towards the end of the ejaculatory process. When the dorsal segment of the bladder neck is relaxed (during sexual rest and in between urethral muscle peristaltic contractions early in the ejaculatory process) semen may flow back into the urinary bladder following the least resistant pathway. If the bladder neck does not contract properly at ejaculation, then the posterior urethra becomes the least resistant pathway through which semen will end up in the bladder; if this happens during a natural breeding semen will not reach the uterus and the bitch will not conceive. Treatment of retrograde ejaculation due to anatomic causes is generally unsuccessful while neuropathic or psychological causes can be treated with sympathomimetic drugs: treatment protocols reported in the dog include phenylpropanolamine (3 mg/kg per os twice daily) and pseudoephedrine hydrochloride

(3-5 mg/kg per os 3 times daily or 3 and 1 hour prior to breeding/semen collection). Retrograde ejaculation has been reported to cause oligozoospermia and aspermia in the dog (5).

Infertility in tom cats – Low libido - Abnormalities in mating constitute a common presenting complaint from cat breeders. Infertile tom cats are frequently referred to the veterinarian for lack of willingness to mate or inability to achieve a normal mating. Low libido may be due to hormonal (testicular hypoplasia/atrophy, use of contraceptive drugs, age, disorders of sexual development (DSD), inheritance), physical (orthopedic/ muscular problems, penile hair ring, phimosis, orchitis/ epidydimitis, general disease) or behavioural (individual preference, previous negative experience, social or environmental stress) causes. Hormonal causes of low libido are rare in tom cats; testicular atrophy is poorly described, age and use of contraceptive drugs can be easily ruled out while DSD are probably more common than once thought due to increased use of line breeding. Physical problems are the most common reasons for reduced libido in tom cats and can be congenital (hypospadias, persistent frenulum, abnormal prepuce or double penis) or acquired due to external factors (hair rings, accumulation of smegma) of pathological conditions of the reproductive system (trauma, inflammation, neoplasia) or of other systems (muscoleskeletal, oral cavity, obesity). Stress due to hierarchy, environment or transport should always be carefully ruled out. Poor semen quality - If breeding occurs normally then failure to impregnate a queen in heat may be due to poor semen quality. Similarly to dogs, poor quality of feline semen may be due to DSDs (monosomy, trisomy, chimerism, mosaicism have all been reported in cats) as well as to other (well known in dogs but uncommon in cats) conditions such as cryptorchidism, trauma, bacterial or autoimmune orchitis, or testicular degeneration. The impact on fertility of the (very rare in tom cats) pathological conditions of the two accessory glands of the feline reproductive system, prostate and bulbo urethral glands, is unknown. Failure to ejaculate – Retrograde ejaculation has been occasionally observed in tom cats during reproductive studies but its incidence as well as its role in feline infertility are unknown. As in other species, spermatozoa are normally found in the bladder in low numbers at any time following puberty. Sperm number may increase following mating or administration of alpha-2 agonists. The finding of high numbers of sperm in the bladder (several hundred thousand or millions) is indicative of retrograde ejaculation

CLINICAL APPROACH TO THE INFERTILE MALE

There are very few male fertility problems which can be solved using specific treatments. Chronic bacterial orchitis, epididymitis or prostatitis should be treated with long-term (4-12 weeks) specific antibiotics. In dogs semen samples should be evaluated for presence of bacteria: a number of colony forming units exceeding 10.000/ml indicates infection and the dog should be treated. Drugs with low protein binding and high lipid solubility should be selected such as chloramphenicol or fluoroquinolones. Determination of prostatic fluid pH (usually neutral or slightly acidic) prior to treatment can be of help since weak base antibiotics (i.e. erythromycin or trimethoprim-sulpha) will achieve a higher prostatic concentration. There are no data on bacterial count in feline semen and the dog can be used as a reference. Once confirmed with AP assay, azoospermia should be investigated with fine needle aspiration to assess whether normal spermatozoa are still present in the seminiferous tubules, in which case a corticosteroid treatment may be attempted.

Natural PGF2alpha (PGF) is reported to improve semen quality in the dog giving >200% increase in sperm numbers when administered at 100 mcg/kg SC once 15 minutes prior to semen collection. PGF increases epididymal contractility. The flare-up phase following administration of deslorelin has been reported to be characterized by an increase in semen output and improvement in semen quality both in dogs and cats, therefore deslorelin could be used for this purpose provided that the implant is removed soon (7-10 days) after administration. If present, improvement will always be slow to appear because of the 60-day spermatogenic cycle in both species. When semen quality is below normal, ovulation must be timed very accurately and artificial insemination performed intravaginally on day 4, 5 and 6 post-LH peak (day 2, 3 and 4 post-ovulation) if total count is ≥100.000.000 spermatozoa. If this approach fails or if total count is <20.000.000 spermatozoa, artificial insemination should be performed intrauterine on day 5 post-LH peak. Serum progesterone should be assayed 3-20 days post-mating in a mated queen to rule out failure to ovulate. Semen collection may allow to rule out failure to ejaculate and can be done in cats by recovering semen from the bladder (after a mating), performing a vaginal smear or wash with 1.0 cc saline on a recently mated queen, or using high doses (100-140 mcg/kg) medetomidine (10). Alkaline phosphatase (AP) should be assayed in azoospermic semen sample to help identifying the site of origin of the collected sample. AP values are similar in dogs and cats.

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CARDIOMIOPATÍAS FELINAS, UNA BOMBA DE TIEMPO QUE SE DEBE SABER DESACTIVAR

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ABSTRACT BODY:

Cardiomiopatías felinas, una bomba de tiempo que se debe saber desactivar

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Las cardiomiopatías felinas son a patología cardiaca más común en nuestros pacientes felinos, y dentro de éstas, la cardiomiopatía hipertrófica es la mas común. Debido a que realmente no podemos afirmar hasta no realizar un examen genético del paciente que se trata de un cardiomiopatía hipertrófica, llamaremos a un paciente con características ecocardiográficas como fenotipo de cardiomiopatía, debido a que algunas patologías pueden semejar remodelación cardiaca similar, como el hipertiroidismo, la hipertensión arterial sistémica, la acromegalia, miocarditis, infiltración tumoral miocárdica, que deben ser descartadas en lo posible para poder acercarnos más al diagnóstico de cardiomiopatía hipertrófica. Además, las diversas cardiomiopatías a veces son difíciles de diferenciar entre una y otra, por ejemplo una cardiomiopatía hipertrófica remodelada puede confundirse con un fenotipo dilatado, o restrictivo, así que a veces la clasificación no es tan simple dependiendo de la presentación fenotípica.

El mayor proble es que estos pacientes son completamente asintomáticos hasta que presentan insuficiencia cardiaca congestiva y llegan a consulta con disnea marcada debido a efusión pleural, edema pulmonar o ambos. Además de otras complicaciones como el tromboembolismo arterial. Debido a eso es necesario que el veterinario realice un examen físico cuidados y se auxilie con exámenes auxiliares que puedan establecer la presencia o no de un fenotipo cardiomiopático y probables complicaciones.

La auscultación cardiaca es primordial, muchos gatos cardiomiopáticos presentan soplos debido a obstrucción dinámica del tracto de salida VI y/o movimiento anterior sistólico mitral. Todo gato con soplo debería ser descartado para cardiomiopatías, teniendo en cuenta que muchos gatos pueden tener soplos fisiológicos considerados normales por obstrucción dinámica del tracto de salida VD en frecuencias cardiacas elevadas. (1)

Existen razas con una alta predisposición a cardiomiopatía hipertrófica y estas deben ser monitorizadas desde jóvenes: Maine con, Sphinx, Ragdol, Bosque de Noruega, Devon Rex y otros.

La presencia de soplos, que deben ser auscultados generalmente en la zona esternal izquierda, hace sospechoso al gato de padecer cardiomiopatía oculta. Por lo que debe ser examinado ecocardiográficamente.

Otras técnicas como la electrocardiografía también pueden ser de auxilio, la presencia de complejos QRS mayores de 0.6 mV ya hace sospechoso al gato, así como patrones de bloqueo del fascículo anterior izquierdo, debería ser también examinados por ecocardiografía.

La radiografía también puede ser un auxilio para la sospecha diagnóstica, u corazón de más de 8.0 vértebras mediante el cálculo de VHS, o con forma de "jalapeño" debe hacer también sospechar al clínico de la posible presencia de cardiomiopatía en el paciente, por lo que debería ser también examinado mediante ecocardiografía.

La ausencia de signos clínicos como por ejemplo la tos en los perros, hace más compleja a situación.

Por eso los lineamientos de las guías del ACVIM (2), recomiendan que todo gato mayor debe pasar por un estudio ecocardiográfico antes de ser sometido a anestesia, cirugía, fluidoterapia o tratamientos con corticoides, ya que estos procedimientos podrían desestabilizar a un paciente compensado y llevarlo a una falla congestiva rápidamente.

Las guías del Colegio Americano de Medicina Interna (ACVIM) (2) para el diagnóstico y tratamiento de las cardiomiopatías felinas clasifican a estas en estadíos similares a los descritos en la enfermedad mixmatosa mitral, pero enfocados hacia el riesgo del paciente según el estadío:

Estadío A: Pacientes con predisposición

Estadío B1: Paciente subclínico sin o con leve crecimiento atrial izquierdo, de bajo riesgo para enfermedad congestiva o tromboembolismo

Estadío B2: Paciente subclínico con moderado o marcado crecimiento atrial izquierdo, de alto riesgo para enfermedad congestiva o tromboembolismo

Estadío C: Paciente con enfermedad cardiaca congestiva o que halla pasado por un episodio congestivo

Esatdío D: Paciente con enfermedad cardiaca congestiva refractaria

Los tratamientos recomendados varían de acuerdo con el estadío. Los pacientes A y B1 no recibe tratamiento, salvo los B1 que padecen de obstrucción dinámica del tracto de salida VI que debe recibir betabloqueantes. debe ser monitorizados. Los pacientes en estadío B2 debe ser monitorizados y aparte si hay riesgo de tromboembolismo deben ser medicados con clopidogrel. Todo esto tratando de anteponerse a eventos clásicos de la enfermedad congestiva, evitando o tomando medidas necesarias en caso de que los gatos necesiten ser sometidos a condiciones que puedan desestabilizarlos, como anestesia, fluidoterapia o tratamientos largos e intensos con corticoides.

Concluyendo, la identificación de los pacientes con fenotipo de cardiomiopatía hipertrófica u otras cardiomiopatías debe ser realizada durante al etapa presintomática, todo paciente felino mayor debe pasar por exámenes ecocardiográficos para descartar la presencia de estos fenotipos y evitar complicaciones posteriores.

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070 / #71

THE FIRST FIVE MINUTES: IDENTIFYING LIFE-THREATENING CONDITIONS AND STABILIZATION PRINCIPLES

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ABSTRACT BODY:

Life-threatening emergencies are presented to veterinary practitioners (both in general, urgent and emergency practices) commonly. In some cases, the prognosis is poor- regardless of the interventions performed by the veterinary staff. In other cases, specific interventions may directly affect the patient's outcome. This session will provide an overview of the most impactful interventions during the 1st few minutes of common emergencies.

The ABC's should be followed when evaluating any patient, regardless of how "stable" they look on presentation. The initial triage consists of the primary survey of the animal. The main goal of that patient's initial assessment (primary survey) is to identify any life-threatening abnormalities in the airway, breathing and circulatory system (ABCs). Common conditions that will result in airway obstruction include brachycephalic syndrome, collapsing trachea, foreign body obstruction, hypersensitivity reaction, laryngeal edema, laryngeal paralysis, severe upper airway or facial trauma. Animals with airway obstruction classically present with audible respiratory noise, typically characterized by inspiratory stridor. Those animals may have an increased respiratory rate and effort and may also be orthopneic. Cyanosis may be present if oxygen intake is significantly affected. If respiratory failure is imminent, rapid anesthetic induction should be performed and the animal should be intubated (This one process MIGHT be lifesaving. Generally if the thought crosses your mind, it means you should do it!) Assisted ventilation should be performed in animals with inadequate ventilation (based on chest excursion or end tidal CO2). An emergency tracheostomy may be required in patients where orotracheal intubation is impossible. The patient may then be transported, ideally with a veterinary professional to a referral center. The patient may be extubated but some patients may need to be intubated for transport. A propofol CRI generally works well but there should be plans made for assisted ventilation during transport.

Diseases that can impair normal oxygenation and ventilation include pneumothorax, hemothorax, chylothorax, pyothorax, pulmonary edema, pulmonary thromboembolic disease or allergic airway disease. Animals with breathing difficulties are usually tachypneic, have increased respiratory rate and effort and may be cyanotic. Radiographs can be used to identify pleural space disease but should not be performed in unstable patients. If available, thoracic ultrasound (TFAST) should be used to identify pleural effusion. TFAST has also been described for identification of pneumothorax. Animals with breathing difficulty should be administered flow by oxygen immediately. A thoracocentesis should be performed when pleural space disease is identified or suspected. Animals with large amounts of pleural effusion or air should not be transported before the pleural space is evacuated. Butorphanol is usually well tolerated with respiratory disease, and provides good sedation to help with anxiety and allow for thoracocentesis.

Circulatory disturbances (shock) may sometimes not be as obvious as airway and breathing difficulties but are equally as important. Poor circulation leads to poor oxygen delivery, which ultimately causes cell death, organ dysfunction and patient death. Shock is usually easy to identify once the veterinarian has a clear idea of what they are looking for.

Causes of shock and poor circulation includes hypovolemia (GI, blood loss, severe dehydration), cardiogenic shock , obstructive shock (GDV, cardiac tamponade) or distributive shock (sepsis). Physical exam findings that indicate shock in dogs and cats include tachycardia, bradycardia, pale mucous membranes, prolonged capillary refill time, weak pulses and hypothermia. Additional diagnostics that supports shock is hypotension and elevated blood lactate levels.

Significant hemorrhage should be addressed immediately with direct pressure or ligation. Abdominal hemorrhage may be slowed with pelvic limb or abdominal compression bandage. Intravenous fluids are indicated in most cases of circulatory disturbances (except cardiogenic shock) . Blood products may be administered before transporting the patient.

Other Considerations Hyperkalemia should be treated with insulin and dextrose infusions. Sodium bicarbonate and beta agonists should also be considered if insulin is unavailable. Severe hypokalemia may affect diaphragm function and ventilation and should be rapidly corrected. Potassium may be infused at a maximum rate 0.5 mEq/kg/hr. Hypocalcemia should also be corrected with a calcium gluconate CRI (0.5-1.5 mL/kg bolus) and may be repeated until

clinical signs of hypocalcemia resolve. Hypoglycemia should be rapidly corrected with intravenous dextrose. Hyperglycemia rarely requires emergent therapy with insulin. Steroids are rarely indicated in all emergency patients except there is a high index of suspicion for hypoadrenocortical crisis or an allergic reaction. Steroids are not indicated in animals with shock or head trauma. Animals with seizures should be treated with injectable benzodiazepines. Long acting anti-epileptic drugs should be considered in animals with status epilepticus or animals with a cluster of seizures (more than 2 seizures in 24 hours). Patients with back or neck injuries should be transported in lateral recumbency on a solid surface. Fractures of the distal limbs should be stabilized with a bandage with the joint above and below the fracture incorporated. Penetrating objects should NOT be removed unless the clinician is prepared for immediate invasive surgery. Pain should be treated with systemic opioids. NSAIDS and alpha-2 agonists should be avoided in animals with cardiovascular compromise.

References available upon request

071 / #70

DO WE NORMALIZE EXTREME CONFORMATIONS IN DOGS AND CATS?

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ABSTRACT BODY:

DO WE NORMALIZE EXTREME CONFORMATIONS IN DOGS AND CATS?

Today there are over 400 dog and 70 cat breeds. Unfortunately, many of these breeds are predisposed to one or more genetic diseases. We selected only a limited number of dogs or cats to breed from for many years, leading to a loss of genetic diversity. That, in turn, led to hereditary diseases. Of course, none of us purposely selected for it, but it is an unwanted negative side effect of selective breeding.

However, we do select for a specific conformation. Some select for a functional conformation, whereas others prefer a conformation that prioritizes the appearance above the function. It can even happen within one breed. For example, a Working Line German Shepherd dog is selected first for its function, whereas a Show Line German Shepherd dog, above all, is selected for its looks.

Some prefer a more extreme conformation, like the sloping back of the Show Line German Shepherd, instead of a more moderate one. Shorter legs, flatter scull, longer ears or hardly any ears, tiny dogs or cats or extra-large, no hair or huge hair, you name it, and people are selecting for it. They consider these extreme conformations as being normal. But is it normal, or are we so used to it that we believe it is normal?

Let's first look at conformation.

Conformation refers to a dog or cat's physical characteristics, appearance, and structure. We could define pedigree dogs and cats as those with a known and recorded parental lineage that are registered with a kennel, breed club, or cat fancy. Most pedigree dogs and cats are bred to meet breed standards that lay down a prior human plan for the expected conformational characteristics of dogs or cats within that breed or type. Non-pedigree, look-alike, and crossbred dogs and cats also each have a set of physical characteristics that may be defined as their conformation but that are often more variable.

But when do we speak of extreme conformation?

Conformation can adversely affect the health and welfare of a dog or cat, particularly when an emphasis is made towards selecting for unhealthy exaggeration of certain traits. For example, a flat-faced (brachycephalic) dog or cat may have difficulty breathing in average day-to-day situations and may be especially challenged in warm weather or when exercising. A dog or cat with extensive skin folds may have partial obstruction of the eyes or nose and may show infection and inflammation of the folds. Features such as these that are associated with reduced health and welfare are viewed as extreme conformation. Some types of dogs, cats, and breeds show more extremes of conformation that impact their health and welfare than others.

What is a normal dog or cat?

The definition of normal varies from person to person, country to country, and culture to culture. An often-used definition of a normal (human) person is "A normal person has no serious physical or mental health problems." If we extrapolate that definition to dogs and cats, it implies they should be healthy to be normal. That raises the question: "what is a healthy dog or cat?"

Many cats have not undergone major changes during domestication, and their conformation remains very similar to their ancestors. Still, at the same time, the conformation of some cats/ breeds changed and became more extreme, like the flat face of a Persian cat. In dogs, the conformation of many dogs, especially breeds, has changed much since domestication started, and many suffer because of the desired look. Does this imply that to be normal, cats and dogs should look like their ancestors? Should dogs look more like wolves to be healthy? Some people would agree with that. Or should we focus on health and welfare, no matter what that looks like? There definitely is a need to define what is a healthy dog and cat. And remember, all these dog and cat breeds are not different species; a Chihuahua or a Great Dane belong to the same species: they are dogs!

Normalization

Normalization is a process whereby behaviors and ideas are made to seem "normal" through repetition, or through ideology, propaganda, etc., often to the point where they appear natural and taken for granted.

"Don't normalize this" has become a kind of rallying cry during President Trump's first year in office, a reminder not to get too acclimated to Trump's norm-breaking and extreme behavior. What happened is that it is not so much about Trump being 'abnormal' as well as

how ‘normal’ he makes other less extreme politicians look. There’s a concept in political theory developed by Joseph P. Overton that explains how this works. It suggests that there’s a “window” of acceptable ideas. Everything inside the window is normal and expected, while everything outside the window is radical, ridiculous, or unthinkable. Overton argued that the easiest way to move that window was to force people to consider ideas at the extremes, as far away from the window as possible. Because forcing people to consider an unthinkable idea, even if they reject it, would make all less extreme ideas seem acceptable by comparison, it would move the “window” slowly in that direction.

That “Overton Window” concept is also helpful for explaining why some extreme conformations in dogs and cats are considered as being ‘normal’. If you see Bulldogs collapsing or gasping for air, a Bulldog that ‘only’ snores becomes more acceptable. And if many Bulldogs snore, in time, you get used to it and might consider it as being normal to the breed.

Looking at some databases listing the frequency of certain diseases and symptoms, it is striking that in brachycephalic dogs, diseases related to Brachycephalic Obstructive Airway Syndrome (BOAS) are not on the top of the list. Is this because these dogs are even more prone to have other diseases? Or did we shift the “Overton Window” and consider less extreme symptoms related to BOAS,like snoring, as being normal, and therefore vets did not put it down in the patient chart, and owners do not realize there is an issue? In other words: did we normalize some health issues related to extreme conformation in dogs and cats?

The” Overton Window” can also be shifted in the other direction: by forcing to consider what is unthinkable for some: banning a breed, less radical ideas, like legislating breeds or excluding certain dogs from breeding, become the middle ground.

In practice, vets are confronted daily with a high number of patients with exaggeration of certain traits. Did we, therefore, arrive at the point where they appear natural to the breed and are taken for granted? Vets, being humans, are not immune to the normalization mechanism. As it is a subconscious process, we must look beyond what we are used to. Are we willing to actively raise the question? Being experts in animal health and welfare, vets should make owners aware of the suffering that can occur because of the exaggeration of certain traits. To do that, we need to be aware of it ourselves first.

072 / #179

IMAGING OF ELBOW DISEASE IN DOGS AND CATS: IMPROVING YOUR DIAGNOSTIC SKILL SET

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ABSTRACT BODY:

Veterinarians need to be proficient in interpreting elbow radiographs so as not to miss a lameness due to elbow dysplasia in their patients. The general practitioner is the front-line care provider who will be able to obtain and interpret elbow radiographs and interpret them accurately for the dog and the owner. Accurate interpretation requires knowing where to look, what to look for and that even mild abnormalities are critical to recognize. Despite the long existence of the IEWG, elbow dysplasia is still being missed and is evidence that continuing education on image interpretation of the elbow is important to learn and refresh as much as possible.

Interpretation begins with obtaining diagnostic images. Although the mediolateral flexed image is the minimal requirement for elbow grading, additional images are helpful, if not crucial, for a complete assessment. Furthermore, when radiographic findings are equivocal in a clinical elbow lameness, more sensitive tests like CT may be warranted. Mediolateral extended, and ML flexed and craniocaudal (Cr15°LCdMO) views are most commonly used to assess the elbow. Radiographs are scrutinized for primary and secondary lesions. The primary lesions are: medial coronoid disease, ununited anconeal process, incongruity and osteochondrosis. Secondary disease is recognized by the presence of osteoarthritis. OFA and IEWG grades are based largely on the presence of osteoarthrosis. The flexed mediolateral image is relied upon for identifying osteophytosis of the anconeal process, a secondary disease finding in ED.

In this lecture, challenging elbow cases will be presented with correlations to CT. Elbow dysplasia will be the main disease discussed but other diseases such as incomplete ossification of the humeral condyles, fractures, and delayed physeal closure of the distal radius and ulna will also be illustrated.

Medial Coronoid Disease

Disease of the medial coronoid process is best seen on the ML extended and Cr15°LCdMO views. Separated fragments (FCP), blunting of the craniomedial border of the MCP, absence of the MCP, trochlear notch sclerosis, and joint incongruity are signs of MCD. Blurring of the medial coronoid process cranial edge and ulnar trochlear notch sclerosis are reliable signs of elbow dysplasia and may be beneficial in screening protocols.

Ununited Anconeal Process

The UAP is best seen on the ML flexed view. There is generally an irregularly marginated gap between the anconeal process and the olecranon. The margins and surrounding bone are often sclerotic and can have osteophytes. Joint incongruity is also commonly seen.

Osteochondrosis

OC is best seen on the ML, Cr15°LCdMO, ML extended and ML flexed views. An articular margin lucency is present at the distal border of the medial humeral condyle, possibly with a sclerotic rim. It is uncommon to see a mineralized flap. Flattening of the condylar articular border can also be present. Kissing lesions of the humeral condylar border are often present with FCP but are more lateral than an OC lesion and often develop with chronic ED.

Incongruity

Incongruity of the humeroulnar joint is best seen on the ML extended view. A step between the lateral coronoid process and radial head and a more proximally positioned MCP, asymmetry of the humeroulnar joint space, widened humeroradial joint, indistinct border of the trochlear notch, and cranial displacement of the humerus. Incongruity can also indicate that the dog has experienced early closure of the distal radial and/or ulnar physes. Radiographs of the entire antebrachium are often necessary to recognize the source of elbow incongruity.

Osteoarthrosis

OA can be identified on all elbow views, but the ML flexed has been found most sensitive for osteophytosis of the anconeal process. Additional sites of osteophytosis include: cranioproximal border of the radial head, proximomedial border of the MCP, lateral border of the humeral epicondyle, and sclerosis of the base of the MCP (ulna, ulnar notch). Grading of the ED is based on the degree of anconeal osteochondrosis height; Grade 1: <2mm; Grade 2: 2-5mm; Grade 3: >5mm. Primary pathology is noted in addition to osteophytosis in the ED grading scheme.

Use of Computed Tomography

CT may be more sensitive (reported up to 93.8%) for

identifying medial compartment disease such as MCP fissures and fragmentation as well as subchondral sclerosis and shape of the MCP compared with radiography. This is likely most important for grade 0 and 1 elbows. For elbow grades 0 or 1 in a cohort of 46 dogs, CT has shown 62% with grade 0 to have ED, and 75% of grade 1, suggesting that ED can be missed on radiographs (Kunst 2014). All dogs in that cohort progressed to have OA after 1 year, in both groups. Proliferation of bone at the anconeal process may not be osteophytosis due to ED in all dogs and CT would be an important test to confirm ED when this is the only radiographic sign present. The use of CT is still under investigation as a screening tool for ED compared to radiography. CT can identify MCP fissure and fragmentation, subchondral sclerosis, osteophytosis, incongruity, radial incisure cyst-like lesions and OC lesions. In a study of 31 dogs, ulnar subtrochlear sclerosis was the most common radiographic finding in Labrador Retrievers less than 1 year of age and blurring of the cranial edge of the MCP was most common in dogs greater than 1 year of age. MCP fragmentation was the most common CT finding in all age groups.

CT is also used to assess for incomplete ossification of the humeral condyles in young dogs. Condylar sclerosis and fissures are the most common features.

073 / #72

DIAGNÓSTICO Y MANEJO DE PANCREATITIS FELINA

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ABSTRACT BODY:

La pancreatitis felina es una enfermedad **difícil de diagnosticar,** especialmente en los casos crónicos en los que los gatos no presentan signos clínicos de forma persistente.

Para aproximar el **diagnóstico** de la pancreatitis en los gatos es necesario que consideremos diferentes métodos como:

1. Los signos clínicos compatibles.

2. Examen físico con detección de dolor abdominal.

3. Ecografía.

4. Análisis laboratoriales: hematología, bioquímica y elevación sérica de lipasa pancreática felina inmunorreactiva (fPLI).

El **tratamiento es sintomático,** pero ha de centrarse en:

- Mantener el volumen de fluidos.

- Controlar el dolor, el vómito y la sensación de náusea.

- Prevenir infecciones secundarias.

Desde el **punto de vista clínico** podemos clasificar la pancreatitis en aguda y crónica, aunque no es fácil diferenciarlas porque la mayor parte de los gatos van a tener pancreatitis como reagudización de un proceso crónico (lo que se denomina aguda en crónica).

En la gran mayoría de los gatos con esta enfermedad no es posible identificar una causa subyacente y la pancreatitis en estos pacientes se considera **idiopática.**

En diversos estudios se ha demostrado una asociación entre pancreatitis, enfermedad inflamatoria intestinal (EII) y enfermedades hepáticas, principalmente colangitis.

El factor de riesgo más determinante para que se produzca pancreatitis es la **hipoperfusión** que, además de desencadenante, empeora el cuadro, por lo que hay que mantener al gato hidratado y normovolémico en caso de sedación o anestesia.

La forma de presentación de la pancreatitis puede ser leve y casi asintomática o aguda/necrotizante en las que el gato no sobrevive sin terapia intensiva.

Los signos clínicos en los gatos son difíciles de reconocer:

Dolor abdominal.

Vómito, menos frecuente que en el perro.

Anorexia y letargia son los signos clínicos más frecuentes.

Diagnóstico

El diagnóstico de pancreatitis debe ir encaminado al diagnóstico de enfermedades concurrentes e identificar las complicaciones como anemia, hipoalbuminemia o lipidosis hepáticas.

Análisis de sangre

La **hematología** puede mostrar leucocitosis o leucopenia.

Leucopenia, disminución de hematocrito y trombocitopenia implican también pronóstico negativo de pancreatitis.

Por otra parte, la **bioquímica** puede mostrar elevación de enzimas hepáticas y bilirrubina. Azotemia, hipoalbuminemia, hipercolesterolemia e hiperglucemia. La hipocalcemia debe considerarse factor pronóstico negativo, incluso en ausencia de signos clínicos asociados.

Debe considerarse el diagnóstico de pancreatitis en cualquier gato con **anorexia, letargia o vómito por causa desconocida.**

El diagnóstico diferencial ha de hacerse con lipidosis hepática, enfermedad inflamatoria hepática, nefritis intersticial, diabetes *mellitus.*

Plan diagnóstico

Es necesario evaluar la **función hepática:** ALT, ALKP, GGT, ácidos biliares, función gastrointestinal (folato y cobalamina).

Los cambios en la bioquímica sanguínea más frecuentes son:

La **fTLI** es útil para el diagnóstico de insuficiencia pancreática exocrina (EPI) pero no es sensible para la pancreatitis. Presenta una sensibilidad del 80 % en pancreatitis aguda, pero solo del 8 % en pancreatitis crónicas. Aumenta también por otras enfermedades gastrointestinales como enfermedad inflamatoria intestinal o linfoma.

La inmunorreactividad de la **lipasa pancreática felina (fPLI)**, tiene una sensibilidad y una especificidad del 100 % para las pancreatitis agudas, pero en las pancreatitis crónicas el grado de sensibilidad es del 54 % La magnitud de la fPLI está relacionada con pronóstico negativo de pancreatitis. La fPLI es más útil

en el diagnóstico de pancreatitis moderadas y graves, pero en las leves puede no estar aumentada.

Diagnóstico por imagen

Radiografía abdominal

En los gatos en los que se sospeche de pancreatitis, la radiografía deberá realizarse junto con **otras pruebas más sensibles y específicas.** Los posibles hallazgos radiográficos en gatos con pancreatitis son la disminución del detalle y del contraste en el abdomen craneal, dilatación del intestino delgado (por líquido o gas), hepatomegalia y presencia de una masa abdominal en posición craneal.

Ecografía abdominal

Los patrones ecográficos varían:

Páncreas hipoecogénico en necrosis pancreática.

Páncreas hiperecogénicos en fibrosis o masas pancreáticas.

Peritoneo hiperecogénico por necrosis de grasa mesentérica.

Región hipoecogénica alrededor del páncreas.

Efecto masa: en abscesos pancreáticos o pseudoquistes pancreáticos.

Dilatación del conducto pancreático.

La **citología** mediante punción con aguja fina del páncreas se considera relativamente segura. La presencia de células inflamatorias es específica de la pancreatitis. Sin embargo, como ocurre para la histopatología, pueden pasar desapercibidas lesiones muy localizadas. Por tanto, los resultados negativos no bastan para descartar una pancreatitis.

En medicina humana, la técnica no invasiva de elección en el diagnóstico de pancreatitis es el TAC, pero el precio, la disponibilidad y la dificultad de interpretación de imagen no hace que sea una técnica útil en la práctica clínica. Además, la combinación de ecografía y fPLI presenta mayor sensibilidad que el TAC.

Histopatología

Requiere estabilización del paciente para realizar toma de muestra de forma quirúrgica. Es la **técnica de elección** para el diagnóstico de pancreatitis. Es importante tener en cuenta que la distribución de la lesión puede ser focal y no detectarse alteración en la muestra tomada.

Dado que, en gatos, la pancreatitis suele aparecer de manera simultánea con enfermedades inflamatorias del hígado o del intestino, también deberá considerarse la realización de biopsias hepáticas e intestinales en casos de sospecha de pancreatitis.

Histológicamente, las pancreatitis se clasifican en agudas necrotizantes, agudas supurativas y forma crónica no supurativa, la más frecuente, que se caracteriza por infiltrado mononuclear y fibrosis.

La **combinación** de la historia clínica, fPLI, ecografía, citología y/o histopatología, cuándo sea posible, son las claves del diagnóstico de la pancreatitis en los gatos .

Tratamiento

Fluidoterapia

Los gatos con pancreatitis suelen estar **deshidratados** debido a los vómitos, diarrea o falta de ingesta de agua. En los casos graves, la pérdida rápida y significativa de líquidos puede provocar hipovolemia o incluso *shock* hipovolémico. Para mantener la perfusión tisular en general y la perfusión pancreática en particular, debe iniciarse, lo antes posible, una **fluidoterapia intravenosa** con cristaloides de reposición, normalmente una solución de lactato sódico compuesta (Ringer) o NaCl al 0,9 %, dependiendo de las anomalías electrolíticas simultáneas.

La administración de **plasma** ayuda a restablecer la presión oncótica mediante el aporte de albúmina y otras proteínas de alto peso molecular, así como factores de coagulación.

Nutrición

En los gatos con pancreatitis que llevan más de dos a tres días sin comer en el momento del diagnóstico debe considerarse la colocación de una **sonda** nasoesofágica, de esofagostomía o de gastrostomía.

La administración de mirtazapina transdérmica puede ayudar a que el gato empiece a comer.

Control de dolor

Los gatos con dolor de leve a moderado pueden ser controlados de manera eficaz con **buprenorfina** (0,01-0,03 mg/kg/4-8 h IV o IM). Los gatos con dolor intenso pueden controlarse con **fentanilo** (0,005-0,01 mg/kg/2 h IV, IM o SC o mediante infusión continua constante 0,002-0,004 mg/kg/hora).

El uso de **ácido ursodeoxicólico** disminuye el dolor porque aumenta el flujo biliar y pancreático.

Por su parte, la **S-adenosil metionina (SAMe)** aumenta las concentraciones de glutatión y mejora los niveles de enzimas pancreáticas.

La **suplementación con enzimas pancreáticas** administradas junto con la comida disminuye el dolor posprandial, según han observado los autores.

Antibiótico

La colonización bacteriana secundaria reduce el epitelio intestinal y causa disbiosis. Los

microorganismos que más frecuentemente colonizan el páncreas son Gram negativos y anaerobios del sistema gastrointestinal: *E. coli, Klebsiella* spp., *Enterobacteriaceae* o *Clostridium* spp.

Se debe administrar antibiótico cuando haya evidencia de **absceso pancreático o de inflamación sistémica:** leucocitosis, fiebre, hipoglucemia… Deben ser antibióticos de amplio espectro con buena penetración pancreática como **marbofloxacina, amoxicilina-clavulánico** o **clindamicina.**

Control del vómito y náuseas

El tratamiento con **antieméticos** debe iniciarse en todos los gatos con pancreatitis que vomiten.

El **maropitant** es un antiemético que actúa como inhibidor de los receptores NK1, muy eficaz en gatos, y puede utilizarse en dosis de 0,5-1,0 mg/kg/24 h IV, SC. Es conveniente utilizar el maropitant aunque el gato no vomite para evitar la sensación de náusea, que es muy molesta y dificulta la recuperación del gato. Los antagonistas dopaminérgicos como la **metoclopramida** (0,2-0,5 mg/kg/6-8 h IV, IM, SC, PO o 0,3 mg/kg/hora IV como una infusión constante) se consideran menos efectivos en los gatos. La metoclopramida como procinético favorece la clínica en gatos con íleo gastroduodenal.

Corticoides

El tratamiento más efectivo tanto antiinflamatorio como inmunomodulador para la enfermedad inflamatoria intestinal (y presumiblemente para la pancreatitis) son los **corticoides.**

Iniciamos la terapia con corticoides en pancreatititis después de 2 semanas de tratamiento antibiótico, pero no hay trabajos que avalen su eficacia.
La **prednisolona** se metaboliza más rápidamente que la prednisona en gatos y se administra en dosis de 0,5-1 mg/kg/12-24 h PO.

Pronóstico

Es **variable** y depende de la prontitud con la que se instaure una terapia intensiva, pero está documentada un 41 % de mortalidad en pancreatitis aguda.

Si observamos lo siguiente, el **pronóstico es negativo:**

Hipercaliemia.

Disnea.

Arritmias.

Concentraciones de fPLI>20 µg/l.

Hipocalcemia.

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SÍNDROME DE LA PIEL ATÓPICA FELINA: ELECCIÓN DEL MEJOR TRATAMIENTO

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ABSTRACT BODY:

La dermatitis atópica felina, atopia felina o síndrome atópico cutáneo felino (SCAF), se define como una dermatitis alérgica, de fuerte componente genético, mediada por la producción de IgE e IgG frente a alérgenos ambientales.

El diagnóstico de la DA felina es fundamentalmente clínico y se establece en función de la historia, cuadro clínico (prurito, lesiones) y tras descartar enfermedades infecciosas-parasitarias que puedan causar un cuadro similar. En esta charla nos centraremos en las opciones de tratamiento.

Tratamiento del SACF

Recomendaciones generales para los gatos con SAC:

- Los gatos atópicos deben recibir adecuado tratamiento antiparasitario (externo e interno) riguroso durante todo el año.
- Es recomendable una dieta de calidad, de limitadas proteínas y adecuada para gatos alérgicos (hay varias opciones en el mercado).
- Es recomendable realizar dos revisiones anuales, para evaluar la presencia de infecciones secundarias cutáneas (*Malassezia*), otitis externa o efectos adversos del tratamiento.
- Se ha comprobado que el stress y la ansiedad agravan notablemente el cuadro clínico del SAF. Se recomienda introducir medidas que mejoren el bienestar del gato, incluido el uso de feromonas (ver artículo de Titeux et al, 2018).

Existen diferentes opciones terapéuticas, que hay que evaluar cuidadosamente y seleccionar de acuerdo con las características de cada paciente.

Inmunoterapia alérgeno específica. Requiere la identificación de los principales alérgenos implicados en un test serológico o intradérmico. El test intradérmico es de difícil lectura en gatos (incluso con el uso de fluoresceína) y solo es una opción para dermatólogos expertos. Es más recomendable un buen test serológico de un laboratorio de confianza y con buen sistema de control de calidad. Si los resultados del test son claros y tienen sentido a la luz de la historia clínica, es posible realizar inmunoterapia mediante la administración semanal SC de extractos antigénicos acuosos. En gatos, la administración sublingual (SLIT) (cada 12h) suele ser ventajosa y muchos dermatólogos la prefieren.

No hay datos procedentes de estudios clínicos controlados sobre la eficacia de la inmunoterapia en gatos alérgicos. Algunos autores afirman que entre un 50% y un 70% muestran alguna mejoría clínica (Roosje et al, 2002). Según algunos autores, en gatos, la SLIT es incluso mas efectiva que la subcutánea (DeBoer, comunicación personal).

La **ciclosporina** es probablemente la opción que en estos momentos presenta un mejor perfil combinado de eficacia y seguridad. Se administra a una dosis de 7-8mg/kg/q 24h durante un mes (mínimo) y luego se intenta la reducción de la dosis, hasta llegar a administra esta dosis cada 48 horas o dos veces a la semana (mantenimiento).

Precauciones antes de iniciar tratamiento con ciclosporina:

- Comprobar que el gato es FelV-FIV negativo.
- Limitar el acceso a comida no controlada y a la caza para prevenir toxoplasmosis. Únicamente debe recibir alimento comercial preparado.

Los principales efectos adversos de la ciclosporina en gatos son gastrointestinales. Vómitos, diarreas, salivación intensa y rechazo a la ingestión son relativamente frecuentes. Sin embargo, hay que tener en cuenta que en la mayoría de casos son efectos leves, que remiten con la interrupción de la medicación y que se pueden controlar o aminorar. Algunas recomendaciones en este sentido:

- Administrar siempre el tratamiento con comida (a la vez, justo después o mezclando la solución con la comida).
- Si hay efectos adversos, se puede interrumpir el tratamiento y empezar otra vez con dosis pequeñas, e irlas aumentando.
- Si la solución no se tolera bien, se puede probar con las capsulas para perros (25mg; 25mg + 10 mg o 50 mg en cao de gatos de más de 6 kg).

Es recomendable realizar periódicamente (cada seis meses o al menos anualmente) una analítica general a los gatos que reciben de forma constante tratamiento con ciclosporina.

Los **glucocorticoides** son otra alternativa de demostrada eficacia (Ganz *et al,* 2013). El esteroide de elección es la prednisolona a dosis inicial de de 1-1.5 mg/kg/24 horas. Las alternativas son la

metilprednisolona (1.2-1.4 mg/kg/24h), triamcinolona (0.18mg/kg/q 24h) y la dexametasona.

Oclacitinib, un inhibidor de la janus kinasa 1, se ha demostrado muy efectivo en el control de las dermatitis alérgicas caninas. Recientemente se ha empezado a utilizar en el tratamiento del SACF. Se recomienda a una dosis de 1 mg/kg inicialmente cada 12 horas y luego cada 24 horas. Tres estudios publicados indican una eficacia entre el 40% y el 80%.

No se ha demostrado en estudios controladas una eficacia de los **antihistamínicos** en el control del SACF y hay opiniones divergentes sobre su valor en el tratamiento de la atopia felina. La opinión de muchos dermatólogos es que hay algunos gatos en los que se observa una buena respuesta, en especial cuando se combina con esteroides, porque permiten reducir las dosis de esteroides. Si se va a considerar el uso de antihistamínicos, algunas recomendaciones son:

- Administrarlos durante un periodo prolongado, de al menos dos semanas antes de evaluar su efecto.
- Administrarlos a dosis adecuada (en la banda alta) y a la frecuencia adecuada.
- Combinarlos con otras terapias.

Las mejores opciones para gatos con SACF son:

- Clemastina: 3-4 mg/kg/12 horas
- Cetirizina: 5 mg/kg/24horas
- Fexofenadina: 10 mg/kg/12-24 horas

Una opción para casos que no han respondido a los tratamientos mencionados anteriormente es el **maropitant** (2mg/kg/24h). No hay estudios controlados, pero parece ser efectivo en algunos gatos con dermatitis pruriginosas.

El **anticuerpo monoclonal frente a la IL-31** canina (lokivetmab) no es efectivo en gatos y no debe administrarse. Es especie especifico y podría generar reacciones adversas. Es posible que en el futuro se disponga de un anticuerpo similar, pero especifico para la IL-31 felina.

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075 / #299

SHINING A LIGHT ON THE IMPORTANCE OF UVB LIGHTING FOR CAPTIVE EXOTIC ANIMALS

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ABSTRACT BODY:

SHINING A LIGHT ON THE IMPORTANCE OF UVB LIGHTING FOR CAPTIVE EXOTIC ANIMALS

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Lighting remains an important component of exotic pet husbandry, and is a commonly discussed topic amongst veterinarians and exotic pet owners. The majority of the discussions related to this topic are focused on “full spectrum” lighting. This is a term that has been used (and manipulated) in many different ways; however, the basic premise behind this type of lighting system is that it provides the essential spectrums of ultraviolet, visible, and infrared light required by captive animals, mimicking natural exposure to the sun. The focus of this lecture will be on ultraviolet B radiation.

Ultraviolet light is produced by electromagnetic radiation. The wavelengths for ultraviolet radiation are shorter than those for visible and infrared light. Ultraviolet radiation is generally discussed in relation to those categories important to vertebrates: Ultraviolet A, B, and C. Ultraviolet C radiation represents the shortest wavelengths of the three classes (<280 nanometers). This range of ultraviolet radiation is germicidal, and is commonly used to control pathogens in aquatic systems. Ultraviolet B radiation provides the medium range ultraviolet radiation (280-315 nanometers). Ultraviolet A radiation represents the longest rays of the group and is characterized as “black light” (> 315-380 nanometers). Ultraviolet B radiation represents the range considered important in the synthesis of vitamin D3. Vitamin D3 is an essential hormone that plays many different important physiologic roles. Its role in calcium metabolism is probably its most recognized function, where it helps to ensure the development and maintenance of healthy bones. In reptiles, maintaining appropriate levels of vitamin D3 has also been found to be associated with increased reproductive success. Ultraviolet C is not generally discussed at any great extent, although it is considered important in regulating behavior in vertebrates.

There are two primary methods for obtaining vitamin D3: synthesizing it from exposure to ultraviolet B radiation or consuming a food source that has synthesized the hormone through exposure to the sun. The production of vitamin D occurs as a result of the photosynthetic conversion of 7-dehydrocholesterol to pre-vitamin D3. Pre-vitamin D3 is converted to vitamin D3 via a temperature dependent process. At this stage, the hormone is transported to the liver where it is hydroxylated to 25-hydroxyvitamin D3. The kidneys serve as the site for the final conversion of the hormone to 1, 25-hydroxyvitamin D3, which represents the active form.

Vitamin D is considered important in vertebrates because it plays many different roles in the body. Because captive reptiles are routinely maintained indoors, the use of “full spectrum” lighting has become an important consideration for ensuring that captive, non-carnivorous species can obtain vitamin D3. Until recently, studies evaluating the importance of full spectrum lighting in reptiles have been limited. However, original research from the author’s laboratory has shown that 25-hydroxyvitamin D concentrations in lizards, snakes, turtles, rodents, and lagomorphs could be significantly increased after exposure to appropriate full spectrum lighting. It has generally been accepted that these animals obtain their vitamin D through their diet; however, the results of the studies suggest that both of these species can generate endogenous vitamin D, like humans, from direct stimulation to appropriate artificial lighting. Moreover, research by the author’s team has found that leopard geckos (Eublepharis macularius), a crepuscular species, were also found to have higher concentrations of 25-hydroxyvitamin D following exposure to UVB. In the leopard geckos, two studies were performed, one evaluating a 12-hour UVB photoperiod and a second evaluating a 2-hour photoperiod. In the latter study, one hour of UVB exposure was provided in the morning and one hour at night. This was done to mimic dawn and dusk, the times a crepuscular species would be expected to be exposed to sunlight. The leopard gecko 25-hydroxyvitamin D concentrations were similar between the two studies, suggesting that these lizards regulate how much vitamin D they produce. The 25-hydroxyvitamid D concentrations were quite different between these three groups, and even species that appear similar can be quite different. Studies done

in our laboratory found juvenile Blanding's turtles (Emydoidea blandingii) to have much lower vitamin D concentrations than the red-eared slider turtles (Trachemys scripta elegans). While these species may share habitat, they bask differently, and we believe this could account for the differences seen. The Blanding's turtles were also younger animals, so this may have also accounted for the difference we found. Compact fluorescent compact light bulbs were used for the studies. The bulbs were placed within 6-9 inches of the study animals' basking spots. The findings of these studies confirm the importance of using full spectrum lighting for captive exotic species; however, additional work is needed to evaluate other exotic species being maintained in captivity.

When making recommendations regarding lighting, it is important to recognize that not all bulbs are created equal. Although "full-spectrum" lights may appear similar, they can produce vastly different quantities of ultraviolet B radiation. To confirm the quantity of ultraviolet B radiation being produced by a bulb, it is important to measure the intensity of the radiation using an appropriate radiometer/photometer. The distance the bulb is placed to a basking reptile can also have an effect on the quantity and intensity of light reaching an animal. "Full-spectrum" lights should not be shown through glass, as the glass can defract the ultraviolet B radiation. Historically, only fluorescent tube light bulbs produced any significant quantity of ultraviolet B radiation; however, some compact fluorescent bulbs and mercury vapor bulbs can also produce appropriate to high levels of ultraviolet B radiation.

While ultraviolet B radiation is generally considered important for animals in captivity, it is not without its risks. Photokeratitis has been observed by the author and reported by others in reptiles exposed to certain types of artificial ultraviolet B radiation. Fortunately, removing the light source corrected the problem in the author's experience. There is also concern that some species (e.g., bearded dragons) may develop skin cancer (e.g., squamous cell carcinoma) from exposure to ultraviolet B radiation. Additional research is needed to elucidate the risks associated with exposure to ultraviolet B radiation from artificial lights.

UVB appears to be an important requirement for the majority of the exotic species that have been studied. Based on our current understanding of UVB, the author believes it is important to provide these animals UVB lighting. However, the amount of UVB exposure (watts, time, etc) needs to be limited. At this time, the author recommends no more than 2 hours of UVB lighting a day. This is recommended to reduce the risk of side effects, while ensuring these animals can produce this important hormone. More research is needed, and the author hopes to continue to pursue this research into the future.

076 / #68

BEHAVIOURAL SIGNS OF PAIN IN CATS AND DOGS

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ABSTRACT BODY:

Cats and Dogs are commonly presented to veterinary clinics with a variety of potentially painful conditions. Despite the development of a variety of resources for the assessment of pain in cats and dogs, pain assessment may still be challenging for many veterinary practitioners. This presentation will outline some of the challenges of pain assessment for canine and feline patients, and outline available resources to support more effective veterinary pain assessment.

Pain in a complex phenomenon and has a huge impact on animal welfare, but is one of the areas of veterinary medicine that the profession is well equipped to deal with. Each individual animal will experience pain differently. Pain is best managed early and aggressively; it is much more difficult to control pain once it is well established than it is to manage pain before it becomes severe. Where possible, preventing pain should be the aim of the analgesic plan. Surgical pain is 100% predictable and therefore a good analgesia plan should be in place prior to undertaking any procedure.

The term 'pre-emptive analgesia' refers to the treatment of pain using analgesic drugs before the introduction of a noxious stimulus (i.e. surgery). Reducing the nociceptive input to the spinal cord reduces peripheral and central sensitization and therefore also reduces peri- and postoperative pain and hyperalgesia. When prescribing analgesics, the type of pain being experienced is important; pain may be caused by inflammation (inflammatory pain), tissue damage (nociceptive pain) or nerve damage (neuropathic pain). An individual may experience several types of pain concurrently and different medications may be required to adequately manage the different types of pain. Different classes of drugs act at different sites along the pain pathway and thus can often be safely used in combination to provide optimal analgesia. The term multimodel analgesia is given to this approach to treating pain. Combining different classes of analgesic drugs ensures a more effective and enhanced management of pain, and the resulting synergistic effect allows lower doses of each individual drug to administered, thereby reducing the possibility of side effects.

All patients benefit from objective postoperative pain assessment, even with multimodal preoperative analgesia, it is still possible that patients may experience postoperative pain and so their individual responses to analgesia must be assessed. Validated pain assessment scales such as the Glasgow Short Form Composite pain scale and the Colorado University Pain scale are useful tools in evaluating acute postoperative pain in cats and dogs. For Veterinarians working in CNR projects, a recent study analysing facial expressions and body posture of 35 dogs analysed before and after surgery identified eight potentially pain-related changes (flank watching, curved back, tucked abdomen, rigid limbs, flattened ears, orbital tightening and lowered head action behaviours were significantly more frequently observed four hours after surgery than pre-surgery, tucked abdomen, curved back, rigid limbs, and flank watching behaviours, were observed significantly more frequently 24 hours after surgery than pre-surgery. Pain assessments are an important tool in safeguarding animal welfare during surgical interventions, such as in spay-neuter programmes and these behaviours may be used as a reliable assessment scale to identify pain in free-roaming dogs after neutering (1, 2).

When using behaviour as an indicator of pain it should be recognised that behavioural responses to pain may be varied and so one of the most reliable indicators is a change in behaviour compared to the baseline. A behavioural response involves not only what an animal does but also when, how, where and why it exhibits the behaviour. Behaviour should not be considered in isolation but in the context of the species and situation in which it occurs (3, 4). Because pain is an emotional as well as sensory experience, there are individual (as well as species) differences in how animals respond to pain and these are in part related to the genetic variation in such factors as the number, distribution and morphology of opioid receptors. However some signs do seem to be applicable across species, and changes in behaviour, reduced activity and sensitivity to noises should always be investigated as potential pain indicators (5). Several studies have demonstrated the link between undiagnosed pain and behavioural change or behavioural disorders in animals (6-8) Assessments such as the Helsinki chronic pain index (9) are useful in assessing chronic pain.

When such signs are recognized, the animal's

analgesic support should be re-evaluated. Additionally veterinary professionals can also mitigate the emotional experience of pain as well as the sensory experience, which is managed through the analgesic plan and provision of a comfortable resting area. Pain is defined as 'An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage', (International Association for the Study of Pain), and so mitigating the emotional as well as the sensory experience is an important aspect of pain management. The emotional response to pain may be manipulated by the provision of a suitable quiet calm environment that allows the patient to sleep and rest and which is free of threatening stimuli such as noises, aversive odours and predatory animals. Additionally, human comfort and reassurance, palatable food, toileting and hiding opportunities, and for longer-term patients, appropriate enrichment, can all help to support positive emotional well-being and mitigate the pain experience.

Whilst tools such as crates and buster collars are useful to restrict activity and support surgical recovery, they do not offer analgesic action and so should always be used in conjunction with effective analgesics. Buster collars also obstruct normal behaviour such as sleeping, eating, and grooming, are stressful for both pet and owner (10), alter auditory ability and do not address the cause of wound interference. Considerate clipping and preparation of the surgery site, delicate tissue handling, pre-emptive and multi-modal analgesia, and adequate post-operative pain relief will all help to prevent the need for buster collars.

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077 / #267

REDEFINING FELINE MAMMARY TUMOR MEDICAL AND SURGICAL MANAGEMENT

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ABSTRACT BODY:

Incidence

Mammary tumours occur less frequently in cats than in dogs. It is estimated that the incidence in cats is approximately half of that of dogs and humans. Mammary tumours in cats are much more frequently malignant than in dogs, with approximately 80% being malignant. The effect of ovariectomy in preventing the occurrence of mammary tumours is also present in the cat as in the dog, however, it is not as strong. The fact that oestrogen receptors were only found in about 10% of mammary tumours in cats (versus approximately 60% in dogs) may account for this difference. Many feline mammary tumours appear to be attached to the skin and may be ulcerated. Lymph node invasion is frequently present. Several studies have shown metastases to be present in approximately 80% of cats with mammary tumours. The main organs of metastasis are the lymph nodes, the lungs, and the pleura. Mammary tumours should be differentiated from mammary hyperplasia.

Mammary hyperplasia

Hyperplasia may be lobular or fibroepithelial. Lobular hyperplasia usually occurs in older cats and is palpated as a mass in one or more glands (enlarged lobule with a cystic dilated duct). Fibroepithelial hyperplasia usually occurs in young cats in cyclus or pregnant cats. The glands are diffusely enlarged, oedematous, and reddened. Hyperplasia is thought to be caused by hormonal stimulation of the glandular tissue.

Clinical signs

Tumours are often presented to the veterinarian at an advanced stage, with ulcerated masses found in a quarter of the cases. In more than half the cases, several different glands are involved. In some cases, extensive involvement of the lungs or pleura may cause respiratory distress. Pleural carcinomatosis may cause a pleural effusion containing malignant cells.

Surgical treatment

Radical mastectomy is the method of choice for treating a mammary tumour in cats and consists of the removal of all the mammary glands and lymph nodes on the affected side.

Surgical principles:

- Cats usually only have four pairs of mammary glands
- Always perform a complete unilateral or bilateral mastectomy
- Remove fascia, if the tumour is fixed to the underlying fascia and muscles
- Bilateral mastectomy may be staged (6 weeks apart) or simultaneous
- The inguinal lymph nodes are usually automatically removed with the fourth gland
- Remove axillary lymph nodes only if enlarged or cytologically positive for tumour

Step-by-step description of a unilateral mastectomy

An elliptical incision is performed around the eight mammary glands with a minimum of 1 cm from the tumour. The incision is continued through subcutaneous tissue to the external fascia of the abdominal wall. Superficial haemorrhage is controlled with electrocoagulation, haemostats and/or ligation. An en-bloc excision of the mammary glands is performed by elevating one edge of the incision and separating subcutaneous tissue from the muscle fascia by using a smooth gliding motion of the scissors (Metzenbaum). Use traction on the elevated skin to facilitate dissection. The inguinal fat pads are to be removed, containing the inguinal lymph nodes. The fascia is excised if the tumour has invaded underlying tissues, and a portion of the body wall is removed if the tumour has invaded the musculature. The gliding scissor dissection continues until major vessels are encountered: the cranial and caudal superficial epigastric vessels. Both vessel pairs need careful isolation and ligation. The specimen is removed, and the skin advanced toward the centre of the incision, if necessary, using walking sutures (see Figure 1). The skin edges are apposed with a subcutaneous suture pattern, tacking the subcutaneous tissue to the underlying fascia. The skin is closed with appositional skin sutures.

Postoperative care

Most often, antibiotics are not indicated after mastectomy except when long surgery times are obtained. Pain management is to facilitate recovery. The use of bandages is often ill-tolerated by cats, and a

simple e-collar will do the trick. Reevaluation should be planned every 3-4 months to check for local recurrence and metastases.

Prognosis

Important predictors of prognosis in both cats and dogs are tumor size (< 2 cm^3 has a significantly better prognosis), tumor type, tumor stage, and ulceration.

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REDEFINING FELINE MAMMARY TUMOR MEDICAL AND SURGICAL MANAGEMENT

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ABSTRACT BODY:

Management of cats with mammary gland tumors (MGT) is a collaboration between a medical oncologist and a surgical oncologist. This presentation is a collaboration between a medical oncologist and a surgical oncologist to highlight the latest thinking and research on the treatment of feline MGT. From the medical oncology standpoint, this presentation will focus on six phases of the management of feline mammary gland tumors. Information on the surgical management of feline MGT is in an abstract by Dr. Jolle Kirpensteijn.

Medical Management of Feline Mammary Tumors

Presurgical evaluation/ tumor staging

The goal of a presurgical evaluation of a cat with mammary tumors is twofold. First, to ensure there are no contraindications to general anesthesia and surgery. Second to determine the extent of tumor. Because feline mammary gland tumors are highly metastatic, the presurgical evaluation will determine surgery is not the right treatment for some cats. A CBC, biochemical profile, urinalysis, T4 form the basis of testing. A three view thoracic radiograph is mandatory since tumor metastasis occur most frequently in the lungs. The need for an abdominal ultrasound depends on the individual case.

Biopsy interpretation, treatment recommendation, prognostication

In general, cats with MGT live about 12 months from the time of diagnosis.[1] Grade and stage help determine if the survival time will be longer or shorter than average. There are multiple feline MGT grading schemes. No single feline mammary tumor grading system is universally accepted; however, histopathological grade (or how malignant a tumor appears on histology) is predictive of outcome. Lower grade tumors have a longer survival.

In addition to tumor grade, tumor stage (or how extensive a tumor is) is set of tumor features used to determine prognosis. Cat with lower tumor stage live longer.

- Stage 1: < 1cm primary tumor; no metastasis
- Stage 2: <= 3cm primary tumor with regional metastatic lymph nodes that are NOT fixed
- Stage 3: any >3cm primary tumor w/o distant metastasis, or any size primary tumor with regional metastatic lymph nodes that ARE fixed but w/o distant metastasis
- Stage 4: any with distant metastasis

Postoperative chemotherapy

The goal of chemotherapy in any patient is to kill any cancer cells that have escaped the tumor prior to surgical removal of the tumor and prevent those cells from becoming metastatic. Chemotherapy is the appropriate treatment to prevent metastatic tumors, but to date, data on effective chemotherapy for feline MGT is lacking. Drugs that have been used to treat feline mammary tumors include doxorubicin, cyclophosphamide, carboplatin and NSAIDS (meloxicam).[2,3,4]

Postoperative monitoring

Guidelines for post operative monitoring of cats following mastectomy have not been developed. It is reasonable to obtain three view thoracic radiographs every 3 months to monitor for metastatic disease. The utility of abdominal ultrasound in detecting metastatic disease has not been determined in feline mammary gland tumors.

	Location of metastatic mammary gland tumors	
	Weijer[5]	Moulton[6]
Lungs	85%	83%
Lymph nodes		83%
Pleura	76%	42%
Adrenal glands	19%	
Pleura	18%	
Spleen	10%	
Kidneys, ovaries, other organs	≤8%	24% liver

Control of metastatic disease

Cats with stage IV MGT generally have a very short survival time, but recent data suggests some cats with stage IV MGT have a longer survival. Cat without pleural effusion or clinical signs have a median survival of approximately 3 months. Cats with effusion of clinical signs live a few weeks. For cats with stage IV MGT, treatment with toceranib phosphate (Palladia®) should be considered if the cat does not already have

pleural effusion and/or respiratory distress. Cats with pleural effusion and/or respiratory distress

Referral to surgery for recurrent disease

Quality of life is important to cat owners. When MGT become ulcerated, it results in a poor quality of life for both the human and the feline. For some cats with limited metastatic diseases, removal of a recurrent MGT to prevent ulceration results in an improved quality of life.

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079 / #178

CO-INFECTIONS WITH MULTIPLE VECTOR-BORNE PATHOGENS IN DOGS WITH LEISHMANIOSIS

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ABSTRACT BODY:

In the Mediterranean basin and in other different Countries of the world, mainly Brazil, leishmaniosis due to *Leishmania infantum* is one of the most prevalent vector-borne disease in dogs. A broad range of clinical manifestations have been described in canine leishmaniosis. Canine leishmaniasis (CanL) is a progressing disease characterized by a broad spectrum of signs, ranging from asymptomatic/subclinical infection, frequently characterized by negative *Leishmania* serology and general healthy status, to severe, fatal disease condition. Subclinical infection is featured by the detection of parasite by the PCR method and/or the appearance of low antibody titers. In these dogs, a strong cellular immune response persists, with a balanced levels of tissue-specific immunity to not cause immune-mediated tissue damage and subsequent disease. The severity of late-stage disease is correlated with high antibody levels and increasing parasite load. Therefore, a clinical staging system in this infection is important to establish an accurate prognosis and treatment. However, host and parasite factors that determine clinical outcome are poorly understood. Dogs with clinical leishmaniosis are often concurrently infected with multiple pathogens, which are frequently vector-borne, such as *Ehrlichia canis*, the causative agent for canine monocytic ehrlichiosis, *Anaplasma platys*, *Babesia vogeli* and *Hepatozoon canis*, resulting in an unpredictable incubation period, atypical clinical outcome and poorer prognosis, compared with dogs infected with *L. infantum* alone. It has been reported that infections with other vector borne organisms can affect the severity of canine leishmaniosis or mimic its clinical signs and/or clinicopathological abnormalities. It is important to recognize the difference between co-infection and co-exposure. Co-infection indicates the presence of the pathogen's nucleic material or direct evaluation of both *L. infantum* and another pathogenic microbe, while co-exposure indicates the detection of antibodies against both pathogens. Several studies have demonstrated that dogs exposed to numerous tick-borne co-infections have a higher relative risk of progression to clinical leishmaniosis. For this reason, veterinarians need to be suspicious of coinfections in dogs with clinical leishmaniosis when 1) presence of uncommon clinicopathological abnormalities; 2) presence of atypical clinical signs; 3) not responding well to treatment and 4) frequent clinical relapses. It is also important to highlight the use of specific treatment for each pathogen diagnosed. Dogs with CanL and co-infections (9 of 99 tested dogs in Portugal) with either *E. canis, B. canis, and Rickettsia conorii* had shorter survival time ($p = 0.0142$) [54]. Additionally, Toepp et al. (2019) found that dogs with multiple tick-borne co-infections had statistically significant increased risk for progressed CanL and increased risk for mortality [10]. The prevention against ticks, fleas and sand flies is also extremely important. The aim of this talk is to provide an updated overview of co-infections with other vector-borne pathogens in dogs with leishmaniosis focusing on clinical aspects, diagnosis, prognosis, treatment and prevention.

To give a more comprehensive example of the diagnostic and follow up complexity of Leishmania co-infected dogs, a brief description of two clinical cases of *L. infantum – B. gibsoni* co-infection is reported following.

Two dogs, a 7-year-old neutered female Pit Bull Terrier (Diana) and a 3-year-old neutered female crossbreed (Biba) were referred to the Veterinary Teaching Hospital of the Department of Veterinary Medicine and Animal Production, University of Naples Federico II, for a two weeks history of lethargy and urine leakage. Three years before she arrived at our University, Diana, has been already diagnosed with *Babesia* spp. and *Leishmania infantum* infections and treated with imidocarb dipropionate and various cycles of therapy with miltefosine and allopurinol. The medical history of Biba included *Leishmania infantum* infection and adrenal insufficiency (Addison's disease) diagnosed at the age of two years. The clinicopathological findings of both dogs revealed lethargy, anorexia, marked splenomegaly, proteinuria and haemoglobinuria. Blood examination demonstrated severe macrocytic normochromic anemia with evidence of marked regeneration, left shift neutrophilia, monocytosis, marked thrombocytopenia, hyperproteinemia and hypoalbuminemia. Blood smear evaluation revealed single to multiple, variable sized (1–3 µm in diameter),

and round to oval to band-like piroplasms within many red blood cells consistent with small forms of *Babesia* spp. or *Theileria* spp. A partial segment of the 18S rRNA gene was amplified and the PCR product was analyzed by direct sequencing. The nucleotide sequence was completely identical to that of *B. gibsoni* present in GenBank. One week after the end of the therapy (10 days) with atovaquone (13.3mg/kg TID, *per os*) and azithromycin (10mg/kg, SID, *per os*), piroplasms were not observed on blood smear but the Diana's clinical condition did not markedly improve and the blood PCR remained positive. Instead, Biba showed improvements at both evaluations of blood work analysis and abdominal echocardiography. However, the PCR test resulted positive. Both dogs were followed for 4 months by blood tests, evaluation of the spleen and kidney, PCR tests for *B. gibsoni*. Moreover, both dogs received another cycle of treatment with atovaquone (13.3mg/kg TID, *per os*) and azithromycin (10mg/kg, SID, *per os*). After the second treatment, two weeks after, Diana showed chronic interstitial glomerulo-nephritis and slightly reduced the size of the spleen, mild anemia and thrombocytopenia. Instead Biba, besides a mild anemia, showed no abnormalities. The PCR test resulted positive for both dogs. After one month from the second treatment, both dogs were negative at the PCR test for *B. gibsoni*. The clinical improvements of Biba were maintained until 4 months of follow-up. Diana, showed constantly mild regenerative anemia and thrombocytopenia, as well as chronic interstitial glomerulo-nephritis. Veterinarians should be aware of the possibility that dogs may harbor more than one vector born pathogen, making difficult the diagnosis and treatment.

080 / #102

TÉCNICAS LAPAROSCÓPICAS DEL APARATO REPRODUCTOR DE PERROS Y GATOS

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ABSTRACT BODY:

Introducción: La creación de la videocirugía y el desarrollo de la laparoscopia es una evolución considerada por muchos como la tercera revolución en cirugía, después de la anestesia y la era de los antibióticos. La edad moderna de la cirugía de mínima invasión en medicina humana tuvo su auge hacia finales de la década de 1980. Por el contrario, en medicina veterinaria, es un paradigma que se está desarrollando en nuestros días. Existe evidencia en pacientes veterinarios que la práctica de laparoscopia, reduce la severidad y la incidencia de ciertas morbilidades quirúrgicas en comparación con la cirugía convencional.

La cirugía laparoscópica durante los últimos diez años, ha despertado gran interés entre los veterinarios debido a las ventajas reportadas para los pacientes, las cuales comprenden menor traumatismo quirúrgico, seguridad terapéutica real y una recuperación más rápida.

La cirugía laparoscópica se está estableciendo en medicina veterinaria. Sin embargo, implica un mayor desafío para el cirujano veterinario debido a: la visión monocular ampliada, perdida de la sensación táctil, ampliación de los tremores del operador, reducción de los grados de libertad y la adopción de posiciones anómalas durante periodos de tiempo relativamente largos de cirugía.

Como desventajas, la inversión económica en cuanto al equipamiento laparoscópico, seguido de la inversión durante el periodo de formación. La cirugía laparoscópica y sus destrezas implícitas requieren de una curva de aprendizaje que deberá superarse de manera gradual con métodos no invasivos, como los simuladores, salvaguardando así al paciente de morbilidades. Los simuladores laparoscópicos permiten al cirujano adquirir suficiente destreza como para manejar nuevos instrumentos quirúrgicos antes de aplicarlos en programas experimentales o en situaciones quirúrgicas reales.

Equipo o torre laparoscópica: Una torre de laparoscopia básica está conformada por los siguientes elementos:

Monitores: Uno o dos colocados frente al cirujano y otro enfrente al ayudante.

Unidad de captura de video: Sistema de adquisición de imágenes proveniente de la cámara, la procesa y envía hacia el monitor para una visualización inmediata.

Unidad de fuente de luz: Emite la iluminación necesaria que es transmitida hasta el telescopio por medio de un cable de fibra óptica.

Unidad de insuflación de CO2: Dispositivo para insuflar CO2, necesario para la creación de un neumoperitoneo mediante la insuflación de CO2 en la cavidad abdominal.

Reproductor y grabadora: Los sistemas digitales modernos de reproducción y grabación (DVD, DV, DVCAM) se han incorporado de manera reciente en la torre de laparoscopia.

Cámara laparoscópica: Son ligeras y compactas, y transmiten la imagen desde la óptica hasta la unidad de video captura. Los elementos esenciales en el sistema de imágenes son los chips CCD (charge-coupled device).

Óptica rígida o telescopios: La pieza ocular se adapta a la cámara por medio de un adaptador universal y el cable de fibra óptica se conecta al conector del telescopio.

Existe cierto equipo accesorio, que podría incluirse como parte de la torre laparoscópica y constituir herramientas útiles durante el desarrollo de procedimientos laparoscópicos. Entre las diferentes alternativas, resaltamos: los sistemas de electrocoagulación y un equipo aspirador e irrigador.

Instrumentos laparoscópicos: Existen múltiples alternativas y variantes de cada herramienta, que pueden variar en diseño, materiales, fabricantes y costos. Los instrumentos de laparoscopia se pueden clasificarse en tres grupos de acuerdo con su función y uso específico durante el desarrollo de procedimientos quirúrgicos:

- El primer grupo incluye instrumentos de acceso, tales como trocares y la aguja de Veress.

- El segundo grupo comprende a los instrumentos de disección y sujeción, pinzas, tijeras y retractores.

- El último grupo considera a los instrumentos específicos utilizados para maniobras adicionales y deberán utilizarse de acuerdo con el procedimiento propuesto.

Insuflación de CO2 y neumoperitoneo: En

laparoscopia, el espacio de trabajo quirúrgico se crea al introducir CO2 en la cavidad abdominal. Hay dos posibles técnicas para lograr el neumoperitoneo: la técnica cerrada con el uso de la aguja de Veress y la abierta o técnica Hasson. Por lo general, la presión intraabdominal de trabajo es de 8 – 10mmHg. Sin embargo, puede reducirse a 6 mmHg en animales pequeños o cuando lo demanden las condiciones fisiológicas del paciente.

Ovariectomía laparoscópica: En ginecología humana, la ovariectomía fue uno de los primeros campos quirúrgicos donde la laparoscopia se aceptó ampliamente como enfoque terapéutico. En cuanto a los caninos, la ovariectomía es una de las aplicaciones clínicas más frecuentes dentro de la cirugía laparoscópica. Desde 1985 se ha informado de diferentes técnicas ginecológicas laparoscópicas, incluyendo ovariectomía laparoscópica, ovariohisterectomía laparoscópica y ovariohisterectomía asistida por laparoscopia y, de manera más reciente, ovariohisterectomía asistida por laparoscopia para el tratamiento de la piometra canina. El enfoque laparoscópico para la esterilización de perras sigue, básicamente, los mismos pasos practicados en la cirugía convencional, con la ventaja agregada de ser un procedimiento mínimamente invasivo. En años pasados, se han practicado y documentado diferentes estrategias con éxito, tal como variación en la cantidad de trocares de acceso; por ejemplo, algunos investigadores emplean ya sea tres, dos o un solo trocar de acceso.

Criptorquidectomía laparoscópica: El examen laparoscópico de la cavidad peritoneal puede ayudar tanto en el diagnóstico como en el tratamiento del criptorquidismo por medio de técnicas laparoscópicas o asistidas por laparoscopia. La cirugía laparoscópica aporta ventajas claras sobre la cirugía convencional, ya que permite la localización fácil del testículo abdominal retenido, menor traumatismo, reduce el tiempo quirúrgico y mejora le recuperación del animal.

Remanente ovárico: Ocurre como resultado de dejar restos de tejido ovárico en el abdomen o presencia de tejido ovárico ectópico. Se desarrollan signos clínicos típico del proestro o estro tales como hinchazón de la vulva, sangramiento o cambios conductuales.

El abordaje laparoscópico es una excelente opción quirúrgica para el síndrome de ovario remanente. Debido a todas las ventajas mencionadas anteriormente, mínima invasión quirúrgica y una excelente visualización de los tejidos.

Complicaciones: Las complicaciones intraoperatorias descritas en medicina veterinaria varían del 2 al 35%. Estos incluyen perforación y laceración de vísceras, hemorragias y enfisema subcutáneo. Asimismo, durante el tiempo postoperatorio se han observado ceromas en la zona de entrada del trocar. Vale la pena comentar que gran parte de estas complicaciones inherentes en laparoscópica se relacionan con la inexperiencia del cirujano y de su equipo, con una incidencia mayor durante las fases iniciales de la curva de aprendizaje.

Las tasas de conversión a laparotomía informadas son de 7-21%. Las complicaciones anestésicas relacionadas con el neumoperitoneo por CO2, tales como anemia e hipotensión, también se han comunicado y se ha observado un aumento importante en la presentación de complicaciones en pacientes felinos de edad avanzada o bajos de peso.

Nuevos abordajes quirúrgicos laparoscópicos y endoscópicos: La cirugía laparoendoscópica de sitio único (LESS) y la cirugía endoscópica transluminal de orificios naturales (NOTES) son enfoques novedosos de cirugía mínimamente invasiva.

LESS: Se realiza con un dispositivo único y es un ejemplo de la evolución laparoscópica, permitiendo reducir la morbilidad y mejorando la recuperación postoperatoria. La principal desventaja para el cirujano es la pérdida de la triangulación y el choque continuo con los instrumentos. Existen publicaciones recientes que demuestran su viabilidad en perros y gatos.

NOTES: Este enfoque novedoso combina aspectos de laparoscopia y endoscopia flexible, cuyo propósito final es la ausencia de cicatrices en la piel del paciente. La cirugía NOTES comprende varios enfoques quirúrgicos: transgástrico, transvaginal y transcolónico entre otros. Las limitaciones de esta técnica incluyen sujeción inadecuada, triangulación imprecisa y probable riesgo de infección. En cirugía de NOTES veterinaria existen algunas investigaciones experimentales y unos cuantos reportes de casos, sin embargo, su aplicabilidad todavía está en duda.

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081 / #78

FLUID THERAPY AND FLUID BALANCE IN THE CRITICALLY-ILL

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ABSTRACT BODY:

Despite the recognized benefits of fluid therapy in emergency and critically-ill patients, evidence-based medicine is rarely applied and often the fluid choice and volume of fluids administered are inappropriate. In most cases, such inadequate fluid regimens may not lead to overt harmful effects because of the ability of the kidneys and cardiovascular system to compensate to the excessive volume or supraphysiological loads of electrolytes delivered to the renal tubules. On the other hand, inadequate fluid therapy prescriptions may either lead to delayed fluid resuscitation during shock states and exacerbation of tissue ischemia, or excessive accumulation of total body water (TBW) leading to volume overload and tissue edema extending cardiovascular, respiratory, and renal dysfunction in the critically ill.

Stages of fluid therapy:

Based on the hemodynamic variability of fluid balance during the acute phase of critical illness, IV fluid therapy prescriptions are not static and should evolve based on the cardiovascular evaluation of the patient. Over the past decade, a conceptual framework that defines 4 stages of fluid therapy has been defined to better guide clinicians during the decision-making process of fluid prescription in patients with dynamic fluid changes. The 4 stages have been defined as:

Rescue stage for life-threatening conditions such as shock secondary to hypovolemia or vasodilation, leading to tissue hypoperfusion and ischemia. This stage of fluid therapy is commonly applied during the first 30 minutes to first 2 hours of presentation to the hospital when the patient is at risk of death if aggressive fluid resuscitation does not partially or completely restore organ perfusion. Over-resuscitation can be harmful, and goal directed therapy can be instituted to prevent organ dysfunction from persistent ischemia due to decreased effective circulating volume.

Optimization stage during the following 6 to 8 hours after the rescue phase, once the patient is no longer at imminent risk of death, but the patient requires ongoing fluid therapy to restore tissue perfusion, mitigate fluid losses due to vomiting, diarrhea or polyuria. This phase also applies to patients presenting to the hospital with signs of dehydration.

Stabilization stage of fluid therapy is aimed at providing organ support while preventing iatrogenic complications such as fluid overload, renal vasoconstriction and decreased GFR secondary to supraphysiological loads of chloride delivered to the renal tubular lumen, hypertension and fluid extravasation. In this stage, clinicians should consider using maintenance or hypotonic fluids, which will decrease supraphysiological doses of electrolytes delivered with replacements (isotonic fluids). The stabilization stage may last days to weeks depending on the underlying condition of the patient and it is here where it becomes imperative to consider incorporation of early enteral nutrition.

De-escalation stage is aimed at recognizing patients who no longer require fluid support and may need fluid removal during their time in hospital. It is common practice to believe that all hospitalized patients should be on IV fluids, but patients that voluntarily drink water and eat, or have feeding tubes placed, and are only having insensible losses may no longer require IV fluid support.

Types of fluids:

Isotonic crystalloids: also called replacement fluids, are electrolyte-containing fluids with a composition like the extracellular fluid composition the patient. These fluids are the most common type of fluid used in veterinary medicine and are of particular importance during the rescue and optimization stages of fluid therapy. These fluids are considered isotonic because they have an osmolality closely resembling that of plasma (290-310 mOsm/L). Most crystalloids contain buffers such as lactate, acetate and/or gluconate, useful for correcting metabolic acidosis commonly encountered is sick and critically ill patients; the only exception is 0.9% NaCl which does not contain a buffer and it is considered an acidifying fluid. Infused crystalloids rapidly distribute into the extracellular fluid and only a small portion (25–30%) of the delivered volume remains in the intravascular space after the first 30 minutes post-infusion. Large doses of chloride-rich containing fluids (0.9% Saline) have recently been shown to increase the risk of acute kidney injury in people. The concentration of chloride in 0.9% saline solution is almost one and one-half times that of other balanced crystalloids.

Hypotonic crystalloids: also called maintenance fluids, are fluids with lower osmolality compared to that of plasma and with lower electrolyte concentration

need to maintain body homeostasis. They are used substitute water and electrolytes lost through respiration, urination, defecation, and other insensible losses. These insensible losses are low in sodium and high in potassium. It is extremely important to realize that maintenance/hypotonic fluids may be dangerous to bolus or administer at high rates, as the water concentration gradient between the intravascular and intracellular compartments may lead to a rapid shift of water into the red blood cell and cause cell lysis. Dextrose is often added to maintenance fluids to supply calories to the patient and increases the osmolality of the fluid, preventing rapid shifts of water in case of an accidental rapid administration of the fluid.

Hypertonic crystalloids: are crystalloids that cause a rapid and transient osmotic shift of water from the extravascular to the intravascular compartment leading to a transient volume expansion. The use of hypertonic saline is common nowadays in emergency settings for the acute management of specific conditions such as severe hypovolemia from acute hemorrhage and increased intracranial pressure from traumatic brain injury. Hypertonic saline decreases blood viscosity which results in a positive rheological effect and enhances microcirculatory perfusion. Hypertonic saline also increases preload and causes a mild vasodilation that helps to increase cardiac output in critically ill animals. The dose of hypertonic saline used in small animals range from 2-5 mL/kg, over 5–10 minutes. This dose can be repeated, but particular attention should be given to serum electrolyte concentrations.

Synthetic colloids: are suspensions containing large molecules (molecular weight > 10,000 daltons) dissolved typically in 0.9% NaCl. Hydroxyethyl starches, dextrans, and gelatins are examples of synthetic colloids. These large molecules do not readily filer across the vascular endothelium and remain in the intravascular space for longer periods of time, when compared to crystalloid solutions. Once these particles are filtered through the glomerulus, they are reabsorbed by the proximal tubular epithelium and cause renal tubular vacuolization, damaging this part of the nephron. The volumes of synthetic colloids administered to critically ill dogs has been correlated with the damage of the proximal tubular epithelium in proportional magnitudes. Despite the promising benefits with the use of colloids, the revision of the Starling's forces, along with the effect of the endothelial glycocalyx in vascular permeability, have shown no benefits of colloid therapy in critically ill patients.

Monitoring fluid balance:

In the last decade, special attention has been paid to the monitoring of fluid balance in hospitalized and critically ill patients, as fluid overload has been directly correlated with increased risk of mortality and development of comorbidities in people and veterinary patients. Fluid overload is defined as an increase of 10% or more from basal or ideal body weight. Liberal fluid therapy is commonly practiced in human and veterinary hospitals with fluids rates at two-, three- or four-times maintenance rates are prescribed to patients without adequate therapeutic goals and monitoring endpoints. High fluid rates lead to increase central venous pressure, increased myocardial atrial stretch and release of atrial natriuretic peptides which cause endothelial dysfunction, increase vascular permeability, and decreased lymphatic flow, leading to water accumulation in the organ parenchyma (kidneys, heart, liver and GI tract). The percentage of fluid overload (%FO) can be calculated by the following formula:

%SF = [(VtotIN – VtotOUT) / basal body weight] X 100

One of the easiest and least invasive ways of monitoring fluid balance in hospitalized patients is by frequent and consistent body weight assessment. In veterinary medicine monitoring body weight is easy, especially in small dogs and cats, and large dogs that are ambulatory. More advanced methods of fluid balance monitoring such as central venous pressure, pulmonary capillary pressure, and arterial pulse pressure variation are not routinely performed in veterinary hospitals. With the widely spread use of ultrasound in veterinary hospital. Point of care ultrasound can be used bedside to the patient to evaluate the presence of fluid accumulation in visceral organs such as development of B-lines in the lungs, or "halo sign" in the gall bladder.

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PRECISION MEDICINE, DTC GENETIC TESTING, AND DO'S AND DON'TS OF GENETIC TESTING

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ABSTRACT BODY:

Precision Medicine, DTC Genetic Testing, & the Do's and Don'ts of Genetic Testing

Topics of the Presentation

How to use Direct to Consumer Testing (DTC)

Pros & Cons / Dos & Don'ts of DTC

What is Precision Medicine & how to use in veterinary medicine

Learning Objectives

Know when to request a genetic test

Know where to get genetic tests

Know how to use the information?

Realize the concerns with genetic tests

The first genetic tests for the domestic cat were developed over 25 years ago. Presently, 167 different mutations affecting 101 genes have been identified, producing interesting phenotypes and traits but also diseases and other health concerns. Current genetic and genomic technologies can be employed to test nearly all the known mutations in one assay (parallel genotyping) using DNA arrays, mass spectroscopy or genotyping-by-sequencing. Unlike in human medicine, where traditionally a physician had to request a genetic test, the owner of a pet can directly purchase genetic tests from a company, i.e., direct – to – consumer testing (DTC). DTC of large panels of DNA variants developed for humans and are now easily available for cats and dogs as DTC tests. Thus, no longer are genetic tests only available from the researcher who identified the mutations or a laboratory associated with a veterinary / research facility. Private companies now offer genetic tests at lower costs by using the massively parallel genotyping, however, provide little genetic counseling and education to the owner, breeder or veterinarian. These large panel tests generally do not have documented sensitivities and specificities for the mutation being tested.

How to use Direct to Consumer Testing (DTC)

Veterinarians should consider using genetic testing, either submitted by the veterinarian or by the client, to support clinical diagnoses. Note that genetic tests are only one aspect of a diagnosis and only a part of the overall healthcare of the patient. Hopefully, the genetic test may lead to a more appropriate treatment plan and prognosis. Genetic testing should be considered once other environmental factors, including nutrition, toxic and viral exposures have been considered. A genetic test will not be useful if the condition is not genetic. Most genetic testing is limited to Mendelian traits, i.e., traits with simple autosomal or X-linked dominant or recessive modes of inheritance. Environment can influence the presentation, including age-at-onset and severity of genetic conditions. If a condition is genetic, likely a veterinary specialist may need to be consulted.

Once a tentative diagnosis has been established and a genetic test may be supportive, use the Online Mendelian Inheritance in Animals (//OMIA.org) to see if this condition has been previously documented and if a genetic test is available. Provided below is a table of the genetic tests for disease variants in domestic cat breeds in which many companies provide genetic testing, including research universities. But also, several genetic service laboratories provide genetic tests for DNA variants that were originally identified in a random bred cat. The rediscovery of these variants is extremely rare but can occur. The OMIA website lists all the DNA variants that are associated with diseases in companion animals. This site also collates the scientific information and publications associated with the disease and will list the year of the variant discovery as well as the researchers involved. Researchers are often keen to be of assistance and realize the genetic implications may not be familiar to the veterinarian. Thus, don't hesitate to ask the researcher first for advice on genetic testing as they may perform the testing within their own laboratory! The published manuscripts often have the email address for the corresponding author of the publications. Otherwise, preferably use a DTC laboratory associated with a research university, which will be invested in the science, provide the best education, genetic counseling and customer service.

Pros & Cons / Dos & Don'ts of DTC

Large panel DTC tests allow an effective screening method for many diseases and traits.

No one technology can accurately genotype all the various different types of mutations that cause diseases, thus, look for services that can use a second method, or use a secondary service, which are generally associated with research universities.

Many tests are not valid in different breeds, which is not generally divulged by some services. Thus, conversations with the researcher who made the discovery will help clarify the risks.

While some services work towards scientific standardizations, others are more profit oriented. WSAVA and other groups are working towards standards that will help clarify genetic testing results. The WSAVA Hereditary Disease Committee can be contacted for assistance.

Don't make health & neutering decisions based on results from large panel DCT testing. Request a single gene test for a second opinion. Use the overall health indications of an animal.

Precision / Genomic (P4) Medicine

- is an emerging approach for disease treatment & prevention that takes into account individual variability in genes, environment, and lifestyle. Precision medicine is used to understand the complex mechanisms underlying a patient's health, disease, or condition, and to better predict which treatments will be most effective – not the average or average for an ethnic group!

DNA testing is a form of Precision Medicine. Large panel genetic testing is an improvement and further facilitates the use of genetics for diagnoses and potentially treatment of health concerns. Whole exome and whole genome sequencing of a patient is a larger scale effort to even further advance precision medicine. Genome and exome sequencing projects are available for cats and dogs and these techniques are becoming common place for the identification of mutations that cause heritable diseases and traits. Generally, if a test is negative for genetic test, the patient may have a novel genetic cause for their presentation. WGS and WES help to find novel causes. Genetic testing only confirms the presence or absence of an already known DNA mutation for a disease or trait. Precision Medicine hence incorporates discovery, which hopefully will lead to diagnosis and more precise treatment. Only a few research laboratories are equipped to conduct WGS and WES sequencing for companion animals on a Precision Medicine basis. The University of Missouri, laboratories of Dr. Gary Johnson and Dr. Leslie Lyons (99 Lives Cat Genome Sequencing Consortium), will attempt to discover new variants for dogs and cats, respectively. Many of the researchers involved with DNA discoveries should also be able to suggest contacts for WGS / WES.

Common inherited diseases in domestic cat breeds.

‡ Mode of inheritance of the variant allele (disease-causing allele). *The breed affected represents the breed in which the DNA variant was identified. Other breeds may be affected if they have common ancestry, especially other breeds in a breed family. In reference to the variant allele, AD implies autosomal dominant, AR implies autosomal recessive, co-D implies co-dominant, X implies X-linked. OMIA: Online Mendelian Inheritance in Animals (http://omia.org) entries provides links to citations and clinical descriptions of the phenotypes and the diseases.

Disease / Trait (alleles) OMIA Entry (-9685)	**MOI‡**	**Phenotype (Breeds affected)***	**Gene**
Acrodermatitis Enteropathica 000593	AR	Turkish Van	SLC39A4
ALPS - Autoimmune lymphoproliferative Disease 002064	AR	non-neoplastic lymphoproliferative disease (British Shorthair)	*FASL*
Centronuclear myopathy 001508	X	Neurological (Maine coon)	*MTM1*
Chediak-Higashi Syndrome 000185	AR	Storage disorder (Persian)	*LYST*
Craniofacial Defect 001551	AR	Craniofacial Defect (Burmese)	*ALX1*
Cystinuria 002023	AR	(MCC, Sphynx, Siamese)	*SLC7A9*
Ehlers-Danlos 002165	AR	Stretchy skin (Bengal, Bombay)	*COL5A1*
Factor XI Deficiency 000363	AR	Bleeding disorder (Maine Coon)	*F11*

Disease / Trait (alleles) OMIA Entry (-9685)	MOI‡	Phenotype (Breeds affected)*	Gene
Gangliosidosis 1 000402	AR	Lipid storage disorder (GM1) (Korat, Siamese, S.E. Asia)	*GLB1*
Gangliosidosis 2 01462	AR	Lipid storage disorder (GM2) (Burmese)	*HEXB*
Gangliosidosis 2 01462	AR	Lipid storage disorder (GM2) (Korat)	*HEXB*
Glycogen Storage Dis. IV 000420	AR	Glycogen storage disorder (GSD) (Norwegian Forest Cat)	*GBE1*
Hermansky-Pudlak syndrome-5 002161	AR	Ocular albinism (Donskoy)	*HPS5*
Holoprosencephaly 002366	AR	Forebrain malformation (Toyger)	*GDF7*
HCM 002316	AD	Cardiac disease (HCM) (Sphynx))	*ALMS1*
HCM 000515	AD	Cardiac disease (HCM) (Maine Coon, Ragdoll)	*MYBPC*
Hypokalemia 001759	AR	Potassium deficiency (HK) (Burmese)	*WNK4*
Hypotrichosis 001949	AR	Hairless, early death (Birman)	*FOXN1*
Hypothyroidism 000536	AR	Small size, low T4 (British shorthair)	*TPO*
Progressive Retinal Atropy 001244	AR	Late onset blindness (rdAC) (Abyssinian)	*CEP290*
Progressive Retinal Atropy 000881	AR	Rod-cone dysplasia (Abyssinian)	*CRX*
Progressive Retinal Atropy 001613	AR	Mid onset blindness (Bengal)	*KIF3B*
Progressive Retinal Atropy 001222	AR	Early onset blindness (Persian)	*AIPL1*
Polycystic Kidney Disease 000807	AD	Kidney cysts (PKD) (Persian)	*PKD1*
Polycystic Kidney Disease 2 002525	AR?	Polycystic kidney disease (Siberian)	*PKD2*
Pyruvate Kinase Deficiency 000844	AR	Hemopathy (PK Deficiency) (Abyssinian, Siberian, MCC)	*PKLR*
Skeletal dysplasia 002485	AR	Skeletal dysplasia (British)	*LTBP3*
Spasticity 001621	AR	Congenital myasthenic syndrome (CMS) (Devon Rex)	*COLQ*
Spinal Muscular Atrophy 000939	AR	Muscular atrophy (SMA) (Maine Coon)	*LIX1-LNPEP*

083 / #182

STATE-OF-THE-ART LECTURE: HOW SMALL INTESTINAL ULTRASOUND CORRELATES WITH HISTOPATHOLOGY IN DOGS AND CATS

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ABSTRACT BODY:

Introduction

Radiography allows an overview of the abdomen while ultrasonography does not. A shortcut to ultrasound in a vomiting animal could lead to misdiagnosis of a disease that could be readily identified radiographically. Therefore, radiography and ultrasonography should be considered complementary diagnostic tests. Barium studies still have importance in assessing animals with chronic gastrointestinal obstructions.

Take home message of this lecture:

A three-view radiographic procedure is advised in every vomiting dog, 2-view in cats

Right and left lateral and a VD image for dogs

Left lateral places gas in the pylorus and duodenum to find foreign material

Wooden spoon compression

Compress the bowel with a wooden spoon to separate loops of jejunum from one another so that foreign material, linear foreign bodies, and corrugations can be detected without superimposition

Repeat radiographs after a few hours to one day can be valuable for determining how suspect dilated intestines or content evolves with time if the animal is stable.

Pneumocolonogram: differentiates gas-filled colon from gas-filled jejunal segments and duodenum

Barium studies and ultrasound should only be performed after 1 and 2 are performed

Practical approach

Assess each radiographic study to determine if 1) a mixed population of jejunal segments is present, 2) if gastric dilation alone is present, or if 3) foreign material is present. The next step is to compare to the presenting signs of acute vs. chronic vomiting and the clinical condition of the animal. If clear evidence of mechanical ileus is not present (no mixed population), then consider a wooden spoon compression study, pneumocolonogram if the large bowel cannot be distinguished from jejunum, or i.v fluid therapy and repeat radiographs. In chronic disease with equivocal radiographic findings, a barium study can also be performed.

Ileus

Ileus is a failure of intestinal contents to be transported and is recognized radiographically by the presence of dilated bowel segments. Survey abdominal radiographs should always be performed in vomiting animals with vomiting to assess for the presence of ileus. Ultrasound alone in such instances does not allow a global view of the abdomen, is much more time-consuming and non-gastrointestinal causes of the dog's clinical signs as well as any secondary abnormalities may be overlooked. The radiographic appearance of ileus is dependent on its duration, location, and degree of obstruction. Acute or very proximal obstructions may show little intestinal dilation radiographically whereas chronic or more distally located ones will show more generalized dilation of the small intestines. The two major types of ileus are obstructive (mechanical) and functional. Obstructive ileus may be partial or complete.

Partial Obstructions

Dogs and cats with partial obstructions tend to have a more chronic course of intermittent vomiting and diarrhea. Common causes include foreign bodies and strictures. Fasted (>12 hours) or anorectic animals should not have small bowel segments containing granular material resembling that of food content radiographically. Granular or more opaque small bowel contents may be detected in partial obstructions. The intestines in such cases may be mildly dilated (1-1.5 times the width of the second lumbar vertebral body) proximal to the obstruction or may even be of normal diameter. Because partial obstructions may be more difficult to diagnose radiographically than complete obstructions, complementary imaging procedures such as barium studies or ultrasound are often necessary for the diagnosis. Repeat radiographic examination has great diagnostic value when abnormal intestinal content is identified. If granular content is seen in the small intestine of an animal that is vomiting or anorectic for more than 24 hours, obstruction should be considered likely. If the same finding of focal granular luminal content is identified on follow-up radiographs, even if intestinal dilation is not evident, in a vomiting animal, the chances of obstruction are high and sonography or exploratory surgery is indicated. Ultrasonography is highly recommended in older animals to screen for intestinal masses prior to exploratory laparotomy.

Complete Obstructions

More severe dilation, usually with air, is seen in complete obstructions. The location of the obstruction can be either intraluminal (foreign bodies), extraluminal (adhesions, herniation, intussusceptions), or intramural (neoplastic wall infiltrations, granulomas). Dilation (1.5-2 times the width of the body of L2) is seen proximal to the site of obstruction and the segments distal to it usually appear empty and contracted. Due to this, the jejunal segments appear to have many varied diameters, some very dilated, others empty or small. This is called a "mixed population". This is due to the continued peristaltic activity in the distal segments. The dilated segments are often referred to as "sentinel loops". Proximal duodenal or pyloric obstructions may show no radiographic abnormalities. 24 hours following a gastric outflow obstruction, the animal has vomited out the intestinal contents, and the intestinal content moves to the colon. Abdominal radiographs may show no abnormalities or gastric distention. Moreover, the entire gastrointestinal tract may actually appear completely empty after some hours due to recurrent vomiting. The most common difficulty in diagnosing complete obstructions is in trying to differentiate small from large bowel when the colon is dilated and especially gas-filled. It is recommended in such instances to perform a pneumocolonogram or small volume barium enema in order to identify the colon and distinguish it from the small intestinal segments that may or may not be dilated. When the abdomen appears normal radiographically in a vomiting animal, a compression study, wait and repeat study, or either a barium study or an ultrasound examination should be the next diagnostic procedure.

Functional Ileus

Another form of ileus that can be detected is a generalized and uniform mild intestinal dilation due to lack of peristaltic activity. This is known as adynamic, functional or paralytic ileus and results from an inhibition of bowel motility. Functional ileus results in obstruction since the intestinal contents pool in the dependent areas of the gastrointestinal tract. Radiographically the gastrointestinal tract appears mildly dilated, can have a mixed content with some gas- and some fluid-filled intestines and colon have generalized fluid or gas filling. The distribution of the intestines in the abdomen is regular. Gas is often present in the stomach. Typically, granular ingesta in the stomach and bowel is not identified. The intestines appear to have a uniform diameter. Animals with this pattern typically have clinical signs of both vomiting and diarrhea. Such an adynamic intestinal pattern can be due to the administration of pharmaceutical agents such as parasympatholytics and sedatives. Other causes are peritonitis, blunt abdominal trauma, electrolyte imbalance, and enteritis of various causes.

Complicated ileus

Complicated forms of ileus include bowel perforation with peritonitis, free air in the abdominal cavity, bowel ischemia due to thromboembolism, intussusception, or volvulus at the root of the mesentery. Linear foreign bodies can also lead to a complicated form of ileus. The presence of pneumoperitoneum together with abdominal effusion on an abdominal radiograph should alert the clinician that bowel perforation has occurred. The detection of free intraabdominal air may require the use of ventrodorsal horizontal beam radiography with the patient in left lateral recumbency. Free air can be detected just under the right abdominal wall and lateral to the duodenum. Volvulus or mesenteric thromboembolism is recognized by the presence of generalized, severely dilated and air-filled jejunal segments. Linear foreign bodies produce characteristic changes on abdominal radiographs in both cats and dogs. The small intestinal loops appear convoluted and gathered or clumped together at one site, usually in the mid-right abdomen and intraluminal gas bubbles appear asymmetrical and irregularly shaped.

Gastric outflow obstructions

Obstruction of the gastric outflow is typically located at the antrum, pylorus, or duodenum. After a period of 24 hours, the stomach will either be empty due to vomiting or be moderate to severely dilated while the jejunum is devoid of content, appearing normal radiographically. The colon may or may not contain feces. This pattern of dilated stomach and empty jejunum in a vomiting animal should raise suspicion of gastric outflow obstruction. If this pattern is present on repeat radiographs after a few hours to one day, then it is much more likely. Ultrasonography can be used to assess the duodenum to confirm the presence of an obstruction. Barium studies can be used similarly.

Cause of intestinal obstructions

It is important to remember that obstruction can be due to disease in three locations: 1)luminal, 2) mural and 3) extramural compression. Any of these three can be the cause of a mechanical ileus and differentiating them is very difficult on survey radiographs. This may not matter in some instances as exploratory surgery will commonly be required in the face of a mechanical obstruction regardless of the cause. Ultrasonography is more sensitive for differentiating the three causes and for that reason can be considered prior to surgery. Mural lesions may be due to both fungal and neoplastic infiltrative disease. Extramural compression can be due to abdominal masses such as at the pancreas or mesenteric masses.

084 / #79

DIAGNÓSTICO Y MANEJO DEL GATO CON EFUSIÓN PLEURAL

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ABSTRACT BODY:

La efusión pleural o el neumotórax pueden provocar distrés respiratorio por compresión del pulmón y disminución de la capacidad de expansión. En el espacio pleural se puede acumular aire, trasudados puros o modificados, exudados o tejidos sólidos, como en neoplasias, que disminuyen la capacidad de expansión de los pulmones y de ventilación. La efusión pleural no es un diagnóstico final y puede aparecer por diferentes causas, por lo que deberemos desarrollar el diagnóstico diferencial para poder mejorar la condición clínica del gato.

El patrón de respiración se ha descrito como de tipo **restrictivo**, con respiraciones rápidas y superficiales, aunque es más característico el **movimiento paradójico** de la pared del tórax. La observación de este tipo de respiración en DP tiene una sensibilidad del 90% y especificidad del 58%, dato muy importante para el manejo plantearnos realizar ecografía torácica (TFAST), radiografía o toracocentesis diagnóstica, que veremos más tarde.

Si además de la observación del movimiento paradójico en la auscultación pulmonar encontramos ruidos cardiacos y pulmonares amortiguados la sensibilidad en el diagnostico de DP aumenta al 99%.

Va a ser muy importante la observación de la **frecuencia respiratoria y patrón respiratorio**:

-Patrón restrictivo: respiración rápida, superficial Ej: enfermedad de parénquima pulmonar y enfermedad del espacio pleural.

-Patrón obstructivo: respiración lenta, profunda. Ej: enfermedad crónica broncopulmonar, parálisis laríngea.

PLAN DE ACCIÓN ANTE UN GATO CON SOSPECHA DE DERRAME PLEURAL

El derrame pleural puede provocar distrés respiratorio por compresión del pulmón y disminución de la capacidad de expansión. En el espacio pleural se puede acumular aire, trasudados puros o modificados, exudados o tejidos sólidos, como en neoplasias, que disminuyen la capacidad de expansión de los pulmones y de ventilación. El derrame pleural no es un diagnóstico final y puede aparecer por diferentes causas, por lo que deberemos desarrollar el diagnóstico diferencial para poder mejorar la condición clínica del gato.

La cavidad pleural en gatos normales contiene aproximadamente 2 ml de fluido. El fluido pleural es seroso y con poca concentración de proteínas.

La acumulación de fluido en el espacio pleural se produce por:

-Aumento de la presión hidrostática.

-Disminución de la presión oncótica, como en casos de hipoalbuminemia.

-Aumento de la permeabilidad capilar, como en casos de inflamación o de neoplasia.

Después de la evaluación inicial, historia y signos clínicos, localización anatómica del distrés respiratorio y lista de diagnósticos diferenciales hay que proceder al manejo terapéutico. Debe ser extremadamente cuidadoso porque la manipulación de estos pacientes con poca capacidad pulmonar es muy complicada.

Ante un gato con distrés respiratorio se debe tener en cuenta los siguientes pasos:

- Administración de oxígeno
- Sedación
- Control de la temperatura: pueden llegar en hipotermia por la situación de shock o en hipertermia por la taquipnea o en caso de procesos infecciosos
- Toracocentesis /colocación de tubo de toracocentesis: con fin diagnóstico y terapéutico
- Monitorización de la oxigenación (pulsioximetría, gasometría)
- Radiografía torácica, ecografía o ecocardiografía. Las pruebas de imagen nos pueden ayudar a detectar la causa específica de proceder a su manejo. Es conveniente tener desarrollados protocolos de ecografías de urgencia (TFAST) para obtener la máxima información en el mínimo tiempo de la forma más segura posible para el gato.
- Evaluación laboratorial del liquido obtenido
- Tratamiento de la causa

Se coloca en posición esternal, se rasura la zona y se lava con antiséptico como clorhexidina de forma quirúrgica. Se puede usar una palomilla de 21 o 23 gauge con llave de 3 vías y jeringa de 20 ml. La toracocentésis se puede realizar entre el sexto, séptimo u octavo espacio intercostal a nivel de la unión costocondral, pero evitando el margen caudal de las costillas por donde transcurre el paquete

vasculonervioso. Si sospechamos de neumotórax se elegirán zonas más dorsales del tórax.

Otro de los procedimientos que en ocasiones tenemos que realizar para estabilizar al gato es la colocación de un tubo de drenaje pleural.

¿ Cuándo está indicado colocar un tubo de drenaje pleural?

- Si persisten los signos clínicos

2.Si tenemos que realizar más de 2 toracocentesis al día

3.Si tenemos que hacer toracocentesis durante más de 2 días

También está indicada la colocación de tubo de drenaje pleural en las situaciones en las que se determine que el animal tiene un neumotórax a tensión, piotórax, postoperatorios de cirugías torácicas o heridas torácicas penetrantes.

ANÁLISIS DEL FLUIDO del DERRAME PLEURAL

El derrame pleural en medicina veterinaria se clasifican en base al contenido en proteínas, densidad, cantidad y tipo celular. Cada esquema de clasificación tiene diferentes valores de corte, además no hay evidencia científica sobre la utilidad de su uso. Cuando obtenemos fluido pleural introduciremos parte en EDTA y reservaremos muestra para enviar a estudio microbiológico.

De la muestra en EDTA realizaremos:

- Citología y recuento celular.

- Determinación de proteínas totales con refractómetro.

- Determinación de Lactato Deshidrogenasa (LDH), test de Rivalta u otras

pruebas específicas dependiendo de la patología de la que sospechemos.

Realizaremos colesterol y triglicéridos en caso de sospecha de quilotórax pero es una patología menos frecuente que otras en caso de derrame pleural

- TRANSUDADO PURO

Concentración de proteínas < 2,5 g/dl.

Células nucleadas < 1000/L

Tipo de células mayoritaria= monoclucleares.

Relacionado con hipoalbuminemia y estadios iniciales de fallo cardiaco congestivo, especialmente en los pacientes con fluidoterápia excesiva.

- TRANSUDADO MODIFICADO

Concentración de proteínas < 3,5 g/dL.

Células nucleadas < 5000/L

Tipo de células mayoritaria=aumenta el número de neutrófilos no degenerados.

Se clasifican como transudados modificados a los que basándonos en la cantidad de proteínas y en la celularidad serían exudados pero se han formado por aumento de la presión hidrostática. Se acumula fluido, irrita al mesotelio produciéndose exfoliación de células mesoteliales e inflamación secundaria lo que produce aumento de células mesoteliales y neutrófilos no degenerados. Es difícil diferenciar células mesoteliales reactivas de células neoplásicas.

Se puede producir exudado modificado por:

Aumento de presión hidrostática: fallo cardiaco congestivo.

Disminución de la presión oncótica: hipoalbuminemia.

Obstrucción linfática: neoplasia, hernia diafragmática.

- EXUDADO

Concentración de proteínas > 3 g/dl- 3,5 g/dL

Células nucleadas > 5000/L

Tipo de células= células mononucleares y neutrófilos.

Los exudados pueden clasificarse como séptica o no séptica.

Exudado no séptico: neutrófilos no degenerativos sin evidencia de organismos:

- PIF
- Neoplasia
- Hernia diafragmática crónica
- Torsión pulmonar
- Pancreatitis
- Exudado séptico en resolución

Exudado séptico: cantidad alta de células nucleadas, las células predominantes son los neutrófilos degenerados y presencia de bacterias.

QUILO

La concentración de proteínas es entre 2 – 6 g/dL, recuento celular de entre 400-10000/l, las principales células son los linfocitos pequeños en estadios tempranos y neutrófilos no degenerativos y macrófagos aumentan cuando el proceso es más crónico. La efusión tiene un aspecto lecho por su alto contenido en triglicéridos. La turbidez desaparece cuando se añade éter. Después de centrifugarlo o de refrigerarlo se forma un anillo blanquecino en la parte superior del tubo. La concentración de

triglicéridos es superior a la del suero y la concentración de colesterol es menor que la del suero. El ratio colesterol: triglicéridos es menor de 1.

Causas:

- Idiopático
- Congénito
- Traumático
- Neoplasia

- Cardiomiopatía
- Hernia diafragmática.

HEMORRAGIA

Causas:

- Trauma
- Coagulopatía
- Neoplasia
- Torsión de lóbulo pulmonar

PUNTOS IMPORTANTES:

- La observación y la auscultación de los pacientes con disnea por derrame pleural (DP) normalmente es suficiente para localizar el problema, la radiología y la ecografía TFAST ayudan a confirmar la presencia de derrame pleural.
- La diferenciación entre enfermedad respiratoria de tracto inferior o enfermedad de cavidad torácica no siempre es fácil; sin embargo , es esencial para manejar al paciente porque el tratamiento es diferente.
- La estabilización y monitorización del paciente son prioritarias.
- El fallo cardiaco congestivo , peritonitis infecciosa felina , neoplasia y piotórax son las cuatro causas más frecuentes de DP.
- Se debe observar los signos clínicos, analizar la historia y el examen físico debe realizarse cuidadosamente y siendo mínimamente invasivos.
- La realización de toracocentésis ayuda para estabilizar al paciente y para obtener datos que ayudarán en enfocar el diagnóstico.

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085 / #80

DERMATITIS EOSINOFÍLICA FELINA

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ABSTRACT BODY:

A efectos de esta charla vamos a definir dermatitis eosinofílicas como procesos inflamatorios cutáneos, causados por reacciones de hipersensibilidad en los que el eosinófilo es la principal célula efectora, la que causa daño tisular y los principales signos clínicos.

En el gato, se han definido varias entidades clínicas bajo este grupo:

- Las úlceras indolentes.
- Las placas eosinofílicas.
- Los granulomas colagenolíticos (lineales o no).
- La hipersensibilidad a la picadura de mosquitos (dípteros).

Las tres primeras entidades generalmente se agrupan bajo en nombre genérico de "complejo del granuloma eosinofílico felino", pues comparten bastantes características clínicas, clínico-patológicas y, sobre todo, histopatológicas.

El complejo del granuloma eosinofílico (CGE)

La causa de la aparición de estas lesiones no se conoce en detalle. Se considera que se trata de la manifestación de una reacción de hipersensibilidad en gatos con una especial predisposición genética a desarrollar reacciones eosinofílicas. En muchos casos se identifica o sospecha un síndrome atópico felino, una reacción adversa a alimentos o una alergia a la picadura de pulga. Sin embargo, es evidente que no todos los gatos con estas alergias desarrollan lesiones eosinofílicas y también que, en muchos casos, no se identifica ninguna causa (CGE idiopático). La importancia del componente genético se pone de manifiesto, por el hecho de que se han desarrollado colonias de gatos que de forma espontánea desarrollan lesiones de CGE.

El cuadro clínico de las tres formas es bastante característico:

Forma clínica	Características clínicas	Principales diagnósticos diferenciales
Ulcera indolente	Úlcera bien delimitada cubierta por exudado amarillo situada en labio superior . Uni o bilateral.	Neoplasia (carcinoma de células escamosas). Criptococosis.
Placas eosinofílicas	Placas eritematosas exudativas en abdomen y cara interna. De muslos.	Neoplasia, dermatofitosis, infecciones virales (herpes, calicivirus, cowpox).
Granuloma colagenolítico lineal	Placa o nódulo eritematoso y amarillento en la superficie, cubierto por costras. Mentón, lengua, manos.	Neoplasia, dermatofitosis – otras micosis profundas, infecciones virales (herpes, calicivirus, cowpox). Micobacteriosis y otras infecciones bacterianas.
Granulomas colagenolíticos no lineales	Lesión alopécica y eritematosa, firme, lineal, en cara posterior de los muslos.	

Las presentaciones clínicas, en la mayoría de casos, son muy características y se puede establecer un diagnóstico clínico. Después, es preciso determinar la causa desencadenante. El protocolo de diagnóstico, de forma resumida, podemos decir que es el mismo que se sigue en el caso de un gato con prurito. En concreto:

- Descartar una ectoparasitosis. Iniciar tratamiento continuado para ectoparásitos (isoxazolina, selamectina).
- Descartar presencia de una infección bacteriana (citología de las lesiones).
- Descartar una reacción adversa a alimentos (dieta de eliminación antigénica durante 8 semanas).
- Valorar la existencia de un síndrome atópico cutáneo felino e iniciar un tratamiento específico para esta enfermedad.

Si en un caso las lesiones no son características o se sospecha de otras entidades (carcinoma de células escamosas, infección fúngica, …), se recomienda tomar biopsias para estudio histopatológico y eventualmente para cultivo.

El tratamiento depende de la causa primaria que ha desencadenado el proceso. Si se identifica una causa, obviamente debe tratarse. Si no se identifica ninguna causa, el tratamiento es muy similar al del síndrome atópico felino. Lo más usual es iniciar un tratamiento con un glucocorticoide (prednisolona, metilprednisolona, dexametasona) con o sin

ciclosporina (ver charla sobre tratamiento del síndrome atópico felino). La eficacia del oclacitinib para el tratamiento del CGE felino en estos momentos se desconoce.

La hipersensibilidad a la picadura de mosquitos

Se trata de una dermatitis estacional, prurítica y poco frecuente desencadenada por la picadura de mosquitos y otros dípteros. El cuadro clínico consiste en la aparición de eritema, pápulas, úlceras y costras en el dorso nasal y en los pabellones auriculares. El diagnóstico es clínico en y el tratamiento consiste en evitar la exposición a las picaduras y administrar glucocorticoides sistémicos (prednisolona, metilprednisolona, dexametasona).

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BUEN PROVECHO! NUTRITIONAL CONSIDERATIONS FOR CAPTIVE REPTILES

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ABSTRACT BODY:

BUEN PROVECHO! NUTRITIONAL CONSIDERATIONS FOR CAPTIVE REPTILES

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Reptiles come in all shapes and sizes and can be herbivorous, omnivorous, or carnivorous, depending on the species. An ideal diet for a reptile in captivity should mimic their natural diet as closely as possible and provide a diversified selection of food. Some herbivorous species will readily eat an omnivorous diet, but eventually these animals will reveal signs of nutritional disorders. Therefore, it is important to remember that food preferences do not always correlate with appropriate nutrition.

Herbivorous species are primarily classified as hindgut fermenters, with microbial fermentation occurring in the large intestine. Consequently, the bulk of the diet of herbivorous species should be plant fiber. The plant fiber offered should be rich in vitamins A and D3 and should have more available calcium than phosphorous. An ideal Ca:P ratio of at least 1.5-2:1 should be present. The diet should also be low in fats, oils, proteins, thiocyanates, and oxylates. Herbivorous species housed outdoors will forage for themselves if provided with an appropriately planted enclosure, but additional food is usually required. It is important to periodically peruse the yard and rule-out the presence of any poisonous plants. A variety of foods should be offered and can be mixed with calcium, iodine, vitamin D3, and vitamin A supplementation. It is important to remember that grocery greens are generally higher in protein, lower in fiber, and may have an inverse calcium: phosphorus ratio when compared to natural forage. Spinach, cabbage, and beet greens should not be fed in excess due to their high oxylate content. Debilitated herbivorous species requiring force-feeding or tube-feedings should be fed a critical care diet designed for their specific needs.

It has been suggested that omnivorous reptiles do best when offered plant and animal matter in proportions that range from 75:25 to 90:10. Dietary requirements in these species tend to change with age, with most juveniles requiring a diet comprised of a higher proportion of animal matter. As the juveniles mature, their dietary requirements shift to a more herbivorous diet. The primary animal proteins offered should mimic a natural diet, including earthworms, slugs, snails, millipedes, pupae, and larvae (mealworms). It is essential to monitor the diets of captive invertebrates in order to avoid nutritional deficiencies in the animals eating them. Offering the invertebrates a diet rich in minerals and vitamins will help to ensure that the prey is “gut-loaded”. Liver and yellow or dark orange colored vegetables (squash, carrots, and sweet potatoes) are excellent sources of vitamin A, and Swiss chard, kale, beet greens, escarole, parsley, watercress, and green beans all have a positive Ca:P ratio.

Carnivorous reptiles are generally the easiest group to provide food for in captivity, as there is a range of invertebrate and vertebrate prey species that can be offered. However, as was mentioned previously, those carnivores that specifically hunt invertebrates do need to have their prey species “gut-loaded”. Most carnivorous aquatic species are piscivorous. If frozen fish are offered, then the diet needs to be supplemented with thiamine, as frozen-thawed fish can produce thiaminases.

Nutritional disorders in reptiles commonly present as a chronic problem, and the diet is often times centered around limited food sources or human convenience. In most cases, deficient diets are comprised of limited numbers of food items and/or are not supplemented with calcium and vitamin powders.

Vitamin A is a critical component in the production and maintenance of epithelial cells, and is intimately associated with several structures related to vision. Hypovitaminosis A is a common clinical entity in reptiles fed predominately vitamin A deficient foods. The most obvious clinical abnormality associated with hypovitaminosis A is squamous metaplasia, which results in the degeneration of epithelial surfaces (e.g., conjunctiva, gingiva, pancreatic ducts, renal tubules, and skin). Due to the multiple epithelial surfaces of the body, squamous metaplasia can manifest itself in several different ways. Blepharospasm, conjunctivitis, blepharoedema, blindness, rhinitis, blepharitis, lower respiratory tract disease (nasal discharge, depression, dyspnea, open-mouth breathing), and/or cutaneous abnormalities may be observed. Middle ear infections

and aural abscesses have also been linked with hypovitaminosis A. The diagnosis of hypovitaminosis A can be met via dietary history, clinical signs, measuring vitamin A concentrations, or histopathology of tissue samples (squamous metaplasia of the epithelia surfaces). Supportive treatment should be utilized concerning the clinical manifestations of vitamin A deficiency, and appropriate husbandry and dietary changes should be instituted. Vitamin A deficiency can be corrected by oral supplementation with vitamin A products, or by offering small amounts of liver once per week. Injectable vitamin A should be used very cautiously, as hypervitaminosis A can occur with a single injection.

Metabolic bone disease (MBD) is defined as any metabolic defect that alters the morphology and functioning of bones. MBD is usually related to low levels of calcium or excessive levels of phosphorus, which consequently bind to calcium and render it physiologically unavailable. Decreased calcium availability results in increased parathyroid activity and mobilization of stored calcium from the shell and bone cortices. Factors predisposing reptiles to the development of MBD include: dietary deficiency of calcium and/or suitable vitamin D3, inappropriate calcium: phosphorus ratio in the diet, lack of exposure to ultraviolet light (ultraviolet B radiation increases activation of vitamin D precursors and facilitates gastrointestinal absorption of calcium), dietary excess of protein during rapid growth periods, anorexia, and abnormal vitamin D3 metabolism secondary to renal, hepatic, intestinal, or parathyroid disease. MBD is commonly observed in rapidly growing juvenile animals and reproductively active females. Clinical signs consistent with MBD vary depending on the age and species of the patient. The most common clinical findings in affected animals include muscle tremors/fasciculations, seizures, soft-shells, pathologic fractures and acute death. A thorough history is required before a diagnosis of MBD can be met. Diagnostically, radiographs and blood work can provide insight into the animal's disease state. Radiography may reveal fibrous osteodystrophy and pathologic fractures. Low blood calcium concentrations are highly suggestive of MBD, but calcium blood concentrations are frequently not low in cases of MBD because of hyperparathyroid activity. It must be remembered that blood calcium concentrations are not reflective of physiologically available levels of calcium. Ionized levels of calcium are more indicative of the availability of calcium, but, unfortunately, published reference levels are difficult to find in the literature. Treatment of MBD is dependent upon the correction of inappropriate husbandry. An unsuitable calcium: phosphorus ratio of the diet should be corrected, the proper provision of ultraviolet light should be instituted and oral supplementation of calcium and vitamin D3 should be initiated. Supplemental calcium during the treatment period is also strongly recommended.

Gout is defined as the deposition of uric acid and urate salts within visceral tissues and on articular surfaces. Gout occurs as a result of hyperuricemia, which arises secondary to increased production or decreased excretion of uric acid. Increased production of uric acid may occur secondary to the ingestion of excessive amounts of protein (e.g., an herbivorous chelonian that is regularly offered animal protein). Decreased excretion of uric acid may occur secondary to reduced perfusion of renal tissues, which may be a result of dehydration, hemoconcentration, water deprivation, or renal disease. Reduced glomerular filtration eventually leads to a decrease in the overall excretion of urate salts, which results in hyperuricemia. Hyperuricemia, in turn, leads to the precipitation of urate complex microcrystals within tissues. These deposits are known as "gout tophi". Common sites of deposition of uric acid include articular joints and viscera. Clinical signs associated with gout include joint swelling and pain, depression, and dehydration. Affected animals are also commonly anorectic and lethargic. The diagnosis of gout may be done with blood work and radiographs/ultrasound. The mainstay of therapy is rehydration to correct the hyperuricemia, and the correction of any dietary imbalances or other predisposing causes of gout. Allopurinol, a urease inhibitor, is commonly used in hyperuricemic animals to reduce uric acid production. It must be mentioned that studies concerning the efficacy of this drug and the possible long-term effects of the drug in exotic pets have not be conducted. Probenecid, which increases the renal excretion of uric acid, should be not be used until the glomerular filtration rate is considered acceptable. Any concurrent infections in affected joints or organs that occur secondary to gout deposition should be treated appropriately. Surgery is occasionally indicated when uric acid deposits are compromising joints.

Providing appropriate nutrition is key to having success with reptiles. By providing the essentials, we can minimize the likelihood for these common nutritional diseases outlined previously. Take the time to learn about the needs of the species you work with, so that you can make the best recommendations for your clients.

087 / #75

MULTI-MODAL ANALGESIA IN PRACTICE

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ABSTRACT BODY:

Veterinarians should aim to use a multimodal approach to analgesia. By using a combination of drugs, different locations on the pain pathway are targeted and more effective analgesia is achieved (1). The treatment of pain will depend on pain recognition in that individual and species, and the type (inflammatory, nociceptive, neuropathic, mixed) and duration (acute/chronic) of the pain experience. This presengtation will discus medical and complementary therapies.

Planned acute pain

A pre-emptive analgesic plan using multiple pain management medications is a necessary part of any planned painful veterinary procedure. Without sufficient analgesia the animal will experience intra- and post-operative pain which will increase the risk of post-operative infection, self-trauma and wound breakdown. The provision of adequate pain relief pre-, intra- and post-operatively is crucial to minimise the negative impact on animal welfare as well as for successful outcome of surgery

A noxious stimulus such as a scalpel blade cutting the skin of the animal will stimulate the nociceptor and afferent nerve fibres in the skin. This signal will be carried by the nerves to the dorsal horn of the spinal cord and then up the ascending pathway to the brain where the brain interprets this signal as pain. The signals sent along the descending pathway from the brain to the spinal cord will try to reduce the acute pain experience. Analgesic drugs will have better efficacy if they are used pre-emptively, before the animal is exposed to the noxious stimulus, such as surgery. This is because once the brain receives the pain signals, it will mount a response which increases central nervous system sensitivity to pain meaning if the same stimulus is experienced again the animal will experience a much greater pain response which is more difficult to treat with analgesics.

We know that surgery is painful, and although the anaesthetised animal may not be consciously aware of pain, the central nervous system will still receive and process this painful stimulus. We must give an appropriate dose of analgesics before the surgery starts instead of waiting for the animal to show signs of pain intra-operatively or post-operatively, as once pain is felt, it is much more difficult to treat effectively. Because of this, a multimodal approach to analgesia is recommended, where multiple aspects of the pain pathway are targeted using several analgesic drugs. Veterinarians should consider the emotional as well as the sensory aspects of pain in their analgesic planning, and have a good awareness of the potential analgesic effects of drugs which may not be used as primary analgesics. For example ketamine and alpha 2 agonists may provide good analgesia despite being utilized as anaesthetics whilst isofluorane will not.

Unplanned acute pain

Continued exposure to a painful stimulus without the administration of pre-emptive analgesia results in wind up. This is where the injured nociceptors send continuous messages to the central nervous system and this can result in process where part of the spinal cord called the ‘dorsal horn’ is activated to send continuous pain messages to the brain and stops responding to the descending pathway’s messages to reduce the pain experience. Wind up may occur if surgery is performed without effective analgesia or in cases of unplanned acute pain, e.g due to trauma or injury. Wind-up can result in a more intense pain experience (hyperalgesia) and because the process of ‘wind-up’ has occurred and the animal is already in pain, any pain relieving medication given post-operatively will be less effective, and higher doses may be required more frequently to successfully manage pain. Unmanaged pain also increases the likelihood of complications such as allodynia (feel pain when a none-painful stimulus e.g. gentle touch) is applied. Where pain is unpredictable or occurs suddenly due to trauma, multimodal analgesia should be used to target multiple aspects of the pain experience and reduce the pain sensations.

Chronic pain (e.g. due to degenerative disease)

Chronic (long-term) pain occurs when ongoing nociceptor and peripheral nerve activity leads to chronic dorsal horn activation in the spinal cord and an ineffective descending pathway response. For example, in untreated dental disease or osteoarthrtitis. Detecting chronically painful conditions can be much more challenging than detecting acute pain, and most animals living with chronic pain will modify their behaviour in order to try and cope with their pain experience. Such animals will reduce their activity, may rest more, and may be more irritable. However

they may also still display highly motivated behaviours (animals with severe dental disease will still eat, dogs with osteoarthritis will still want to chase balls despite pain in their joints) and so we should evaluate overall activity and not to assume that the continuation of one or two highly motivated activities represent a lack of pain. Perhaps counterintuitively, animals with chronic pain may also increase their activity. For example abnormal repetitive behaviours are associated with chronic pain in mammals (2), and birds (3). There are a number of chronic pain assessment scales such as the Helsinki chronic pain index (4).

Multi-modal analgesic options

Access to analgesic drugs may vary depending on local geography and regulations. Veterinary drugs should only be used if their mechanism of action and side effects are understood and administered under the direction of suitably qualified veterinary professional. The WSAVA Global pain council guidelines are an invaluable resource for developing pian management strategies https://wsava.org/global-guidelines/global-pain-council-guidelines/.

The neurological targets of analgesic drugs may include the inflammatory cascade (NSAIDS), peripheral neurons (Local anaesthesia), the spinal cord processing of pain (ketamine, alpha 2 agonists) the ascending pathway and the brain processing (tricyclic antidepressants, ketamine, opioids and tramadol) and the descending pathway (opioids, tramadol, xylazine, paracetamol). By using a combination of drugs, working on different locations on the pain pathway, more effective analgesia is achieved. Additionally the type of pain that may be being experienced should be considered as not all drugs are effective at treating all types of pain. Drugs such as amitriptyline or nortriptyline, and pregabaline or gabapentin are the only types of drug that are reliably effective against neuropathic pain for example.

In addition to pharmacotherapeutic agents, complementary therapies may be effective in mitigating the pain experience. Nutraceuticals or nutritional supplements including glucosamine-chondroitin, green-lipped mussels, and omega-3 fatty acids have been shown to improve mobility in animals experiencing osteoarthritis (5). Physical therapy including targeted massage and activities, have been used in a range of species to support recovery from surgery and maintain activity despite chronic illness. Low level laser therapy (6) has biomodulation properties, and promotes wound healing and cell renewal as well as reducing the pain experience in animals with tissue injury or underlying arthritis. Both traditional and electro-acupuncture is also documented in a range of species including rabbits, rodents, dogs, cats, birds and snakes (7) and may be helpful for alleviating painful conditions and improving mobility.

The social and environmental provisions for painful animals should also be considered. The pain experience is modulated by the emotional response to pain, so enhancing an animal's emotional state mitigates their experience of pain. Supporting positive emotional experiences has also been shown to mitigate chronic pain experiences (8-10), and so it is important that animals with painful conditions are comfortable, have good social relationships with people and other animals, and enjoy a range of enrichment activities to enhance their emotional wellbeing.Euthanasia of painful animals is a valid approach to preventing and alleviating further suffering, after a cost:benefit analysis of the animal's welfare and likely outcomes.

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088 / #181

TICKS, DOG BITES, AND VERTICAL TRANSMISSION: CURRENT KNOWLEDGE ON MECHANISMS OF TRANSMISSION OF BABESIA SPP. IN DOGS

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ABSTRACT BODY:

Tick-borne pathogens have always attracted the interest of researchers due to their increased prevalence and distribution, hence representing a serious problem for animals and humans.

Canine babesiosis has been recognized as one of the most important worldwide tick-borne diseases of dogs. It is caused by numerous species of *Babesia* and transmitted by ixodid ticks, such as *Dermacentor reticulatus* (*Babesia canis*), *Rhipicephalus sanguineus* (*Babesia vogeli*), *Haemaphysalis leachi* and *H. elliptica* (*Babesia rossi*). These 3 species have been classified as "Large Babesia", due to the merozoites size. "Small Babesia" forms comprise *Babesia gibsoni, B. conradae, B. vulpes*, and a new a new piroplasmid species, *B. negevi*, recently identified in different areas of Israel. (Baneth et al., 2020). In addition to the emergence of new pathogenic species, the spread of the disease to non-endemic areas by population movements and the expansion of the tick habitats has been reported (Krämer, 2009). Wild animals, such as gray wolves and jackals are asymptomatic carriers of *B. canis* (Sukara et al., 2018), and over the last couple of years, there has been growing evidence that stray dogs contribute to the spread of the parasite (Milanović et al., 2020). Babesia sporozoites from the tick's saliva are transmitted during their bloodmeal on the host and infect the erythrocytes, where they propagate by merogony. *Babesia sensu lato* (s.l.) display transstadial transmission exclusively, that requires the larvae or nymphs to acquire the parasite during the blood meal. Parasite is transmitted to the next nymph or adult stage during during tick molting. (Florin-Christensen and Schnittger 2009). Besides transstadial transmission, *Babesia sensu stricto* (s.s.) to transmit the parasites have the ability to transmit the parasites by transovarial transmission. Babesia invade tick ovaries and eggs, and, subsequently, salivary glands of the tick larvae of the next generation (vertical transmission). Transplacental transmission has been demonstrated as an alternative form of vertical transmission for an increasing number of piroplasmid species, ensuring parasite progression into the next vertebrate generation without the need for a tick vector (Florin-Christensen et al. 2021). Other types of transmission cannot be ruled out: for *B. gibsoni* together with the transplacental mode, iatrogenic routes, transfusion, bites and ingestion of infected blood has been described; for *B. canis* and *B. vulpes* (*Babesia s.l.)* the vertical route also seems plausible.

Almost all species of *Babesia* spp. have been identified in Europe, with high prevalence for *B. canis* and *B. vogeli* (Solano-Gallego et al., 2016). Few studies have reported the presence of *B. rossi* in the European territory, in particular a very recent one conducted in Romania allowed to estimate its prevalence (2.2%; p = 2/90) (Ciuca et al., 2021). *B. microti-like* and *B. gibsoni* have been reported in several European countries (Lempereur et al., 2017). Canine babesiosis in Latin America Countries is mainly caused by *Babesia vogeli and Babesia gibsoni.*

The predominant tick in Italy is *R. sanguineus* (n = 769/1217; p = 63.6%), less present but still widespread are the species of *I. ricinus* (n = 372/1217; p = 30.6%) and *I. hexagonus* (n = 68/1217; p = 5.6%) (Maurelli et al., 2018). In a recent study, *Babesia* spp. infection rates were significant for *I. hexagonus* (3.1%), *I. ricinus* (2.7%), and *R. sanguineus* (5.4%) (Zanet et al., 2020). In Italy, *B. canis* is more widespread in northern regions, in agreement with the distribution of the related vector *D. reticulatus*, while *B. vogeli* is reported mainly in central-southern Italy, where *R. sanguineus* is the predominant tick (Olivieri et al., 2016). The epidemiology and geographic distribution of *B. gibsoni* in dogs on the Italian peninsula remains unclear until now. In an experimental study conducted in Naples, Avellino, and Salerno, provinces of the Campania region, the seroprevalence for *B. canis*/*B. vogeli* was 14.0% (n = 184/1311) and for *B. gibsoni* 0.2% (n = 3/1311). The molecular analysis showed a prevalence 0.15% (n = 2/1311) for *B. canis* and 1.1% (n = 15/1311) for *B. vogeli* (Veneziano et al., 2018). Canine babesiosis to date still presents a challenge for the clinicians, especially due to the wide range of clinical presentations, due to the limited options of specific diagnostic during the acute phase and due to the side effects and lack of efficacy of the current drugs for *B. gibsoni.* Infection

of dogs by *Babesia spp.* results in very varying clinical presentations, depending on the species as well as the age, immune status, and concomitant infections of the affected animal. Clinical signs can include fever, anemia, lethargy, anorexia, thrombocytopenia, tissue anoxia, and organ dysfunction, among others, and infections can be fatal (Baneth 2018b). Accurate molecular detection and species identification are important for the selection of correct therapy, for predicting the course of disease in dogs with babesiosis and for the evaluation of subclinical infections. Awareness should be raised in case of co-infection with leishmaniosis, taking into account the possibility of occurence of glomerural disease in *B. gibsoni*-infected dogs. Several drugs and drug combinations have been described for the treatment of acute canine babesiosis; however, they are not able to eliminate the parasite leading to asymptomatic carrier animals that may relapse and transmit the infection.

089 / #85

CHOOSE YOUR ADVENTURE- MANAGEMENT OF THE COLLAPSED PATIENT

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ABSTRACT BODY:

Acute collapse is often a very alarming situation for the pet owner and can represent a diagnostic challenge for the veterinary practitioner if the event is not witnessed or recorded. A careful history, as well as a thorough physical examination should be obtained in order to decipher the cause of acute collapse in dogs and cats. The term “collapse” is generally defined as a loss of postural tone with or without a loss of consciousness. One of the first questions to ask is whether the animal experienced a loss of consciousness. Some pet owners cannot answer that question accurately, as the animal usually keeps the eyes open, even when loss of consciousness has occurred. I often will ask whether the animal was responsive or unresponsive to verbal or tactile stimuli during the event. If the animal was not responsive, then I err on the side that there was a loss of consciousness until proven otherwise. If there is loss of consciousness, primary differential diagnoses include seizure or syncope, versus neuromuscular or metabolic causes of collapse. One other question to ask, particularly if there was not a loss of consciousness or a history of being unresponsive, is whether the collapse affected all four limbs, or affected only the front or rear, right or left side of body. This can lead to pursuing neuromuscular causes of collapse, or generalized weakness from metabolic disorders (hypoglycemia or hypokalemia for example) or anemia.

Seizures

Tonic clonic, or grand mal seizures, are one of the primary differential diagnoses when a client reports that their animal has collapsed. In many cases, animals with seizures will have muscle twitching and rhythmic rigid contractions of the muscles of the face and limbs, often with hypersalivation, movement of the jaws and teeth, and paddling movement of the limbs. Urinary and fecal incontinence also may occur. Unlike syncope, animals often will experience abnormal behavior before (pre-ictal) and after (post-ictal) the actual seizure event. There are numerous causes of tonic-clonic seizures that can include intracranial mass lesions or infection, increased intracranial pressure, toxin exposure, hypoglycemia or cerebrovascular accident in addition to idiopathic or cryptogenic epilepsy.

Syncope

Syncope, more commonly known as fainting, occurs due to temporary lack of blood flow to the brain. Syncope can be secondary to a variety of causes, however cardiac dysrhythmias are high on the list of differential diagnoses. In general, unless there is an ongoing cardiac dysrhythmia (sustained severe brady- or tachycardia), the episode of collapse secondary to syncope is short-lived, with a rapid recovery within seconds to a minute or less. At the time of presentation, it is useful to perform an ECG to investigate for sinus arrest, idioatrial rhythm (usually secondary to hyperkalemia, or can be lone atrial standstill in English Springer Spaniels), or high grade AV block. Less commonly but noteworthy are tachyarrhythmias such as atrial fibrillation, atrial flutter, and ventricular tachycardia that can also lead to hypotension and syncope.

Questioning the owner about any event that preceded the episode of collapse/syncope can help reveal whether neurocardiogenic or vasovagal syncope has occurred. During an episode of syncope, an animal can demonstrate muscle rigidity with opisthotonos, or can be flaccid with little to no muscle tone. Urinary and fecal incontinence may also occur, making the episode difficult to discern from seizures.

Neurocardiogenic syncope occurs under certain situations that cause a sudden decrease in heart rate with vasodilation and increase in stimulation of the vagus nerve, causing acute vasomotor collapse and loss of consciousness. Situations that increase vagal tone, such as extreme excitement, coughing, vomiting or retching or urination/defecation can result in increased vagal tone with resultant syncopal collapse. Rare cases of syncope associated with swallowing have also been reported.

In addition to ECG monitoring, thoracic radiographs should be performed to evaluate for cardiomegaly, pulmonary pattern consistent with pulmonary hypertension or heartworm disease, pulmonary nodules or intrathoracic masses. Echocardiography should also be considered to investigate for primary valvular or infiltrative cardiac disease. Depending on the findings, Holter or event monitoring may be recommended, in case the cardiac dysrhythmia is intermittent and not apparent at the time of presentation to you.

Anemia/Blood loss

Weakness with subsequent collapse can be associated with loss of intravascular fluid volume from

hemorrhage, as well as from acute or chronic anemia. A minimum database that includes a PCV to investigate for anemia should be performed to determine if anemia is present. In the event of acute hemorrhage, such as a bleeding intra-abdominal or retroperitoneal mass or acute pulmonary hemorrhage from neoplasia or Vitamin K antagonist rodenticide intoxication, peripheral PCV may not immediately reflect anemia due to splenic contraction. Even in the absence of anemia, a rapid AFAST/TFAST ultrasound examination should be performed to investigate for the presence of abdominal and thoracic effusion. While performing these examinations, the clinician can also rule out pericardial effusion/tamponade as another cause of acute weakness and collapse.

Metabolic Causes of Weakness/Collapse

Animals with diabetes mellitus or chronic kidney disease could develop severe hypokalemia that contributes to muscle weakness. Hypoglycemia may be found in neonates (particularly toy breeds), animals with portosystemic shunting, inadvertent insulin overdose, insulin-secreting tumors, hypoadrenocorticism or sepsis. Other potential causes of muscle weakness and collapse include thiamine deficiency in aged cats. A complete biochemical and electrolyte panel can help determine whether hypoglycemia is contributing to signs of weakness, collapse, and/or seizure activity. If an insulin secreting tumor is suspected (albeit rare, but does occur in both dogs of all sizes and cats), obtaining a citrated blood sample at the time of glucose nadir, prior to administration of supplemental dextrose, is recommended. Severe hypothyroidism also can cause severe weakness, lethargy, and somnolence. In such instances, the animal may appear to have facial edema secondary to myxedema, as well as clinical signs of being overweight and having a poor haircoat.

Exercise Induced Collapse

Collapse can occur secondary to exercise and can be associated with a genetic defect in ability to process calcium. This has been reported in lines of Labrador retrievers. Exercise induced collapse also occurs in some lines of Border Collies. Phosphofructokinase deficiency, noted rarely but primarily in English Springer Spaniels, can be associated with acute intravascular hemolysis, weakness and collapse after periods of exercise or excitement/panting. Laryngeal paralysis, laryngeal obstruction, brachycephalic airway disease, as well as hyperthermia secondary to high ambient humidity and temperatures can also result in collapse and heat-induced illness or heat stroke.

Narcolepsy

One of the most rare forms of collapse and loss of consciousness is narcolepsy. Animals with narcolepsy will collapse spontaneously, usually while in the process of a normal activity, then wake up and immediately resume its normal activity. Having a client video an episode and keep a diary can potentially help make a diagnosis of narcolepsy. While there is no cure for narcolepsy, some drugs to treat hyperactivity or tricyclic antidepressants may decrease the frequency of episodes.

References available upon request

090 / #84

SUCCESS AND CHALLENGES IN THE COLLABORATIVE EFFORTS BY BREEDERS, JUDGES AND VETERINARIANS TO ENHANCE CANINE GENETIC HEALTH

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ABSTRACT BODY:

Breeders and veterinarians do share- among other issues – a responsibility for wise selection of breeding stock to ensure health and well-fare in dogs to be produced.

Breeders are the ones making the ultimate decisions on dogs to be mated. Veterinarians in various capacities are assisting in those decisions.

Formal screening programs to indicate superior breeding stock with reference to health status in i.e. hips and eyes have been in place for more than half a decade. Many more phenotypic screening programs have evolved over the years and now also possibilities for molecular genetic screening programs.

Likewise formal health schemes/ certificates for evaluation of puppies at time of delivery are in place and even mandatory for breeders by some Kennel Clubs.

Since formation of Kennel clubs and dog shows also show judges have been involved in the evaluation of the outcome and potential breeding stock. Unfortunately the perception by show judges and veterinarians of a desired dog of some breeds is not always compatible.

To overcome that Breed Specific Instructions for judges *with reference to exaggerated anatomical (BSI) have been developed in collaboration between breeders, judges and veterinarians* as a complement to Breed Standards.

To develop and implement an effective usage of all these features and further collaborative efforts is an important task for the veterinary profession as well as cynological organisations

How to collaboratively handle exaggerated anatomical feature is a highly actual challenge.

Excessive skin folding and compromised airways are examples of anatomical features by intention not promoted by breed standards and their interpretation is not compatible with the perception of a healthy dog by the veterinary profession

An alarming high prevalence of the Brachycephalic Obstructive Airway Syndrome in some dog breeds have created a polarisation between some breeders of these breeds and the veterinary profession regarding extent as well as measures to be taken to handle it.

To somewhat lesser extent that also goes for the appearance of many individuals in the Shar Pei breed.

The extent of clinical signs related to anatomical features have called for banning of some breeds in a number of countries.

The veterinary profession may assist in pre-breeding examinations for clinical signs related to exaggerated anatomical features.

An evaluation of breathing capacity as in Respiratory Function Grading RFG Scheme developed at the University of Cambridge is a good example. Pre-breeding examination of the adnexa of the eyes is another.

However neither banning of breeds nor veterinary examinations are enough to make a substantial impact on the prevalence of dogs suffering from exaggerated anatomical features.

It is also needed that the perception - by the public and breeders of brachycephalic breeds - of a desired dog in these breeds are changed to something less exaggerated. To achieve that an ongoing dialogue between breeders and veterinarians is needed.

To enhance a dialogue between various stakeholders on how to enhance canine genetic health a number of Dog Health Workshops have been arranged since 2012 and the formation of the International Partnership for Dog (IPFD). https://dogwellnet.com/

As a frame for collaborative efforts the International Collaborative on Extreme Conformation in Dogs (ICEC dogs) have been created. https://dogwellnet.com/ipfd/

I urge the national small animal organisations worldwide to initiate the establishment of collaborative platforms and efforts to counteract Extreme conformations in Your country/region. Take the lead and keep it until the perception of a desired dog in brachycephalic breeds have improved by less exaggerated anatomical features.

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091 / #185

IS IT RUPTURED? DIAGNOSTIC IMAGING OF GALLBLADDER DISORDERS IN DOGS

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ABSTRACT BODY:

IS IT RUPTURED? DIAGNOSTIC IMAGING OF GALLBLADDER DISORDERS IN DOGS

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Gallbladder disease in dogs can have serious consequences including ultimately gallbladder rupture with bile peritonitis. It is thus important to identify and address gallbladder disorders with imaging. It is equally important to recognize signs of gallbladder rupture in patients presenting with signs of acute abdominal disease. Gallbladder diseases that may lead to gallbladder rupture, or require surgical intervention include gallbladder mucocele, gallbladder wall compromise due to severe inflammation, ischemia, or neoplasia, and biliary obstruction.

Radiographs are not sensitive to detect gallbladder disease. Mineralized choleliths can be recognized radiographically, but it is difficult to determine with certainty where exactly they are located. Biliary rupture is radiographically characterized by loss of peritoneal detail in the cranial abdomen, but this is a nonspecific finding and can be associated with other diseases such as pancreatitis as well. Although computed tomography may recognize biliary obstruction and gallbladder mucocele, ultrasound is the current method of choice to identify and characterize biliary disease, determine presence of rupture, and obtain samples if indicated.

Biliary mucocele is an emerging gallbladder disease that has increased in frequency over the last 10-15 years. Older small breed dogs are typically affected with certain breed. It is a devastating disease due to the fact that a large number of mucoceles ultimately lead to gallbladder rupture, with a prevalence of rupture between 21 and 60%. Surgical gallbladder removal is the treatment of choice in most dogs, but is associated with a relatively high perioperative mortality rate of approximately 27%. The risk of perioperative death is significantly higher in dogs with gallbladder rupture compared with dogs without presence of rupture. Dogs with biliary mucoceles can be asymptomatic to severely ill and can have signs of bile duct obstruction. Mucocele formation is caused by an excess of a gel forming mucin by the gallbladder epithelium. Upon secretion of the mucus there is abnormal un-packing of mucus granules with then become tethered to the epithelium.

Biliary mucoceles in dogs and are characterized histologically as mucinous gland hyperplasia and inspissation of bile. The result is a characteristic appearance of the gallbladder on ultrasound: it is enlarged and filled with immobile bile in a stellate, striated or mixed pattern. Peripheral columns of inspissated bile results in hypoechoic striations extending from the inner lining of the gallbladder wall into the lumen. The inspissated bile can extend into the bile duct leading to obstruction and dilation. Bile duct obstruction is thought to occur secondary to the mucocele formation rather than to cause it. Since the gallbladder wall is prone to rupture in presence of a mucocele, surgery and removal of the gallbladder is usually indicated. Evidence of gallbladder rupture includes focal peritonitis characterized by echogenic fluid and hyperechoic inflamed tissue in the gallbladder fossa, discontinuous gallbladder wall. However, it is important to know that the sensitivity and specificity of ultrasound for detection of rupture is limited and the gallbladder wall may be intact even if there is suspicion ultrasonographically for rupture, and the other way round. In rare cases the actual clump of inspissated bile may be observed free in the peritoneal cavity but usually preserves its typical striated structure. The free floating mucoceles can migrate quite far through the peritoneum and may be found as far as at the level of the urinary bladder.

There are many causes for gallbladder wall thickening, and most of them are not associated with gallbladder rupture. Possible causes include cholecystitis, where necrotizing cholecystitis may in fact lead to gallbladder wall compromise. Liver disease can cause gallbladder wall inflammation and edema in chronic liver disease can be associated with impaired gallbladder contractility. Systemic disease such as heart or renal failure, hypo album anemia, or sepsis may lead to gallbladder wall edema. Extra cholecystic inflammation such as acute hepatitis, pancreatitis, pyelonephritis, or peritonitis can lead to spread of inflammation to the gallbladder wall. Finally, rarely we see neoplasia such as carcinoma or lymphoma in the gallbladder wall. Gallbladder wall infarction also leads to gallbladder wall thickening and immobility, with subsequent rupture due to necrosis. It is therefore important to gather all

the clinical, clinicopathologic, and additional imaging findings to evaluated patient with gallbladder wall thickening.

Ultrasound is the current method of choice to determine presence of an extrahepatic biliary obstruction. The normal gallbladder has a very thin, smooth and slightly hyperechoic wall, if it is visible at all. In dogs presence of biliary sludge is common and usually not associated with hepatobiliary disease. From the gallbladder neck the cystic duct emerges and is joined by the hepatic biliary ducts. After the junction with the hepatic duct, the common bile duct extends caudally, ventral to the portal vein to the major duodenal papilla. The normal bile duct is rarely visible in dogs and should not exceed 3mm in diameter.

Bile duct enlargement is the main indicator of extrahepatic biliary obstruction. However, bile duct dilation may be absent if there is presence of periductal inflammation, fibrosis and bile duct stricture. Bile duct peristalsis is also sometimes able to propagate a small amount bile forward and through the duodenal papilla despite cholestasis and hyperbilirubinemia and bile duct dilation is consequently not as severe as one would expect. Dilation of the gallbladder and / or intrahepatic bile ducts is seen in a percentage of animals and is likely related to location, duration and severity of the obstruction. Nevertheless, enlargement of the biliary system always has to be interpreted in the light of clinical and blood chemistry findings, as common bile duct dilation can persist after previous obstruction. Sequential bilirubin levels should be monitored to determine if current obstruction is present.

Inflammatory stricture of the bile duct due to pancreatitis is one of the most common causes for extrahepatic bile duct obstruction. Ultrasonographic changes include an enlarged, irregular and hypoechoic pancreas. Inflammation of the surrounding mesentery leads to hyperechoic peripancreatic fat and focal peritoneal effusion. It is rarely possible to determine of the bile duct obstruction in patients with pancreatitis is functional in the sense of a spasm or paralysis, or if it is mechanical due to pressure from the surrounding inflamed or necrotic tissues. In severe cases, exploratory laparotomy may be needed to relieve the obstruction and place a bile duct stent. Sluggish bile seen with inflammatory diseases (cholangitis). Gallbladder wall and bile duct wall thickening is often present in these cases, and can result in obstruction. Cholelithiasis and bile duct neoplasia which tends to occur at the level of the duodenal papilla are additional causes for bile duct obstruction. A thorough ultrasound examination therefore has to include evaluation of the entire bile duct and duodenal papilla.

The indications for exploratory surgery in case of suspected bile duct obstruction are not very well defined. Generally, surgery is recommended if there is clear imaging evidence of bile duct obstruction such as choleliths within the bile duct, and if this is supported by clinical and clinicopathologic signs of biliary obstruction. Risks of non-surgical treatment could include rupture of the bile duct and liver damage by refluxed bile, both of which are not well described in the veterinary literature. In people, as few as 7.3% of people get cirrhotic changes in the liver after biliary obstruction and surgical intervention is often only performed if signs of bile duct obstruction last for more than a month. Guidelines in the human literature indicate that conservative management is preferred unless there is severe pain, suspicion of neoplasia, clear mechanical obstruction or severe icterus for over a month.

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PEDIATRÍA FELINA: CUIDADOS INTENSIVOS PERINATALES

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ABSTRACT BODY:

EXAMEN FÍSICO DEL RECIÉN NACIDO

Para realizar el examen físico del recién nacido debemos saber cuáles son los parámetros fisiológicos.

TABLA 1

PARÁMETRO	VALOR
PESO	90-110 GR
Tª nacimiento	36-37ºC
Tª MES	38ºC
FC	220-260 LPM
FR nacimiento	10-18 rpm
FR SEM	15-35 rpm
DEO	1.006-1.020
PRODUCCIÓN ORINA	25 ML/KG/DÍA à 2,5 ml 100 GR
LIQUIDO	130-200 ML/KG/DÍA
CALORIAS	20 Kcal EM/100 gr/DÍA
CAPACIDAD ESTÓMAGO	4-5 ML/100 GR

La determinación de la edad de los gatitos se puede realizar con los hitos del desarrollo.

TABLA 2:

HITO DE DESARROLLO DE LOS GATOS	EDAD
CAÍDA DEL CORDÓN UMBILICAL	3
APERTURAS DE LOS OJOS	DÍA 7-10
REFLEJO PUPILAR Y DE AMENAZA	DÍA 28
VISIÓN NORMAL	DÍA 30
COLOR NORMAL DE IRIS	4-6 SEM
APERTURA CANAL AUDITIVO	DÍA 9
OÍDO FUNCIONAL	4- 6 SEM
SE DESPLAZAN	DÍA 7-14
CAMINAN	DÍA 14-21
ELIMINACIÓN VOLUNTARIA	SEM 3
INCISIVOS/CANINOS , PREMOLARES	3-4 SEM 5-6 SEM

También se puede aproximar la edad del gatito a través de su peso.

TABLA 3

VALORACIÓN DEL ESTADO DE HIDRATACIÓN

Las indicaciones para la fluidoterapia en neonatos incluyen reanimación posparto, shock, trauma, deshidratación, hipoglucemia, hipotermia, separación de la madre, sepsis, desnutrición e incapacidad para amamantar.

Tienen una mayor relación área superficial/volumen y una mayor permeabilidad de la piel, lo que los hace susceptibles a pérdidas rápidas de líquidos. Los recién nacidos también tienen un mayor contenido de agua corporal total (80% del peso corporal en comparación con el 60% en animales adultos) y tienen una función renal inmadura. Los recién nacidos no pueden concentrar su orina hasta las 8 semanas de edad en los gatitos y las 12 semanas de edad en los cachorros.

Los recién nacidos tienen una gluconeogénesis hepática ineficiente y reservas limitadas de glucógeno, y no pueden mantener la homeostasis de la glucosa si se les priva de alimentos durante más de unas pocas horas. Siempre se debe tener en cuenta la necesidad de glucosa durante la rehidratación y reanimación de los recién nacidos enfermos.

Los recién nacidos también están mucho más predispuestos a la hipotermia que los animales adultos

debido a su mayor área de superficie y la falta de respuesta de escalofríos (la respuesta de escalofríos no se desarrolla hasta el día 6 de vida) . Es importante administrar siempre líquidos tibios para tratar o prevenir la hipotermia. Los recién nacidos hipotérmicos desarrollan bradicardia e íleo gastrointestinal.

La alimentación oral no es efectiva hasta que se restablece la normotermia.

La piel delgada en el abdomen ventral es el mejor lugar para evaluar el estado de hidratación en los recién nacidos. En animales adultos, la deshidratación severa causa taquicardia y orina concentrada. No se puede confiar en estas variables en los recién nacidos porque la frecuencia cardíaca normalmente es rápida y la orina no se concentra en los recién nacidos.

Las membranas mucosas a menudo permanecen húmedas en los recién nacidos hasta que la deshidratación es severa.

El color de las mucosas y el tiempo de llenado capilar son buenos indicadores de perfusión en neonatos y animales adultos. Los recién nacidos normalmente tienen membranas mucosas **hiperémicas durante la primera semana de vida.** Se observan membranas **mucosas pálidas y tiempo de llenado capilar lento (> 1,5 segundos) cuando la deshidratación es lo suficientemente grave como para causar un shock hipovolémico**. Los cachorros con mala perfusión aún pueden tener una turgencia normal de la piel incluso cuando el color de la membrana mucosa y el tiempo de llenado capilar indican shock. Los signos clínicos de sépis, hipovolemia o shock en los recién nacidos incluyen membranas **mucosas pálidas, disminución de la producción de orina, extremidades frías, tono corporal flácido, llanto constante y no maman**. El veterinario debe determinar si el animal tiene deshidratación simple o déficit de perfusión real. Cuanto más enfermo está el animal, más agresiva es la vía elegida para la fluidoterapia. Los recién nacidos con sepsis o hipovolemia grave necesitan reanimación rápida y agresiva con líquidos por vía intravenosa o intraósea . Los recién nacidos con deshidratación de leve a moderada pueden recibir líquidos por vía oral (por sonda), por vía subcutánea o intraperitoneal.

Fluidos orales

La alimentación por **sonda** se puede usar para administrar líquidos y nutrición a los recién nacidos que no están hipotérmicos, hipoglucémicos o deshidratados.

Los recién nacidos también se pueden alimentar con una jeringa, un cuentagotas o un biberón. Se debe tener cuidado no administrar el líquido demasiado rápido porque la neumonía por aspiración es una complicación común.

Líquidos subcutáneos

Los recién nacidos con deshidratación de leve a moderada pero perfusión normal pueden tratarse mediante la administración subcutánea de líquidos. Los requisitos de mantenimiento son de dos a tres veces mayores que los de los animales adultos (120-180 ml/kg/d. No se debe agregar dextrosa a los líquidos isotónicos porque el líquido resultante se vuelve hipertónico y en realidad atrae líquido hacia el espacio subcutáneo. El mejor líquido para corregir la deshidratación leve es una solución electrolítica equilibrada, como la solución de Ringer lactato o Normosol-R. Si se desea un suplemento de dextrosa, se puede administrar de forma segura NaCl al 0,45% con dextrosa al 2,5% por vía subcutánea. Si hay hipopotasemia, se pueden agregar hasta 30 mEq/L de KCl a los líquidos para administración subcutánea.

Los recién nacidos que no pueden amamantar durante las primeras 24 horas se ven privados de calostro, desarrollan fallos en la transferencia pasiva, lo que los pone en alto riesgo de desarrollar infecciones . La administración subcutánea de suero de la madre o de algún otro gato adulto bien vacunado puede proporcionar anticuerpos protectores.

Líquidos intraperitoneales

Los líquidos cristaloides se pueden administrar por vía intraperitoneal. Las soluciones de dextrosa hipertónica no deben administrarse por vía intraperitoneal, especialmente a los recién nacidos deshidratados, porque el líquido se extrae del espacio intravascular y del intersticio hacia la cavidad abdominal. La instilación de líquidos tibios en la cavidad peritoneal puede ser un método útil para tratar la hipotermia y el aumento de la temperatura corporal central.

Líquidos intravenosos

Los recién nacidos con déficit de perfusión no absorben bien los líquidos administrados por vía subcutánea debido a la vasoconstricción periférica asociada con el shock hipovolémico. La reanimación agresiva y la restauración del volumen intravascular se pueden lograr con líquidos administrados por vía intravenosa. Se puede administra un bolo inicial de 1 ml por cada 30 g de peso corporal (30 a 45 ml/kg) por vía intravenosa lenta durante 5 a 10 minuto y continuar hasta que el color de la membrana mucosa y el tiempo de llenado capilar han mejorado a un color rosa brillante con un tiempo de llenado de 1 segundo. El líquido cristaloide tibio, como la solución de Ringer lactato o Normosol-R con dextrosa al 5% añadida, es el líquido de elección. Después del bolo inicial, la

fluidoterapia se continúa a razón de 80 a 120 ml/kg/día.

Fluidos intraóseos

Cualquier fluido que se pueden administrar por vía intravenosa (p. ej., sangre, solución electrolítica balanceada, glucosa) también se pueden administrar por vía intraósea.

El período infantil en pacientes pediátricos oscila entre las 2 y las 6 semanas de edad. Durante este tiempo, los problemas que más amenazan la vida son los parásitos internos y externos, la hipoglucemia juvenil, la deshidratación por diarrea y los traumatismos . Debido a que la función renal no madura hasta al menos las 8 semanas de edad, los lactantes siguen siendo propensos a la toxicidad del fármaco por la disminución de la eliminación renal y la deshidratación por la disminución de la capacidad para concentrar la orina.

convulsiones, estupor y coma. El tratamiento consiste en la administración intravenosa o intraósea de glucosa (0,5 a 1 g/kg) diluida en una solución del 5% al 10%, seguida de tratamiento de soporte.

EL PERÍODO JUVENIL

Los juveniles son pacientes pediátricos de 6 a 12 semanas. Son más similares a los adultos en términos de función renal y signos vitales, pero aún tienen mayores requerimientos de agua de mantenimiento (120–200 mL/kg/d) y requerimientos calóricos (180 kcal/kg/d). Debido a que los anticuerpos maternos se pierden durante este tiempo, los gatitos en éste período son susceptibles a enfermedades infecciosas a menos que estén protegidos por vacunas. La panleucopenia felina es potencialmente mortal que requieren un apoyo agresivo con fluidoterapia y tratamiento de soporte.

RESUMEN

- Los pacientes pediátricos tienen requisitos de mantenimiento más altos para la terapia de fluidos que los animales adultos y son más propensos a la deshidratación y al shock hipovolémico debido al mayor contenido de agua corporal total y la función renal inmadura.
- El tratamiento agresivo con fluidos intravenosos o intraóseos, o las estrategias preventivas con fluidos subcutáneos pueden ser técnicas que salvan vidas en estos pacientes frágiles.

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093 / #87

LINFOMAS CUTÁNEOS FELINOS Y LINFOCITOSIS

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ABSTRACT BODY:

En el gato se han descrito y caracterizado tres procesos linfoproliferativos cutáneos:

- Linfoma cutáneo no epiteliotrópico (de células T, raramente de células B)
- Linfoma cutáneo epiteliotrópico (de células T)
- Linfocitosis cutánea (normalmente de células T)

En esta charla revisaremos las características clínicas y el tratamiento de cada una de estas tres entidades.

Características clínicas

En la tabla que sigue se presentan las características clínicas de las tres entidades.

Enfermedad	Presentación	Cuadro clínico
Linfoma cutáneo no epiteliotrópico	Más frecuente que el epiteliotrópico. La conexión con las infecciones por retrovirus no está clara.	Placas y nódulos dérmicos, únicos o múltiples. Existe una variante que afecta la piel de los tarsos exclusivamente. Cualquier parte del cuerpo. Afectación de nódulos linfáticos frecuente.
Linfoma cutáneo epiteliotrópico	Raro. 13 años de media. No hay otras predisposiciones.	Eritrodermia y exfoliación generalizada. Placas eritematosas que se ulceran. Tronco.
Linfocitosis cutánea	12-13 años de media. No hay otras predisposiciones.	Zonas de alopecia y eritema, con descamación, que evolucionan a placas que se ulceran. Tronco, extremidades o cabeza.

Diagnóstico

El diagnóstico se alcanza tras el estudio citológico, histopatológico, inmunohistoquímico y molecular (PARR - clonalidad).

Tratamiento

En la tabla que sigue se presentan los tratamientos recomendados para cada una de las tres entidades clínicas.

Enfermedad	Tratamiento recomendado
Linfoma cutáneo no epiteliotrópico	Extirpación quirúrgica si es factible. Poliquimioterapia. Como en linfoma nodales (ej. CHOP). Lomustina.
Linfoma cutáneo epiteliotrópico	Corticoides tópicos. Lomustina. Poliquimioterapia: vincristina – ciclofosfamida.
Linfocitosis cutánea	Glucocorticoides tópicos y sistémicos. Clorambucilo. Lomustina.

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OUTBREAK! A REVIEW OF THE EPIDEMIOLOGY OF VIRAL DISEASES IN REPTILES

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ABSTRACT BODY:

OUTBREAK! A REVIEW OF THE EPIDEMIOLOGY OF VIRAL DISEASES IN REPTILES

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Introduction

As the popularity of captive reptiles has exploded over the past three decades, there has been a significant rise in the number of emerging and re-emerging infectious diseases reported in reptiles. Emerging infectious diseases include newly identified pathogens, while those characterized as re-emerging include those that have been previously characterized but are being reported with increased frequency. Veterinarians play an important role in the diagnosis of infectious diseases in herpetological collections and should closely monitor the literature to keep abreast of new findings and current research.

The rise in emerging infectious diseases in reptiles may be attributed to several factors, including the increased number of reptiles being imported into the United States and Europe, poor quarantine and sanitation programs, and improved diagnostic assays. The popularity of reptiles in the United States remains high, with millions of reptiles being imported or captive-bred annually. The popularity of reptiles has led to the growth of reptile swap meets, where herpetoculturists have the opportunity to select from a large number of different reptile species. At these swap meets, large numbers of reptiles are held in relatively small areas with minimal to no biosecurity. Herpetoculturists routinely handle different specimens without washing their hands, possibly introducing and disseminating pathogens between the reptiles. The sanitation methods used to control or eliminate pathogens in reptile collections may also be suspect. Inappropriate use of disinfectants may lead to the development of resistant strains of microbes.

Infectious diseases are not only being disseminated via captive reptiles because of newly imported animals, but because of poor biosecurity in captive raised animals too. Mixing of different brood stock, not quarantining newly acquired animals, and poor biosecurity are also common issues with captive bred animals. When the author graduated veterinary school, it was common to suggest to clients that captive animals were “healthier” than wild caught animals. However, the author finds this hard to believe now, especially after 25 years of watching captive animals serve as the reservoirs for many of the viral, diseases being discussed in this presentation.

The number of diagnostic tests available to the clinician treating reptiles has increased dramatically over the past two decades. Historically, clinicians treated all “infections” in reptiles as bacterial diseases. However, over the past twenty years, there have been an increased number of reports of viruses being isolated from diseased reptiles. The advent of molecular diagnostic testing has led to the development of highly sensitive and specific enzyme-linked immunosorbent assays and polymerase chain reaction assays. These advanced assays allow clinicians to identify pathogens from a variety of different samples and allowed us to practice a higher level of clinical epidemiology.

Viruses

The incidence of herpesvirus infections in chelonians has been on the rise since originally being isolated from sea turtles in 1975. Herpesvirus infections have been identified in freshwater, marine, and terrestrial species of chelonians. Transmission of the herpesvirus is believed to be via the horizontal route, although it has been suggested that a vertical route of transmission is also possible. Affected animals may present with rhinitis, conjunctivitis, necrotizing stomatitis, enteritis, pneumonia, and neurological disease. Molecular diagnostics, electron microscopy, and viral isolation have been used to diagnose herpes infections in chelonians. Affected animals should be provided appropriate supportive care (e.g., fluids, enterals, and antibiotics) to control clinical signs. Acyclovir has been used with some success by reducing viral replication. However, there is no effective treatment for this virus. Affected animals should not be released into the wild to prevent translocation of the virus to naïve chelonians.

Bearded dragon atadenovirus was first reported in Australia in the early 1980’s. The virus was not characterized in the United States until more than a

decade later. Since that time, the virus has spread through the bearded dragon population in the USA and should be considered endemic. Transmission of the virus is primarily by the direct route (fecal-oral), although vertical transmission may also be possible. Affected animals may present with anorexia, weight loss, limb paresis, diarrhea and opisthotonous. Concurrent dependovirus and coccidial infections have also been observed in neonatal bearded dragons. Biopsies of the liver, stomach, esophagus, and kidney may be collected to confirm diagnosis (ante-mortem). On histopathology, basophilic intranuclear inclusion bodies are strongly suggestive of adenoviral infection. Currently, the best non-invasive ante mortem diagnostic test to confirm atadenovirus in a reptile is a polymerase chain reaction assay to detect the virus in oral secretions, blood, or feces of affected animals. There is no effective treatment for atadenoviral infections, although supportive care (e.g., fluids, enterals, antibiotics) may be useful in stemming the secondary effects of the disease. Again, very little is known regarding the epidemiology of this virus; therefore, special precautions should be taken when working with affected animals.

Ranavirus is an emerging disease of chelonians. This virus has a high morbidity and mortality. It has been isolated from both captive and wild chelonians. Affected animals typically develop upper respiratory signs (e.g., palpebral edema, conjunctivitis), lower respiratory signs, oral ulcers, cervical edema, and gastrointestinal signs. Diagnosis can be done using PCR. There is currently no effective treatment for affected animals, although temperature may play a role.

Inclusion body disease is something that has plagued snakes for many decades. It was only recently that this disease was found to have a high causal relationship with arenaviruses. Affected snakes primarily show neurologic signs, although regurgitation also occurs in some species. To date, this disease has only been reported in pythons and boas. Eosinophilic inclusions are a common finding on biopsies and at necropsy. There is currently no treatment available for this disease.

Viruses in reptiles remain a challenge for veterinarians because of our limited understanding of the epidemiology of these pathogens. It is important for us all to collect and share the knowledge we gain working with reptiles to develop a better understanding of these pathogens and work towards identifying novel treatments and better biosecurity methods to protect populations.

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HOW PAIN AFFECTS THE HUMAN-ANIMAL BOND

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ABSTRACT BODY:

HOW PAIN AFFECTS THE HUMAN-ANIMAL BOND

Humans have been keeping pets for a very long time and have been developing relationships with these animals. These relationships are known as the human-animal bond and has been defined by the Human Animal Bond Research Institute as a mutually beneficial and dynamic relationship between people and animals that is influenced by behaviours that are essential to the health and well-being of both[1].

The human-animal bond brings about benefits to the pet owner in terms of companionship as well as other documented benefits on their physical and mental health, as well as increasing positive social interaction and relationships with other people[2].

It has also been shown that strong human-animal bond relates to the provision of higher level of veterinary care for the animal, with the owners being more likely to seek preventative healthcare and are more compliant to veterinary advice[3]. Thus, this brings about a direct benefit to veterinary practices and to the veterinary profession in general.

In this presentation we will investigate ways in which pain can affect this bond and vice versa. Pain can be defined as an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage[4]. An animal that experiences pain in a way that causes a negative affective state can be said to be suffering. Pain is also an important measure of animal welfare as represented in one of the Five Welfare Needs; the 'need to be protected from pain, suffering, injury and disease'.

When an animal is in pain, they can react aggressively as a defensive action[5]. For example, a dog with spinal injury may not outwardly show signs of pain while at rest but the affected area is extremely pain upon touch. So, upon petting or hugging this dog, the dog may react by biting at the human. This could happen especially when owners do not pick up on subtle signs of pain. Children, in particular, can be very vulnerable to dog bites due to their limited understanding of dog behaviour and signs of animals in distress, and due to their size and height they tend to get more severe dog-bite injuries. Cats can also react aggressively towards humans when they are in pain, biting and scratching people when they are handled or want to be left in peace. These natural reactions of dogs and cats in pain can harm the human-animal relationship.

Another way in which pain can impair the human-animal bond is through changes in the animals' behaviours. Animals in pain may not be able to perform their routine behaviours. A common example is an arthritic cat may not be able to get into its litter box, which involves crossing over an elevated barrier. This can result in inappropriate elimination outside the litterbox or in unusual places. An unaware owner may see this as an animal that is misbehaving.

Chronic pain can cause changes in behaviours that develop very gradually and can be very subtle, therefore difficult to assess. The table below are some broad categories of behavioural changes in chronic pain[6].

CAT	DOG
• General mobility (e.g. ease of movement, fluidity of movement) • Performing activities (e.g. playing, hunting, jumping, using a litter-box) • Eating, drinking • Grooming (e.g. scratching) • Resting, observing, relaxing (how well these activities can be enjoyed by the cat) • Social activities involving people and other pets • Temperament.	• Vitality and mobility – how energetic, happy, active/ lethargic, contented, playful is the dog; ease of lying, sitting, jumping up, toler- ance to exercise • Mood and demeanour including states of alertness, anxiety, whether it is for example withdrawn, sad, dull, confident, its playfulness and sociability • Levels of distress (e.g. vocalization [moaning, groaning], demeanour [e.g. depressed] and response to other dogs and humans) • Indicators of pain (e.g. comfort levels, stiffness, lameness).

Table 1: Behavioural categories to assess for chronic pain taken from WSAVA's guidelines for recognition, assessment, and treatment of pain[6]

When an animal 'misbehaves' or is assumed dangerous to people or other animals, it breaks the bond between human and animal. Often, the owners of these animals may feel that relinquishment to shelters or euthanasia would be the best option for these animals. Behavioural problems are a major reason for euthanasia of companion animals[7].

Although the human-animal bond can increase empathy in humans[8], it can also affect animal welfare when people anthropomorphise their pets and treat them in a way that they think is good for the animals, but it may not actually be what the animals' need[7]. People's fear of pain can be mirrored to their pets; therefore, they might take steps to avoid causing any pain to their pets even though it may be beneficial. For example, people may be reluctant to neuter their pets because of the potential pain the surgical procedure may cause. Or they might avoid a visit to a veterinarian entirely due to the fear and distress it could cause to their pets, causing their pets to not get the adequate care that they needed.

People also fear that death may be painful and attribute this fear of death to their animals[5]. Therefore, there are many pet owners who are reluctant to euthanise their pets even though it may provide relief from pain and suffering. Some pet owners may opt for aggressive treatments or have unrealistic demands for their pets, even though it may have poor prognosis or have little benefits from the procedure.

Obesity in pets is becoming a widespread problem due to the anthropomorphic nature of the human-animal bond. Pet owners tend to overfeed or provide inappropriate treats to their 'hungry' pets. This can predispose their pets to painful conditions such as arthritis or joint diseases. Coddling painful pets with even more treats creates a spiral into poor health and welfare conditions for these pets.

There are three main ways vets can play a role in advocating and strengthening healthy human-animal bond through their practices. First, is to recognise that pain plays a major role in this bond. Vets must learn to anticipate and prevent pain whenever possible, and then recognise and relief pain in animals[5]. Teaching pet owners to recognise signs of pain is also equally important. Second, is to reduce stress and anxiety of animals during their visits to vet practices[9]. Examples includes using low stress handling and restraint techniques, having separate waiting areas for cats and dogs, using calming pheromones, and training staff to recognise signs of stress and anxiety in animals. The final method is to have good communication with pet owners[3,7]. The three main aspects of communication for this purpose are empathic communication, client engagement and education[7].

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096 / #268

TRANSMISSIBLE VENEREAL TUMOR CLINICAL SIGNS AND DIAGNOSIS

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ABSTRACT BODY:

Introduction

The overall prognosis for canine transmissible venereal tumor is generally considered to be very good to excellent. A large number of immunocompetent dogs may spontaneously regress; in turn, the vast majority of dogs treated with vincristine sulfate alone or conservative radiation protocols will respond completely and with long-lasting clinical remission.

The casuistry of transmissible venereal tumors refractory to chemotherapy treatment is very low, but when it occurs, resistance appears after 6 weeks of treatment with vincristine. Faced with this, doxorubicin at a dose of 30 mg/m2 IV, every 21 days, may be indicated, expecting positive results in the first two sessions.

General considerations

- The transmissible venereal tumor is transmitted horizontally between dogs, occasionally being observed in other canines such as foxes, jackals and coyotes. The controversies about the histological nature and transmission mechanisms of this neoplastic disease explain why it received countless names.

- It has a worldwide distribution , although its prevalence is higher in warmer climates and in enzootic areas, including the southern United States, Central and South America, certain parts of Africa, the Far East, the Middle East, and southeastern and Eastern Europe. In some regions it is the most frequent tumor by far; They are places where there is no adequate population control of dogs, there are a large number of dogs in urban areas and there are

vagrants wandering through their streets. These conditions make it possible for dogs to act as reservoirs of the disease.

- For some authors, it is more common in temperate regions and mainly in tropical ones. Both the increase in rainfall and the average annual temperature show a positive correlation with prevalence. Occasional cases are reported in regions free of transmissible venereal tumors, due to travel to endemic areas.

- Coitus is the classic mode of transmission, with free-living and sexually intact dogs of both sexes being the ones with the highest risk of developing this neoplasm. Biting and scratching could be also transfer mechanisms for the clones of the tumor cells.

- Primarily, the most frequent location of the tumor is the external genitalia, clinically characterized by a tumor formation on the mucosa of the penis and foreskin in males; and from the vulva and vestibule in the female.

- However, other sites may be affected, including the nasal and oral cavities, skin and the subcutaneous tissue, and the eyeballs.

- no sexual predisposition is observed, with the exception of the oronasal form, in which males (in 82% of all published cases) are affected more often than females

- Spontaneous regressions are observed in canine venereal tumors, both experimentally acquired and naturally acquired; Several studies have shown varied immune responses in different tumor stages. Therefore, it seems rational that immunosuppression of any nature is a risk factor for contagion and maintenance of the transmissible venereal tumor, and may also increase the rate of dissemination of the disease.

- This tumor would be an allograft that behaves like a parasite.

- It is transmitted directly between dogs through the downregulation major histocompatibility complex (MHC), killing B-cells, supressing NK and dendritic cells by transplantation of viable tumor cells on the surface of damaged mucosa, during sexual intercourse or other social habits such as sniffing and licking each other.

- This characteristic of the transmissible venereal tumor is the only one in which a naturally occurring tumor in dogs is transmitted as an allograft that behaves more or less like a parasite. It becomes independent from the original host to continue growing and surviving.

- A viral etiology is suspected and some authors describe type C particles; Others defend that transmission is only possible with free cell extracts, where viral particles have not been observed through electron microscopy.

Clinical signs

- single or multiple neoplasms in the form of papul, nodules, cauliflower from 2mm to more than 10 cm in diameter. Quickly begin to bleed and ulcerate.

- most often localized in the external genitalis, but can be located on the skin, mucous membranes, in the nose,conjunctiva.

lesions may be located deep in the vagina or caudally in the region of the bulbous glands of the penis, not visible at the first clinical examination. Hemorrhagic discharge can be mistaken for estrus in females or urethritis, prostatitis, cystitis in males.

- clinical lesions in the extragenital form of tvt will depend on the location:

in the case of the nasal form: sneezing, epistaxis, asymmetry of the muzzle, damage to the orbit and oral cavities, epiphora, halitosis, oronasal fistulas.

the cutaneous form may present as single or multiple nodular and cauliflower-like rapidly ulcerating lesions on the skin. Neurological manifestations were present in patients with tumor involvement in the brain or spinal cord.

- metastasis are not common, may disseminate in regional lymph nodes, but distant multiple metastasis can also be observed.

- tumors are often necrotic, with a secondary bacterial contamination, clinically manifested by serous-purulent discharge and an unpleasant odor.

- Large tumor volumes can cause obstruction and difficulty in urination, defecation.

- The general state of health in patients, even with large tumors, is often not changed.

Diagnostic:

- Anamnesis, signalment, signal clinical signs, cytology are important for diagnosis.

- Cytology is a quick and effective method for diagnostic tvt (FNA more effective as smears)

- Cytologically distinguish between lymphocytic, plasmocytic and mixed types.

- The lymphocytic subtype is characterized> 60% of the rounded cells with fine -grained cytoplasm containing several transparent vacuoles; centroblastic nuclei with a rough pattern of chromatin and 1-2 distinct nucleenus.

- The plasmocytic subtype consists of> 60% of ovoid cells with a more basophilic cytoplasm, several clear vacuoles and a more eccentric core.

- The mixed type contains both lymphocytoid and plasmocytoid cells, while not a single type exceeds 59% of the total number of cells

- The plasmocytic form is more often found, more aggressive, has a higher expression of P-glycoprotein, which can lead to accelerated excretion of cytostatics, and, as a result, chemotence.

- Figures of mitosis are often found.

- Diagnosis can be difficult in the case of extragenital forms of tvt due to the discrepancy between the anamnesis, localization and cytological features.

- Histological diagnosis when staining with hematoxin-eosin can be difficult in the differentiation of tvt and histiocytoma, due to similar morphological manifestations. The groups of rounded individual cells in a branching fibrovascular network can help in histological diagnosis of the tumor. In complex cases, molecular diagnostic in situ PCR of the rearranged LINE–c-myc gene sequence may be needed to determine TVT.

- Visual diagnostic methods are usually not used with the exception of the nasal form of tvt.

- CBC and biochemistry profiles are often without pathological changes. Mild anemia and leukocytosis may be present.

- Paranoplastic erythrocytosis in patients with a large tumor volume is described.

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TRANSMISSIBLE VENEREAL TUMOR CLINICAL SIGNS AND DIAGNOSIS

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dear colleagues,
Dr Martin Soberano send our main abstract earlier

ANESTHESIA FOR THE TRAUMA PATIENT

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ABSTRACT BODY:

Trauma is defined by the medical dictionary Merriam Webster as 'an injury (such as a wound) to living tissue caused by an extrinsic agent'. [1]

The physiological consequences of trauma are manifold leading to a metabolic and immunologic response in an attempt to restore homeostasis. Depending on the severity of trauma changes will be overt or more hidden. Centralization of blood flow by increase of systemic vascular resistance and heart rate (HR) will decrease peripheral tissue perfusion and gastro-intestinal blood flow. Systemic inflammatory response syndrome (SIRS), infection and multiple organ dysfunction can be consequences of severe trauma. [2]

While trauma patients need **immediate care**, which often include surgery and thus anesthesia, proper **stabilization** of the animal, taking of pertinent **history** and **examination** are of outmost importance. The initial examination and stabilization can follow the **ABC-D approach**:

Airway

Patent?

Protective reflexes intact?

Breathing

Listen for airflow / *Look* for respiratory movement & pattern / *Count* breaths / *Monitor* oxygenation (SPO2)

Circulation

Look for consciousness - color (mucous membranes) - capillary refill time - external bleeding

Listen: cardiac auscultation

Feel for peripheral pulses / *Monitor* blood pressure, electrocardiogram, temperature

Detect

Closely examine the patient for bruising and obvious & less visible **injuries**

Check for **neurologial function**: brain injury versus spinal cord injury; mentation – motor sensory function

Assess and treat **pain**

Additional diagnostics

- Blood work including biochemistry and complete blood count
- Blood gases
- As indicated
  - Electrocardiogram
  - Radiographs (chest!)
  - Ultrasound
  - Etc.

Complicating factors / considerations

Often times trauma patients are **not fasted** and exhibit a slower gastrointestinal transit time. This increases their risk of vomiting, regurge and *aspiration pneumonia*. A thorough **history** prior to stabilization is typically not possible due to the urgency of the situation. Similarly, the animal might need to be treated without full **diagnosis**. In general a **difficult airway** should be anticipated in trauma patients. This might be due to direct injury in the head / neck region, but might also be due to the fact that the animal cannot be positioned in the ideal way for intubation due to trauma.

The attempt to rush a traumatized animal immediately to the operating theatre is under most circumstances not indicated. However, assessment and **stabilization** needs to be performed in an efficient manner **without delay** in a **multidisciplinary team approach**.

Ideally **two large bore intravenous catheter** should be placed and in most severe trauma cases. An arterial catheter for monitoring of invasive arterial blood pressure and arterial blood gases is indicated, but venous access needs to be prioritized.

Shock is a common sequela of trauma patients resulting in inadequate tissue perfusion. Thus, the primary goal is to *optimize tissue perfusion and oxygen delivery to the tissues* prior to anesthesia. Unfortunately no clear end points in regards to hemodynamic data exist so far. There seems to be some evidence that slightly higher mean arterial pressures could improve outcome in critically ill animals underoing anesthesia. Yet, this might depend on the type of injury and/or shock.

The **type of shock** is important to recognize for choosing the correct treatment approach. For example hypovolemic shock requires fluid therapy and potentially pressores. Blood products will be indicated in hemorrhagic shock. Obstructive shock e.g. due to a

tension pneumothorax needs causative treatment, i.e. relieve of the tension pneumothorax. Neurogenic shock requieres an increase in heart rate and vasopressors. [3]

In case of **dysrhythmias**, most frequently of ventricular nature, fluid therapy including correction of electrolyte imbalances will preceed pharmacological treatment. The use of sodium channel blockers such as lidocaine for ventricular tachyarrhythmias or in case of supraventricular tachycardia the ultrashort acting beta-blocker esmolol or the calcium channel blocker diltiazem can be used.

Analgesia is an essential part of stabilization of the critical animal. Intravenous opioids can be titrated to effect while tightly monitoring response and vital signs. Loco-regional anesthesia should be considered whenever possible to avoid systemic side effects.

When choosing the **anesthetic protocol** one should consider the problem list of the individual animal and the potential complications related to surgery and anesthesia.

In general **anticholinergics** such as atropine or glycopyrrolate are rarely used considering preexisting tachycardia in many cases. An additonal increase in heart rate would lead to an increase in myocardial oxygen consumption which could be detrimental in the presence of ongoing malperfusion/ischemia. This does not preclude the use of anticholinergics in bradycardic animals, e.g. due to high doses of μ-Opioid agonist or in neurogenic shock.

Anti-emetic drugs such as maropitant seem sensible, however, one should remember their slow onset of action, i.e. approximately one hour independent of route given (subcutaneous or intravenous).

Opioids, in particular fentanyl, are commonly used in trauma cases as they provide good analgesia, have sedative effects and decrease the amount of inhalant anesthetic needed while having lesser cardiopulmonary side effects compared to most induction drugs or inhalant anesthetics. *Sedatives* like the long acting phenothiazine acepromazine and alpha-2-agonists like (dex-)medetomidine are usually not indicated due to their cardiovascular side effects. Instead, benzodiazepines such as midazolam or diazepam can be used.

Propofol is a short acting anesthetic providing rapid and smooth induction. It is not indicated in hemorrhagic shock due to its negative inotrope and chronotrope effect, and causing of vasodilation. In compensated shock propofol administration can lead to decompensation. A bruised myocardium might respond to propofol with sustained ventricular dyshrythmia. However, in cases of brain injury, propofol might be the induction agent of choice as it decreases ICP. Similarly, in other trauma patients that have been sufficiently stabilized propofol might be a decent induction agent.

Etomidate (plus benzodiazepines) has very little cardiovascular side effects. The use of etomidate in trauma patients is controversial as it causes a transient depression (approximately 4-6 hours) of the adrenal cortex. Some retrospective studies in human medicine found no difference between etomidate and ketamine in mortality rates[4], while others found a higher mortality with etomidate compared to propofol[5]. Yet, etomidate was mainly used directly in the emergency room, while propofol was used in the operating theatre. This could potentially induce a bias as patiens induced in the OR might have been more stable compared to the once in the ER. [5]

Ketamine (plus benzodiazepines or propofol) provides some analgesia and hypnosis, is a positive chronotrope and mild negative inotrope. It's use in neurologial animals is. Neurological exams are less hampered in animals that received ketamine compared to propofol[6]. There is evidence of ketamine having a neuro-protective function. However, the scenarios under which this holds true versus not are complex, making a general endorsement in these cases difficult. [7]

A **combination** of fentanyl and benzodiazepines +/- lidocaine is another viable options to induce anesthesia in compromised trauma patients.

For **maintenance** of anesthesia a balanced anesthesia approach in these cases is imperative. Partial intravenous anesthesia can help to avoid the need of higher concentrations of inhalant anesthetics which are typically not well tolerated considering their vasodilatory property and negative intropy.

In summary, a systematic, multidisciplinary approach to the acute trauma patient is key to success. Swift stabilization prior to anesthesia will reduce the risks and improve outcome.

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099 / #114

RESOLUCIÓN DE LESIONES MÁS COMUNES DE LA COLUMNA VERTEBRAL DEL PERRO Y EL GATO

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ABSTRACT BODY:

- Hernia discal espinal

Es una patología muy común en los perros y no tanto en gatos que tiene como precursora a la degeneración del disco intervertebral. Este disco degenerado tiene una mayor susceptibilidad a herniarse y comprimir/contusionar el cordón medular produciendo alteraciones neurológicas medulares y/o dolor. El método diagnóstico ideal para las hernias discales es el estudio de resonancia magnética, además a través de este examen se han podido reconocer 6 tipos de hernia discales: extrusión discal, protrusión discal, extrusión de núcleo pulposo hidratado, extrusión aguda no compresiva del núcleo pulposo, extrusión discal intradural/intramedular y embolismo medular fibrocartilaginoso. Cada uno de estos tipos de hernia pueden acompañar diferentes tipos de presentación y curso clínico. El tratamiento puede ser conservador usando farmacos paliativos para el dolor y fisioterapia para rehabilitar la función neurológica o puede ser quirúrgico para remover la hernia discal y descomprimir el cordón medular.

- Discoespondilitis bacteriana

Colonias bacterianas pueden trasladarse desde otros tejidos a través de la circulación y anecdóticamente llegar al disco intervertebral y a las epífisis de los cuerpos vertebrales adyacentes. La clínica de estos pacientes generalmente se caracteriza por dolor espinal y en algunas ocasiones déficits neurológicos. El acercamiento diagnóstico es posible a través de hallazgos en imágenes radiográficas, por tomografía computarizada y aun con mayor detalle utilizando resonancia magnética. El tratamiento es en base a antibioticoterapia prolongada (4 a 6 semanas) y en algunas ocasiones se necesitará hacer una biopsia del tejido para cultivo/antibiograma.

- Neoplasias espinales

Se pueden presentar en diferentes zonas de la columna vertebral: cervical, torácica, lumbar y sacro. Pueden encontrarse en posición extradural, intradural/extramedular e intramedular. En mi práctica privada a través de cirugía para biopsia y/o citoreducción e histopatología he encontrado diferentes tipos de neoplasias como, por ejemplo: meningiomas, mastocitomas, osteosarcoma vertebral, oligodendroglioma, nefroblastoma, leiomiosarcoma entre otros.

- Inestabilidad atlantoaxial

Suele deberse a la falta o malformación congénita de estructuras de la unión atlantoaxial, por lo que habitualmente esta subluxación vertebral se da en pacientes caninos jóvenes y mayormente en razas toy como chihuahuas, yorkshire, poodle, etc. Los signos clínicos suelen ser agudos y progresivos: dolor cervical y tetraparesia/plejía. El diagnóstico es a través de imágenes radiográficas, tomografía computada y/o resonancia magnética. Obteniéndose más detalle y datos diagnósticos con las 2 últimas. El tratamiento paliativo es a base de fármacos analgésicos y antiinflamatorios y acompañado idealmente con cirugía utilizando poliimplantes y PMMA para reducir y estabilizar la subluxación.

- Tromboembolismo aórtico e infarto medular espinal en gatos

Son 2 patologías vasculares que traen alteraciones neurológicas y con frecuente presentación en gatos.

En el caso del tromboembolismo aórtico lo que sucede es que un tromboembolo habitualmente, pero no siempre, originado en el ventrículo izquierdo del corazón se traslada por la circulación hasta llegar a la bifurcación de la aorta abdominal donde podría obstruir la o las arterias iliacas. La obstrucción genera la parálisis de los miembros posteriores, perdida de pulso femoral y perdida de temperatura del tren posterior. El diagnóstico se puede hacer a través de una ecografía Doppler abdominal y el tratamiento consiste en analgésicos y anticoagulantes. El pronóstico suele ser de reservado a malo.

Sobre el infarto medular espinal, es una patología de presentación aguda donde la irrigación de un segmento medular queda interrumpida y provoca signos clínicos de lesión medular sin acompañamiento de dolor. Actualmente se considera un hecho impredecible y no estaría asociado a otras patologías metabólicas o cardiovasculares. El diagnóstico se realiza a través de resonancia magnética. El tratamiento se basa en fisioterapia/rehabilitación y el pronóstico es reservado, aunque podría ser bueno siempre y cuando se mantenga intacta la sensibilidad profunda de las extremidades posteriores.

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MANAGEMENT OF HEAT STROKE

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ABSTRACT BODY:

Heatstroke is a life-threatening condition that can be identified in people, dogs and other species. Heatstroke is caused by the inability to dissipate accumulated heat after strenuous exercise (exertional heatstroke) or exposure to a hot and humid environment (classical heatstroke). This is characterized in humans by a core body temperature above 40°C (104° F) and above 41°C (105.6°F) in dogs with evidence of neurological dysfunction (collapse, disorientation, seizures, and coma), that ultimately causes direct hyperthermal injury to tissues and eventually multi-organ dysfunction. The lack of elevated body temperature at presentation to the emergency room should not rule out the possibility of heat stroke in a patient with acute neurological signs and exposure to high environmental temperatures, as owners may cool down their pets with cold water baths, and some dogs with advanced stages of shock may present hypothermic, a factor that has been correlated with poor outcomes. There are multiple factors that can increase the risk of heatstroke in dogs including: obesity, brachiocephalic airways, larger breeds with body weight higher than 15 kg and more specifically retrievers, elevated temperature and humidity, and lack of acclimation. Heat stroke in cats is not commonly reported, in the author's experience, cats most commonly develop heat stroke after being accidentally locked inside a dryer machine.

Several hematological abnormalities are found during initial bloodwork evaluation. One of the major consequences of heat stroke is disseminated intravascular coagulation. Due to the damage to the endothelium and activation of the platelets, thrombocytopenia is commonly identified in hemogram due to platelet aggregation and microthrombosis, along with sequestration in the spleen. In one study, 84% of dogs with heatstroke developed thrombocytopenia, but only 50% of those dogs had systemic evidence of DIC, therefore the presence of thrombocytopenia is not always correlated to the development of DIC. Due to the consumption of circulating coagulation factors, along with liver damage and dysfunction, coagulation times are usually prolonged in patients with heat stroke, along with decreased protein C activity, increased D-dimers and low antithrombin concentrations. Another common finding is hemoconcentration (increased PCV) and hemoglobin concentrations, along with increased numbers of nucleated red blood cells. These findings are secondary to significant free water loss and direct damage to the bone marrow.

Biochemical abnormalities are also commonly identified in dogs with heatstroke. Direct liver injury may progress into cholestasis and liver failure. This is manifested by elevation in ALT, AST, ALP and GGT. Hypoalbuminemia usually develops during hospitalization due to translocation, loss, and excessive fluid administration. Hypoglycemia at presentation is also a common finding in dogs with heat stroke and may play a role in progressive neurological dysfunction. [10,14]. One of the most common complications of heatstroke is acute kidney injury (AKI). The mechanism for the development of AKI following a heatstroke event in dogs is multifactorial and results from direct renal thermal injury, dehydration causing hemoconcentration, hypoxia, endotoxemia and release of cytokines, disseminated intravascular coagulation (DIC), decreased renal blood flow, rhabdomyolysis and secondary myoglobinuria. The lack of clinically available biomarkers to detect early renal injury prevents early initiation of renal protective strategies that may decrease the progression of renal dysfunction during hospitalization. One study evaluating acute kidney injury in heat stroke patients at a veterinary hospital found that those dogs that did develop AKI had significantly lower systolic blood pressures, higher shock index but lower baseline lactate and lower platelet count than those that did not develop AKI. In fact, this study found that increases of 10 mmHg of systolic blood pressure decreased the odds of developing AKI by 27%. Rhabdomyolysis is caused by direct heat damage to myocardial and skeletal muscle, along with decreased perfusion and microthrombosis, creatinine kinase elevation is directly correlated with the degree of muscle damage.

Treatment of heat stroke:

Treatment of heat stroke is largely supportive. Active cooling and fluid resuscitation are the two main components to preventing ongoing cellular and tissue damage during the initial stage of treament. It is recommended to perform cooling of patients with lukewarm water and fans until a temperature of 103.5 F is reached, as their bodies will often continue to cool beyond that. The use of ice-baths, alcohol application of paw pads and use of extremely cold water is not recommended as it will cause peripheral

vasoconstriction and decreased heat release, increasing the body core temperature. Patients with dense haircoats, may require shaving of the hair to allow for better heat dissipation. Oxygen should be supplemented with facemasks during tirage, and in patients with evident stridor and swelling of their upper airway, endotracheal intubation is mandatory to take control of the airway. Dextrose should be administered to patients with evidence of hypoglycemia at presentation, and glucose levels should be monitored at least every 6 hours. An initial bolus of dextrose of 0.5-1 ml/kg of 50% dextrose (diluted 1 to 5) can be administered during triage, then followed with 1.25-2.5% dextrose added to crystalloid fluids.

Dogs with heatstroke may present to the emergency room with seizure activity, and seizures should be immediately managed. The use of benzodiazepines, Midazolam or diazepam, at doses of 0.25-0.5 mg/kg IV bolus is recommended for short term management of seizure activity. For patients in status epilepticus and those that develop more seizures while in hospital, it is recommended to start anticonvulsant therapy with Levetiracetam (Keppra). A loading dose of 60 mg/kg is given IV initially, followed by 20 mg/kg IV or orally every 8 hours thereafter. It is important to rule out hypoglycemia as the cause of seizures before starting long-acting anticonvulsants. The use of phenobarbital is not recommended due the risk of liver failure in patients with heat stroke.

Fluid resuscitation with ¼ shock doses (10 ml/kg dose for a cat and 15-20 ml/kg dose for a dog) of a balanced replacement fluid such as LRS, Normosol-R, or Plasmalyte are recommended until normotension is achieved. The use of 0.9% saline solution and synthetic colloids is contraindicated in patients with heatstroke due to the high risk of AKI. The remainder fluid deficits can be corrected more slowly during the rehydration process with the same balanced replacement fluid solution. In patients with evidence of coagulopathies (prolonged PT/PTT), the early use of plasma products is recommended. Fresh frozen plasma can be administered at doses of 10-20 ml/kg and should be incorporated in the fluid plan and balance of the patient. The use of plasma products is thought to decrease glycocalyx damage. Due to the damage to the glycocalyx and vascular endothelium, these patients are at greater risk of fluid overload and interstitial edema. Conservative fluid regimens are recommended, and if hypotension is persistent, the early use of vasopressors such as norepinephrine or vasopressin is warranted. Progression of AKI to anuria, fluid overload, and hyperkalemia necessitates hemodialysis. In this author's experience, AKI patients secondary to heat stroke requiring dialysis have an overall good prognosis, though this would be affected by other organ failures.

Animals with heat stroke may present with severe gastrointestinal disease: vomiting, regurgitation, diarrhea, hematochezia and severe ileus. The use of antimicrobials is only warranted in severe cases where bacterial translocation is suspected. Broad spectrum antibiotics such as Ampicillin-sulbactam (30-50 mg/kg IV every 8 hours), is recommended to provide broad spectrum antimicrobial therapy against Gram-positive, Gram-negative and anerobic bacteria. The use of antiemetic and prokinetic therapy is highly recommended to prevent aspiration pneumonia secondary to vomiting and regurgitation, especially in dogs with severe neurological dysfunction and increased risk of aspiration. Antiemetic therapy includes maropitant (1 mg/kg IV q 24 hours), metoclopramide (2 mg/kg/day IV CRI) and/or ondansetron (0.5 mg/kg IV q 8 hours). Finally, the use of gastroprotectants in critical illness is still controversial, common practice is to provide gastric acid reducers such as Pantoprazole or Famotidine (1 mg/kg IV q 12 hours), the author recommends such therapies to patients with signs of GI ulceration such hematemesis, melena or such. Early enteral nutrition is imperative for faster recovery, once coagulopathies are controlled the use of nasogastric tubes is recommended during hospitalization to provide nutrition.

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PRE-BREEDING HEALTH EXAMINATIONS AND GENETIC SCREENING TO IMPROVE DOG AND CAT HEALTH

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ABSTRACT BODY:

Domestic dogs and cats are contrived animals, created by man through selective breeding. Many are further manipulated into breeds. Artificially selected domestic animals are not under the pressure of natural selection where only the healthiest thrive and reproduce. Therefore, health is not a given when it comes to cats and dogs. Artificial selection only produces what is selected for or against. If health is not purposefully selected for then health deteriorates. This is additionally true if selection is applied towards disease predisposing anatomy, such as excessive size (large and small), excessive skin, excessive angulation, excessive hair, excessive skin folds, and extreme brachycephaly. If healthy parents are not purposefully selected for, then there can be no expectation of producing healthy offspring.

There are many fallacies about health when it comes to domestic dog and cat breeding. Some feel that crossbreeding (to other breeds) or outbreeding (to those in a breed that are the least related) will produce healthier offspring. However published data show that common genetic disease occurs at similar frequencies in mixed-breed and purebred (pedigreed) animals. Differences in disease frequencies can also have to do with predisposing conformations, such as patella luxation and mitral valvular disease in small statured dogs. Ninety-five percent of cats presented to veterinary clinics are random-bred and not pedigreed cats. However, the common genetic diseases including inflammatory bladder disease, diabetes mellitus, allergies, hypertrophic cardiomyopathy, and inflammatory GI disease occur regularly in practice.

There needs to be a paradigm shift in the purposeful breeding of cats and dogs – whether pedigreed, designer-bred, or mixed/random-bred. Health-focused breeding is the only way to improve their health. It is no longer ethical to purposely breed two animals together without regard to their health. Pre-breeding health examinations need to be as commonplace as pre-purchase examinations in horses. This is simple health quality control.

With the rapid evolution of commercial genetic testing and multiplex genetic panels in dogs and cats, an animal can be tested for over 200 known disease-related variants. However, for each individual it is likely that none or only a few of those genetic markers relate to disease risk. Almost all disease-related markers in panel testing relate to breed-specific disease. Having panel test results that state that none of the 200+ disease related gene variants are present in an individual provides a false sense of health, rather than an actual measure of the individual's health status and disease risk.

A pre-breeding health examination must begin with a medical history review. If the cat or dog has not been a long-term patient, then its prior medical history should be acquired from the previous veterinarian. The medical history should be reviewed for evidence of genetic disease that cannot be tested or screened for. This includes a history of allergies, seizures, bloat, inflammatory bladder disease, inflammatory bowel disease, cancers, bleeding disorders, bladder stones, etc. It should be noted how far back there is a documented medical history.

Pre-breeding health examinations begin with a general physical examination of the dermatologic, cardiovascular, musculoskeletal, ophthalmologic, and gastrointestinal systems. If breed or conformational risk factors are present, then examinations should be performed for specific diseases at risk. These may include hip and elbow radiographs, patella examinations, auscultation, ECHO, or EKG exams, and specific blood work. Validated DNA tests for specific risk variants should be performed based on breed or conformation.

Many kennel or cat registries list appropriate breed-specific health tests. In the USA, the parent breed clubs list the required pre-breeding health assessments on the OFA-Canine Health Information Center (CHIC) website: https://ofa.org/chic-programs/browse-by-breed/. For cat, similar list are not available, but the International Cat Care (iCat) website lists breed specific diseases and their available tests: https://icatcare.org/advice/?per_page=12&categories=cat-breeds. Caution must be raised on running DNA tests for common variants that may not imply risk to all individuals. These include the SOD1 variant for degenerative myelopathy and CRD4/cord1 variant for late onset blindness in dogs, and the pyruvate kinase variant in cats.

If owners are instructed to ask for documentation of pre-breeding health examinations including genetic screening and genetic testing of parents of kittens and

puppies, then health-focused breeding will increasingly become the standard in dog and cat breeding. Health is not automatic or expected. It must be selected for.

THE ROLE OF IMAGING IN ADRENAL DISEASE

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THE ROLE OF IMAGING IN ADRENAL DISEASE

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Ultrasonography is currently the main screening imaging modality to assess the adrenal glands in dogs and cats. However, there are certain indications where advanced imaging such as computed tomography (CT) is indicated, for example assessment of adrenal masses for surgical planning.

Knowledge of anatomic landmarks to identify the adrenal glands is essential for any imaging modality, but particularly ultrasound. The left adrenal gland is located at the craniomedial aspect of the left kidney, lateral to the abdominal aorta. The adrenal gland is located cranial to the left renal artery and caudal to the origin of the cranial mesenteric and celiac arteries. The left phrenicoabdominal vein can be seen crossing the adrenal gland when imaging with high-resolution transducers, and/or Doppler imaging. It is important to follow the vascular landmarks to identify the adrenal gland and not the kidney alone, as the position of the kidney and length of the renal vasculature is variable. The right adrenal gland is more cranially located than the left and can be more difficult to identify. It is located along the lateral aspect of the caudal vena cava craniomedial to the cranial pole of the right kidney. The right adrenal gland is at the level of or slightly cranial to the cranial mesenteric and celiac arteries. Both the left and right adrenal gland in cats are oval or bean shaped and usually mildly hypoechoic relative to the surrounding fat. In dogs, the shape of the adrenal glands varies. The left adrenal gland is typically bilobed (peanut, dumbbell, or biconcave disc shape) but can be slightly flattened with angulation (lawn-chair shape) particularly in large breed dogs. The right adrenal gland in dogs is V-shaped or is also described as having the shape of an arrow. In dogs the adrenal glands should normally be iso-or hypoechoic to the surrounding fat. Occasionally the adrenal cortex and medulla can be differentiated, given the adrenal gland a striated appearance. Normal ultrasonographic adrenal gland size is detailed in table 1.

Table 1: Normal adrenal gland dimensions

Signalment	Caudal pole thickness
Cats	0.43 +/-0.03 cm
Dogs <10kg	</= 0.54 cm
Dogs 10-30kg	</= 0.68 cm
Dogs >30kg	</= 0.8 cm

The measurements in table 1 illustrates that there are no reliable lower size values for adrenals. Estimation of reduced renal size is therefore somewhat subjective and there is overlap between small and normal adrenal gland size. Smaller adrenal in size may be seen in dogs and cats with spontaneous (Addison's) or iatrogenic hyperadrenocorticism. Reduced size may occasionally be seen in patients with unilateral functional adrenal tumors resulting in atrophy of the contralateral gland. However, these changes are often subtle and due to the range of normal measurements and lack of a lower limit of normal diameter this is often not definitively identified.

Identification of enlarged adrenal glands is the more common scenario in dogs and cats. Patients with pituitary-dependent hyperadrenocorticism may have normal or bilaterally enlarged adrenal glands that retain a normal shape. Nodular changes as with nodular hyperplasia may be noted as well. Additional ultrasonographic findings associated with hyperadrenocorticism include enlarged liver size with rounded liver margins and diffuse parenchymal hyperechogenicity. Not uncommonly, hypoechoic liver nodules representing nodular regeneration are identified simultaneously. Additional findings may include multifocal splenic mineralizations, urinary calculi and renal mineralizations. Increased echogenicity of the pancreas has also been reported to be associated with endocrinopathies. Differential diagnosis for hypoechoic or hyperechoic nodules in the adrenal glands include both benign and malignant changes such as nodular hyperplasia, myelolipoma, adenoma, or early carcinoma or pheochromocytoma. Benign adenomas can occur unilaterally or bilaterally and may be functional or nonfunctional.

Malignant adrenal tumors include adenocarcinoma and pheochromocytoma. Adrenal myelolipoma creating a mass like appearance with composition of fat and hematopoietic cells has been reported in dogs. Presence of an adrenal mass larger than

2 cm is usually associated with malignant disease. It is important to note that approximately 10% of patients have bilateral tumors. It is also possible for adenocarcinoma and pheochromocytoma to occur simultaneously in the same patient. Mineralization of an adrenal mass is more commonly seen with adenocarcinoma and adenoma, however can be observed in pheochromocytoma is as well. Mineralization of adrenal glands of normal size and cats can be in age-related and is not associated with clinical significance. Adrenal tumors are uncommon in cats. A portion of adrenal tumors in cats can be aldosterone secreting (Aldosteronoma), resulting in Conn's syndrome. Presence of an adrenal mass should prompt investigation of presence of vascular invasion. Both adenocarcinomas and pheochromocytoma is have the potential to invade the adjacent vasculature, most commonly the phrenicoabdominal vein and subsequently the caudal vena cava. Infiltration of the renal vasculature may be present as well. Careful examination including Doppler or contrast-enhanced CT is warranted in these cases particularly if surgical removal will be attempted. Contrast-enhanced ultrasound has been described as being helpful for differentiating between different types of adrenal tumors in dogs, however it is not widely available.

Computed tomography is often used for surgical planning and staging of dogs with adrenal tumors. In people, CT is often used to characterize and differentiate adrenal neoplasms, as well as detect early vascular invasion. Overlap of pathologic features between different tumor types in dogs hinders the ability of CT to differentiate different tumor types. However, it is of excellent method to detect signs of vascular invasion. The peripheral contrast enhancing rim in the late post-contrast CT images has been described to be associated with a fibrous capsule formation of a tumor. CT is particularly helpful in patients presenting with acute abdominal signs and retroperitoneal hemorrhage. Differential diagnoses for retroperitoneal hemorrhage include trauma, ruptured renal tumor, retroperitoneal hemangiosarcoma, and ruptured adrenal mass. These conditions can be difficult to differentiate using ultrasound as hemorrhage is causing a mass effect that may incorporate both kidneys and adrenal glands which makes it difficult to identify the origin of the lesion. Both a normal adrenal gland and an abnormal ruptured adrenal gland may be difficult to identify within the heterogeneous mass lesion. Contrast-enhanced CT allows identification of the supplying vasculature and allows an overview of location, size and involvement of the retroperitoneal structures. Incidental discovery of an adrenal mass is not uncommon when performing abdominal ultrasound, abdominal or spinal CT and MRI. Given the potential for clinically silent neoplastic lesions, further workup is warranted in these patients.

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GERIATRÍA FELINA: MANEJO DE ENFERMEDADES CONCURRENTES

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ABSTRACT BODY:

Las comorbilidades son comunes en los gatos geriátricos al igual que en medicina humana.

Las razones de la alta frecuencia de comorbilidades a medida que los gatos envejecen siguen sin saberse, pero probablemente se relaciona con una combinación de estrés exógeno que causa lesiones oxidativas, agentes infecciosos y el desequilibrio del sistema inmunitario. A medida que la homeostasis del sistema inmunitario falla, se reducen las defensas frente a los patógenos externos y se producen cambios de "envejecimiento por inflamación".

El concepto de fragilidad ayuda a entender las condiciones complejas que pueden coexistir en el gato.

El concepto de un "modelo de prevención de la fragilidad" define tres niveles de intervención:

1) Minimizar los factores de riesgo y la aparición de la enfermedad

2) Retrasa la progresión de la enfermedad

3) Reducir o limitar las complicaciones que producen la enfermedad.

Cuando los gatos llegan a edad geriátrica, los dos primeros niveles ya han quedado atrás. El enfoque ahora está en la tercera categoría de reducción y limitación del deterioro.

El objetivo de un envejecimiento saludable es la calidad de vida de nuestro paciente. Aunque el envejecimiento saludable significa diferentes cosas para diferentes personas con respecto a ellos mismos y a sus gatos, y la perspectiva de un envejecimiento saludable a menudo cambia con el tiempo y las circunstancias, es probable que la comodidad y el disfrute de la vida sean objetivos comunes. También es importante reconocer que las expectativas del dueño para un gato reflejarán la propia actitud del dueño hacia el envejecimiento saludable.

A medida que los gatos envejecen, aumenta el riesgo de desarrollar más de una enfermedad, lo que a menudo complica el diagnóstico y el tratamiento. El diagnóstico se ve facilitado por una combinación de un examen físico completo, pruebas diagnósticas de detección y atención a las comorbilidades comunes.

Problemas que surgen en los gatos mayores con comorbilidades en el diagnóstico:

El efecto de la "polifarmacia" y el riesgo de interacciones medicamentosas.

El aclaramiento renal o hepático disminuido puede requerir menos dosis de fármacos o mayores intervalos de medicación.

El incumplimiento del plan terapéutico por parte del cuidador, particularmente cuando hay una gran carga de medicamentos, puede afectar negativamente el vínculo gato-cuidador.

El impacto acumulativo de múltiples enfermedades: CKD, DJD, DM, IBD, CDS y problemas de comportamiento, pueden agravar la eliminación inapropiada.

Hay situaciones clínicas, como la hipertensión, que pueden aparecer en varias enfermedades como hipertiroidismo, enfermedad cardiaca o renal.

Existe el riesgo de diagnosticar una enfermedad mientras se pasa por alto otra, o los signos clínicos corresponden a varias enfermedades como:

– Enfermedad inflamatoria digestiva con colangitis, enfermedad inflamatoria intestinal y pancreatitis concurrente.

– La pancreatitis crónica puede pasar desapercibida en un paciente diabético.

– El hipertiroidismo puede pasar desapercibido en los gatos con enfermedad renal grave, hepática o cáncer porque los signos típicos están enmascarados y T4 total puede suprimirse o estar solo en rango alto.

– El hipertiroidismo puede pasar desapercibido en gatos con DM debido a la similitud de los signos.

– El diagnóstico de bacteriuria en gatos con enfermedad renal, hipertiroidismo o diabetes puede ser complicado porque no siempre están presentes signos de enfermedad del tracto urinario inferior, piuria y/o sedimento urinario activo. El diagnóstico solo puede confirmarse mediante la realización de un análisis de orina y un cultivo bacteriano

– El hipertiroidismo y la enfermedad cardíaca pueden presentarse de forma conjunta.

– La mayoría de las enfermedades tienen un componente de dolor y debemos evaluación de otras fuentes de dolor en el gato mayor, como DJD, dolor de tejido blando o neurológico, utilizando escalas de dolor estandarizadas. El examen miofascial ayuda a detectar dolor en casa porque en ocasiones el estrés

de la consulta en la clínica no permite evaluar de forma adecuada.

Identificación de los objetivos terapéuticos:

La base de cualquier plan de tratamiento es lograr la hidratación del paciente, la nutrición adecuada, analgesia y satisfacer las necesidades ambientales y de comportamiento para que desarrolle su etograma.

La complicidad y el entendimiento con el cuidador es la clave para establecer el plan terapéutico que debe ser revisado y consensuado periódicamente, haciendo un acompañamiento eficaz en esta fase de la vida del gato en la que no curamos la enfermedad sino que el objetivo es proporcionar calidad de vida.

- Enfermedad renal crónica

Optimizar la hidratación, apetito y aumentar peso. La dieta renal que controle los niveles séricos de fósforo y minimizar los episodios urémicos, controlar la hipertensión, la proteinuria y por lo tanto mejorar la calidad de vida.

Los estimulantes de apetito como la mirtazapina transdérmica, antieméticos como el maropitant, aumento de hidratación con dieta húmeda o fluidoterapia subcutánea mejoran la calidad de vida.

- Hipertensión Arterial Sistémica:

La hipertensión es una enfermedad común en los gatos, en su mayoría secundaria a otras enfermedades (por ejemplo, enfermedad renal crónica, hipertiroidismo, hiperaldosteronismo), pero también se produce como un problema primario (hipertensión esencial). Debido a los efectos de la hipertensión no tratada en los órganos diana altamente vasculares (es decir, los ojos, el cerebro, los riñones y el sistema cardiovascular), es muy importante detectar elevaciones de la presión arterial temprano para evitar daños permanentes.

Hasta el 65 % de los gatos con ERC son hipertensos. No hay una relación lineal entre el grado de gravedad de ERC y la presencia o gravedad de la hipertensión. La hipertensión es menos común en gatos con hipertiroidismo (aproximadamente el 10-23 % en el momento del diagnóstico), pero aproximadamente el 25 % de los gatos desarrollan hipertensión después del inicio de la terapia o incluso una vez que se ha logrado el eutiroidismo. La diabetes mellitus está asociada con la hipertensión en humanos, pero esto no parece ocurrir en gatos.

Reducir el riesgo de daño a los órganos diana (TOD) que puede afectar a la supervivencia. Los tratamientos deben ser palatables y seguros para no producir complicaciones. Se deben realizar controles periódicos para ajustar la medicación.

- Hipertiroidismo:

La enfermedad renal es bastante común en gatos hipertiroideos no tratados. La disminución de la TFG se estabiliza en aproximadamente cuatro semanas post tratamiento y se puede valorar el estadío IRIS en el que se encuentra el gato.

El enfoque práctico para un paciente con hipertiroidismo y ERC simultáneos es tratar médicamente hasta que el T4 sérico se controle adecuadamente, momento en el que se puede predecir el efecto de la terapia permanente. Hay que tener especial cuidado con evitar el hipotiroidismo iatrogénico porque puede empeorar la azotemia y disminución de la supervivencia.

- Diabetes mellitus:

Regular los niveles de glucosa en sangre a través de la terapia con insulina y la dieta para reducir la toxicidad de la glucosa. Los signos clínicos de polifagia, polidipsia, poliuria y pérdida de peso tienen que mantenerse controlados.

- Enfermedad degenerativa articular DJD:

Controlar el dolor, aumentar la movilidad, facilitar el acceso a los recursos

a través de medicamentos como antiinflamatorios no esteroideos (AINE), opiáceos, gabapentina o anticuerpos monoclonales. Se debe realizar un seguimiento con vídeos valorando los cambios de comportamiento y evitar efectos secundarios de los medicamentos.

Modificaciones ambientales son importantes en los gatos mayores: el recorte regular de uñas, las rampas y facilitar el paso hacia los lugares favoritos para dormir, superficies suaves y acolchados, elevar cuencos de comida y agua puede ayudar al gato con cambios vertebrales cervicales. Añadir una bandeja para reducir la distancia entre las cajas, bordes de la bandeja bajos, abiertas y con limpieza regular, favorecerán su uso y evitará las micciones y defecaciones fuera de sitio.

Una de las mejores formas de detección precoz de enfermedades de los gatos mayores es realizar un control periódico por parte del cuidador del peso y de la masa muscular. La pérdida de peso comienza tres años antes de que se haga el diagnóstico de ERC.

El PLAN NUTRICIONAL debe tener en cuenta el peso corporal, la condición corporal (BCS) y el Índice de Masa Muscular (MCS).

Se recomienda una intervención nutricional temprana si un paciente hipo o anoréxico durante > 3-5 días.

-La intervención incluye garantizar un entorno adecuado y de bajo estrés y el uso de estimulantes del apetito y la colocación de sondas de alimentación a

medio plazo, como las sondas de esofagostomía.

El objetivo final será proporcionar calidad de vida y bienestar.

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LEISHMANIOSIS FELINA

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ABSTRACT BODY:

Hasta hace pocos años el gato se consideraba una especie resistente a la leishmaniosis. Se pensaba que, aunque se infectaba, era muy raro que desarrollara lesiones o signos clínicos. Ahora sabemos que esto no es cierto. Aunque con mucha menos frecuencia que los perros, los gatos enferman de leishmaniosis. La enfermedad, es más pleomórfica que en el perro y mucho menos conocida y por ello es mucho más difícil de diagnosticar, tratar y prevenir. En esta charla presentaremos la información esencial para el veterinario clínico sobre la leishmaniosis felina.

¿Cual es la etiología de la leishmaniosis felina?

Aunque se ha demostrado que seis especies del Gº Leishmania pueden infectar al gato, la más importante es *L. infantum*, que es la que causa la leishmaniosis felina en Europa (España, Portugal, Francia, Italia; entre otros países) y en América Latina (Brasil). Aunque no hay estudios específicos sobre el tema, se asume que la principal vía de transmisión es la picadura de dípteros del Gº Phlebotomus (Europa) o Lutzomia (América Latina). No se puede descartar que ocasionalmente se pueda transmitir por otras vías (vertical, heridas, transfusiones, venérea), pero sin duda son trasmisiones excepcionales.

¿Cuál es la prevalencia de la infección y de la enfermedad?

Los estudios de seroprevalencia muestran que en zonas endémicas (cuenca mediterránea, por ejemplo), la seroprevalencia oscila entre 1% y 25%, dependiendo de la zona. No hay información precisa sobre la incidencia de la enfermedad. Desde luego, parece ser poco frecuente, mucho menos que la leishmaniosis canina. Se postula que los gatos son, por lo tanto, más resistentes que los perros a la infección por *L. infantum*, pero no se puede excluir que la enfermedad siga estando infradiagnosticada. La mayoría de los casos reportados proceden de zonas endémicas europeas y mediterráneas, donde el número de gatos de compañía es mayor. En las últimas tres décadas se han notificado más de un centenar de casos clínicos en Europa (Italia, España, Francia, Portugal) con algunos casos diagnosticados en otros países (Suiza), en gatos importados de regiones endémicas. Se piensa que hay factores del huésped que predisponen a la susceptibilidad, ya que aproximadamente la mitad de los casos clínicos notificados se han observado en gatos que podrían haber tenido un sistema inmunitario deteriorado a causa de una infección por el virus de la inmunodeficiencia felina o por el virus de la leucemia felina, por terapias inmunosupresoras o por enfermedades concomitantes graves (neoplasia maligna).

¿Cuándo se debe sospechar clínicamente que un gato sufre leishmaniosis?

El cuadro clínico es bastante pleomórfico. Lo más frecuente es la aparición de lesiones cutáneas o mucocutáneas (nódulos, úlceras), en especial en la cabeza, junto con linfadenomegalia. Se estima que más del 50% de gatos con leishmaniosis presentan lesiones cutáneas. Menos frecuentes son las lesiones oculares (blefaritis, queratoconjuntivitis, uveítis), las lesiones orales, anorexia, caquexia, y enfermedad renal. Las alteraciones clínico-patológicas más frecuentes son la hipergammaglobulinemia, la anemia no regenerativa y la proteinuria.

¿Cómo se puede confirmar el diagnóstico?

En gatos que presenten signos clínicos o anomalías clinicopatológicas compatibles con la leishmaniosis y vivan o hayan visitado en zonas endémicas, para confirmar el diagnóstico de leishmaniosis debe realizarse una prueba serológica cuantitativa. Sin embargo, en caso de títulos de anticuerpos negativos o positivos bajos, debe utilizarse una técnica parasitológica para confirmar la existencia de infección activa (citología, histología, inmunohistoquímica, PCR), antes de emitir el diagnóstico.

¿Cual es mejor tratamiento para un gato diagnosticado de leishmaniosis?

No se han publicado estudios controlados sobre la terapia de la leishmaniosis en gatos. A falta de pruebas que indiquen lo contrario, el tratamiento empírico con los mismos fármacos recomendados para los perros suele considerarse eficaz y aparentemente seguro:

- Alopurinol (10 mg/kg q12 h o 20 mg/kg q24 h P.O., durante al menos 6 meses)
- Antimoniato de meglumina (20-50 mg/kg q24 h S.C., durante 30 días).

Los gatos tratados deben seguir una rigurosa monitorización para detectar posibles efectos adversos (los fármacos se utilizan fuera de registro y no hay mucha información sobre su seguridad en gatos). Ocasionalmente se ha reportado sospecha de lesión

renal aguda (antimoniato de meglumina, alopurinol) y reacciones cutáneas reversibles (alopurinol) asociados al uso de estos fármacos en el gato.

¿Y el pronóstico?

El seguimiento cuidadoso tras el final del tratamiento anti-*Leishmania* debe incluir un examen físico, un hemograma, perfil bioquímico, un análisis de orina y una serología cuantitativa; que deben repetirse periódicamente. La esperanza de vida de los gatos con leishmaniosis suele ser buena (años) a no ser que existan condiciones concurrentes (neoplasia maligna, infecciones por FIV/FeLV) o complicaciones (enfermedad renal). De hecho, se puede decir que el pronóstico depende de la enfermedad subyacente.

¿Como se puede prevenir la leishmaniosis felina?

No existen vacunas para la leishmaniosis felina. En zonas de alto riesgo, se puede prevenir la infección mediante el uso de insecticidas-repelentes tópicos (piretroides), como en el perro. Hay que recordar, sin embargo, que la mayoría de los piretroides son tóxicos para los gatos. Los collares de flumetrina son actualmente el único piretroide autorizada para los gatos y es el que se recomienda para prevenir la leishmaniosis, pues se ha demostrado que su uso reduce la incidencia de infección por *L. infantum* en los gatos en un estudio de campo.

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DRINK UP! FLUID THERAPY MANAGEMENT FOR CAPTIVE EXOTIC SPECIES

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DRINK UP! FLUID THERAPY CONSIDERATIONS FOR EXOTIC PETS

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The majority of the exotic pet cases presented to veterinarians are dehydrated because of chronic disease. Animals primarily lose moisture through the gastrointestinal and respiratory tracts, although the integument and eye (non-spectacle) are also sites where moisture is lost. Diseases associated with the gastrointestinal tract (e.g., diarrhea) or traumatic injuries to the integument (e.g., thermal burns) can increase the amount of moisture lost in our patients.

Characterizing the extent of dehydration and providing appropriate fluid replacement is necessary to successfully resuscitate the patient. An understanding of the “water” make-up of these patients should be considered before selecting a fluid. For example, reptiles have higher total body water than mammals and the extracellular space of reptiles contains higher sodium and lower potassium concentrations compared to mammals (the reverse is true for the intracellular space). Therefore, fluid selection should be based on both the type of dehydration and the physiological status of the patient. Hypertonic dehydration is common in animals that have limited access to water or do not drink. Isotonic dehydration generally occurs because of hemorrhage, diarrhea, and short-term anorexia. Hypotonic dehydration is a common sequela to prolonged anorexia. Characterizing the state of dehydration is important to ensure that the patient is re-hydrated correctly.

Historically, isotonic balanced fluids, such as lactated Ringer’s solution (273mOsmol/l, pH 6.6) and Normasol® (294mOsmol/l, pH 6.6) have been used when large fluid volumes are required. These fluids generally expand the extracellular space without a significant shift into the intracellular space. Prolonged usage of these fluids can lead to hypokalemia. In mammals, physiologic saline (0.9%)(308mOsm/l, pH 5.6) is preferred when there is a need for a rapid expansion of the circulatory volume, or to correct hyponatremia or alkalosis. Hypertonic solutions, such as 5% dextrose (252 mOsm/l, pH 4.3), are used when fluids are required to expand both the intracellular and extracellular spaces. The caloric input from the glucose is probably negligible and veterinarians should use appropriate enteral sources of nutrition to provide essential calories and nutrition. Combination fluids, such as 0.45% saline and 2.5% dextrose (280 mOsm/l, pH 4.3), are also commercially available.

Maintenance fluids should be provided to those animals not consuming sufficient fluids, in addition to correcting their fluid deficit. In general, the fluid maintenance rate for rabbits and small exotic mammals is 80-100 ml/kg/day. For birds there is a wide range of recommended fluid rates (50-100 ml/kg/day), although the author always bases the maintenance level on the high end of the range (100 ml/kg/day). The maintenance fluid rate of reptiles is 10-30 ml/kg/day.

Fluids can be given per os (PO) in patients that are mildly dehydrated (<5%) and have a functional gastrointestinal tract. The PO route is less invasive than other techniques. Per os fluids are not recommended in cases where patients are regurgitating, vomiting, or have diarrhea, as the administration of fluids by the PO route could potentiate an osmotic diarrhea. The patient must be restrained properly to ensure safe administration of fluids. Fluid administration generally requires two individuals, one to restrain the animal and one to deliver the fluids. Attempting to administer fluids PO using inappropriate techniques could lead to fluid aspiration. Fluids can be administered PO using a stainless steel gavage tube or red rubber feeding tube. The required tube length can be determined by measuring the distance between the snout and the midbody, which is the approximate location of the stomach.

Subcutaneous (SC) fluids can also be used to rehydrate mildly dehydrated patients (<5%). In severe cases of dehydration (>8%), other routes of fluid administration, such as intracoelomic, intraosseus, and intravenous fluids, should be used. There are a number of advantages to using SC fluids, including ease of administration and an ability to deliver large volumes of fluids. The primary disadvantage of using SC fluids is that the subcutaneous space in most animals is relatively avascular, leading to variable absorption rates. The most common site to administer

SC fluids in snakes and lizards is the lateral body wall. In chelonians, SC fluids are generally administered in the inguinal/femoral space. In birds, the inguinal and scapular areas are the most common sites of SC fluid administration. In mammals, the dorsal thoracic (scapular) and lateral body walls are the preferred sites for SC fluid administration.

Intraosseus (IO) fluids can be used in cases with moderate to severe dehydration. The IO route may be used when peripheral vasoconstriction limits intravenous access. Intraosseous catheters are clinically advantageous and appropriate in small and fractious patients due to ease of placement, catheter stability and clinical response. The femur, tibia, and humerus may be used for IO catheter placement in reptiles. In birds, the ulna and tibiotarsus are preferred. The femur and humerus should never be used because they may be associated with air sacs. In mammals, the proximal femur and tibia are preferred sites for IO catheters. A local anesthetic, such as lidocaine, should be used to reduce the pain associated with catheter placement. The author prefers to use spinal needles for IO catheters, as they have a stylet that prevents plugging the needle with a bone core.

Intracoelomic (ICo) fluids can be administered to reptiles and mammals with moderate to severe dehydration; however, this route should never be used in birds as it can lead to accidental drowning. The large serosal surface area of the viscera and the coelomic/peritoneal membranes of reptiles and mammals serve to resorb the fluids. Irritating compounds should not be administered ICo. Intracoelomic fluids are not recommended in reptile patients that have respiratory compromise, as they may place an additional burden on the animal. Intracoelomic fluids should be given in the caudal coelomic cavity.

Intravenous fluid administration is the preferred route of fluid administration in moderately to severely dehydrated patients. The jugular vein can be used for lizards, chelonians, and snakes. Placement of the jugular catheter in the snake and lizard requires a surgical cut-down. A local anesthetic, such as lidocaine, should be used to reduce discomfort. The cephalic vein is another site for catheterization in the lizard, whereas the heart may be directly catheterized in severely moribund snakes. In birds, the jugular, basilic or medial metatarsal veins can be used to place IV catheters. The medial metatarsal vein is generally large (and approachable) in waterfowl, raptors, and large psittacines. In smaller psittacines, the basilic vessel is preferred. In mammals, the jugular, cephalic and lateral saphenous sites can be used for IV catheters. When collecting blood samples from exotic pet patients, it is important to consider possible IV sites prior to sample collection. For example, the author never collects a blood sample from a rabbit cephalic vein, preferring to save the site for IV catheterization.

To the author, every case should begin with rehydrating a patient. For any other treatments to be successful, the patient must have the fluid resources to ensure different organ systems are capable of processing the medications. By starting every treatment plan off with providing essential fluids, veterinarians will increase their success rate with their exotic pet patients.

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MANAGING FEAR, STRESS AND PAIN IN A SHELTER ENVIRONMENT - CHALLENGES AND OPPORTUNITIES

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ABSTRACT BODY:

INTRODUCTION

Animal shelters face a myriad of challenges including but not limited to a large animal population and high turnover, suboptimal facility, and limited budget. These challenges contribute to a fast paced and often chaotic environment, which has the potential to cause fear and anxiety for animals and staff alike. This can have significant consequences on animal health, causing immunocompromise, increased susceptibility to infectious disease, and decreased adoptability. There is also the added consideration that animals might be painful, from either emotional or physical trauma. Learning how to recognize signs of stress and pain and applying practical methods to address them are key to ensuring shelter animal welfare.

RECOGNITION

Because individual dog and cat personalities vary, behavioral signs of stress can vary. Some animals may attempt to hide in their kennels or cages, or stop eating. Others may behave aggressively or perform repetitive behaviors. Over time, such behaviors have the potential to lead to behavioral deterioration and decreased adoptability.

One of the most challenging aspects of managing fear, stress and pain in a shelter environment is recognizing it in the first place. Shelter staff members often lack training on assessing fear and pain. One potentially useful tool to facilitate this in cats is the feline grimace scale (FVS) which can be used to make decisions about shelter housing, required treatments, and behavioral care (1).

Shelters can and must manage pain in shelter pets. According to the Association of Shelter Veterinarians Guidelines for Standards of Care in Animal Shelters, failure to provide treatment for pain is unacceptable and when adequate pain relief cannot be achieved, transfer to a facility that can meet the animal's needs or humane euthanasia must be provided (2). While there is no gold standard for assessing pain in dogs and cats, objective measures include heart rate, respiratory rate, and blood pressure.

SHELTER INTAKE

Shelter admission is undoubtedly one of the most stressful events that a dog or cat can endure. Animals are separated from their previous families, confined, and faced with unfamiliar scents, noises, and people. Intake examinations should be performed as gently as possible, minimizing restrain while conducting necessary procedures such as vaccination, deworming, and scanning for a microchip. Soft non-slip flooring and gentle support can also help. During an examination, the simple act of placing a blanket or towel over a dog's head can prevent him or her from being stressed by the sight of other animals.

THE FIVE SENSES

While in the shelter, animals can become overwhelmed by visual and auditory stimuli. By reducing unpleasant stimulation, we can minimize fear and stress. Simple actions such as draping a towel over the front of a cage door can create an inexpensive visual barrier. Cats should always be provided with a place to hide. This can include a hiding box or a towel can be draped on the front of the cage door.

Treats can be an effective way to gain an animal's trust, and ultimate mitigate their fear. Canned food, meatballs, and soft cheese spread and be used to coax animals out of their cages, and distract them during examinations.

Research suggests that animals housed in shelters are significantly influenced by environmental scents (3). Shelters should use cleaning products at recommended dilutions to minimize noxious odors. The use of different scents including lavender and chamomile may help calm shelter animals. Controlling acoustic stimulation should also be considered. Sound absorption panels can reduce the decibel level associated with barking.

Time spent outside the kennel dramatically supports physical and mental well-being. This can be accomplished, for dogs through regular exercise, supervised play groups, and walking. While in the kennel, use of bedding and toys can help dogs feel mentally stimulated and reduce anxiety.

SPAY NEUTER

Spay/neuter surgery is a common practice in shelters, which is potentially painful and stress inducing. Adequate pain management is a must as it ensures successful patient outcomes, optimal anesthetic recovery, and shorter shelter length of stay.

The use of anesthetics and analgesics in animal shelters is often similar to those used in private practice settings, with an emphasis on uniform dosing charts and simplification. While many shelters adopt a one-sized fits all protocol for post-operative pain management, they should maintain the ability to tailor pain management plans to individual animals when needed. In shelters, a fairly cost-effective, safe, and very efficacious option to consider is the use of local anesthetic blocks for post-operative spay/neuter pain control.

FEAR FREE SHELTERS

Simple practices can make a huge difference in the comfort of both people and animals. A variety of training resources for those involved in the care and oversight of shelter and rescue animals is available at fearfreeshelters.com (4). The Fear Free shelter program aims to improve the experiences of animals by educating shelter, rescue, and animal welfare employees to reduce fear, anxiety, stress, and frustration of shelter animals.

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THE POSTANESTHETIC PERIOD - ANYTHING TO WORRY ABOUT?

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ABSTRACT BODY:

The risk of **anaesthesia-related complications** is reported to be 2-10 %, with some publications reporting much higher rates of up to 80 %. The most commonly reported complications were intra-anaesthetic cardiovascular impairment, e.g. cardiac arrhythmias and hypotension, hypoventilation and hypothermia. Perioperatively, regurgitation was the most frequently reported gastrointestinal complication, sometimes with a fatal outcome. Similarly, gastrooesophageal reflux was reported in 16% to 60% of animals undergoing anaesthesia. Prolonged recovery time, agitation during recovery, but also decreased mentality after (presumed) recovery, renal failure, perioperative blindness, prolonged hypothermia and hyperthermia are found in the literature.[1] However, studies reporting **morbidity** are scarce in the veterinary literature. This is probably due to the lack of clear definitions for the respective complication as well as difficulties in recognition and recording. **Mortality**, on the other hand, has a clear endpoint (death). While inconsistent time intervals have been used and clearly distinguishing anaesthesia-related mortality from other causes is not always easy, the data are more consistent over the last 70 years. In the largest multicentre study[2], anaesthesia-related mortality was reported to be 0.05% in healthy dogs (American Society of Anesthesiologist Physical Status = ASA-PS 1 or 2, i.e. no/low preoperative disease) and 0.11% in cats (ASA-PS 1-2). In dogs and cats with a higher ASA score of 3-5 (moderate to severe preoperative disease), mortality was significantly higher at 1.33% and 1.4%, respectively. The timing of deaths varies between studies, but in most cases the **postoperative period** carries the **highest risk** of death, with 50% to 75% of deaths occurring during or after recovery. Especially the first 3 hours after recovery seem to be of great importance. However, fatal long-term consequences up to a few days post-operatively have also been reported.[1]

In the immediate post-operative phase, the animal is still under the influence of the anesthetics used, which affects the cardiovascular and respiratory systems. Heart rate, blood pressure, ventilation, thermoregulation, gastrointestinal motility including swallowing reflexes and responses to external stimuli may still be impaired. While during anesthesia the animals' airways are usually protected and cardiopulmonary function is monitored, this is often not the case during the recovery phase. In the following part, frequently occurring complications and their possible implications are discussed.

Hypothermia is one of the most common intra- and postoperative complications. It has many serious consequences, including delayed recovery from anesthesia, delayed wound healing, increased risk of infection and more. **Shivering** during hypothermia significantly increases oxygen demand and is described as extremely uncomfortable in humans. On room air, shivering can quickly lead to **hypoxemia**, and supplemental oxygen is indicated. Good temperature management from the beginning with circulating hot water blankets, recirculating air warmers and blankets is better than excessive postoperative warming. The use of hot water gloves for this purpose is strongly discouraged, as there is a significant risk of the patient getting burned or even chilled if the water temperature is below the desired body temperature. When providing external heat to animals in recovery, close monitoring of the animal's temperature is essential. The newly recovered animal may still be too weak to leave the warm place, putting them at risk of **hyperthermia**. In cats, opioids, particularly hydromorphone, have been associated with postoperative hyperthermia. Interestingly, the more hypothermic the cat was intra-operatively, the worse this appears to be.

The negative effects of most anesthetics on **blood pressure** usually subside after the anesthetics are stopped. The hematocrit also usually increases slightly during the recovery period. However, in dogs and cats that are hypovolemic, e.g. due to intraoperative blood loss, fluid therapy must be continued beyond anesthesia to ensure adequate intravascular volume, blood pressure and ultimately tissue perfusion.

In critical patients, blood pressure should be monitored throughout the recovery period and beyond to ensure blood flow to the vital organs. Prolonged **hypotension** can lead to kidney failure and

(temporary) blindness not only intra-operatively but also post-operatively. The latter is due to the fact that the optic nerve is very sensitive to **ischemia**.

The appropriate fluid regimen is determined by the patients need. Assessment of **blood glucose** is indicated in very young, but also very small animals as they easily become **hypoglycemic,** as well as in **diabetic** animals.

Especially in small dogs and cats, fluid therapy should be administered via a pump or micro-burette to avoid **accidental fluid overload**.

In 40-50% of cases of anesthesia related mortality in dogs and cats **respiratory complications** are the the cause.

While **hypoventilation** is another common complication during surgery, it is often overlooked in the postoperative period. This could be due to the difficulty of monitoring ventilation in extubated animals and/or the inability to assess arterial blood gases. However, it is important to assess the respiratory pattern in the recovery phase and monitor **oxygenation** using a pulse oximeter. If the animal is suspected of hypoventilating, oxygen should be supplied to prevent hypoxemia.

Airway obstruction is especially a problem in brachycephalic breeds. Late endotracheal extubation and close monitoring of respiration and oxygenation (SPO2) is important in these breeds. The use of sedatives for recovery in brachycephalic dogs is controversial. Their use is often recommended in animals with respiratory distress. However, heavy sedatives such as acepromazine and alpha-2 agonists relax the muscles of the oropharynx. Furthermore, in combination with opioids or other medications, they may contribute to respiratory depression.

An increased risk of **aspiration pneumonia** has been associated with endoscopies, thoracotomies, neurologic procedures, upper airway surgery, and abdominal surgery [3].The effect of drugs on aspiration pneumonia is somewhat controversial/ unclear. It was demonstrated that hydromorphone administered at induction of anesthesia and intraoperative infusions of constant rate anesthetics or analgesics such as morphine, lidocaine, ketamine, fentanyl, or propofol increased the risk of aspiration pneumonia [3]. Again, brachycephalic breeds are at higher risk for **regurgitation** and **aspiration** compared to non-brachycephalic breeds [4]. Silent reflux, or **gastroesophageal reflux**, is difficult to detect and, according to recent studies, is much more common than previously thought. High-dose metoclopramide may reduce the incidence of reflux. Toward the end of anesthesia, the depth of anesthesia might be deeper than needed considering a lack of surgical stimulation. Thus, the animal is relaxed and esophageal sphincter tone low. When the animal is brought out of the operating room, the endotracheal tube should still be in place and cuffed. It also makes more sense to transport the animal on a gurney rather than carrying it on your arms, which would put pressure on the animal's stomach.

However, denial of analgesics is not appropriate. **Pain** is not only of ethical concern, but also has a significant negative impact on the body. Whenever possible, locoregional anesthesia can help minimize the need for systemic pain medication. The use of appropriate pain assessment tools, such as the Glasgow Pain Scale, is useful to administer pain medication as needed rather than only after a specific time interval. Documenting pain levels before and after the procedure also helps evaluate the effectiveness of treatment.

Considering the high prevalence of complications within the first three hours of recovery, it seems advisable to keep animals under close veterinary care. Animals should therefore be discharged no earlier than three hours after recovery, when all parameters are within normal limits. The risk of earlier discharge lies especially in re-narcotization accompanied by hypoventilation and hypoxemia. Similarly, severe pain cannot be treated by the owner at home. Finally, owners should be provided with detailed **anesthesia discharge instructions** to assist with at home support [5]. This helps to identify, treat, and document postoperative complications to improve future patient care.

In summary, the post-anesthesia period is a critical time associated with a significant risk of morbidity and mortality. Close monitoring and care are critical, especially during the first 3 hours of recovery.

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PAIN MANAGEMENT USING OPIOID-FREE AND OPIOID-MINIMAL TECHNIQUES

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ABSTRACT BODY:

Opioids are the cornerstone of acute pain management in veterinary medicine (Simon & Steagall 2017). The international veterinary community does not always have access to drugs for a basic standard of care (Pesteon et al. 2016). Unfortunately, drugs like opioids are not readily available in many countries making anesthesia and analgesia suboptimal. Limited availability of opioids makes pain management challenging leading to unnecessary animal suffering. It results in poor welfare for animals and compassion fatigue for veterinary health care professionals. Veterinarians are struggling to find reasonable options to effectively manage pain (see references below).

Recently, a study was performed in cats undergoing ovariohysterectomy (Diep et al. 2020). The study aimed to evaluate the anesthetic and analgesic effects of an opioid-free injectable protocol. In a single intramuscular injection, cats were administered ketamine [5 mg/kg, n = 10, K5; or 7 mg/kg, n = 13, K7] with midazolam (0.25 mg/kg) and dexmedetomidine (40 mg/kg). During surgery, cats were also administered bupivacaine (2 mg/kg) by the intraperitoneal route, and after surgery, subcutaneous meloxicam (0.2 mg/kg) was also administered for postoperative analgesia. Postoperative pain was assessed at various time points after surgery using the Feline Grimace Scale (FGS) and the Glasgow feline composite measure pain scale (CMPS-Feline). Rescue analgesia was provided with buprenorphine (0.02 mg/kg IV) when pain scores were ≥ 5/20 (CMPS-Feline) and ≥ 0.39/1.0 (FGS). Within 2 minutes of intramuscular injection, most cats were in lateral recumbency. The injectable protocol provided an adequate depth of anesthesia and none of the cats required a ketamine bolus for maintenance of anesthesia. The prevalence of anesthetic complications was not significantly different between treatment groups K5 and K7. These included tachycardia, tachypnea and desaturation. The following complications were not observed in any cat: bradycardia, hypotension, bradypnea, hypercapnia and hypothermia. Thirteen out of 23 cats required rescue analgesia; 6 cats in K5 and 7 cats in K7. Prevalence of rescue analgesia was not significantly different between treatment groups; however, when cats were divided according to age group and adults and kittens were compared, the prevalence of rescue analgesia was significantly greater in adult cats than in kittens. Rescue analgesia was usually required at 2h and 3h after surgery (Diep et al. 2020).

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HOW TO GUIDE CLIENTS TO FACT-BASED INFORMATION ABOUT PET FOOD

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ABSTRACT BODY:

Pet owners or caregivers in many countries have a huge variety of diet choices for feeding their dog or cat. Information is available from multiple sources and can have conflicting and confusing advice and often contains misinformation. One survey found nearly half of 900 dog owners felt that diet choice was the most difficult part of pet ownership[1]. Over half of owners, and 68% of Millennial aged owners (18 to 34 yr), said their dog's nutrition was more confusing than their own and 25% felt overwhelmed by the choices available.

Where People Source Pet Food Information

Recommendations for diet may be received from the veterinary healthcare team, friends or family members, pet stores, breeders, on the internet from "information websites" not obviously aligned with a petfood manufacturer, or from manufacturer websites. One study noted that 44% of dog owners cited "past experience" to determine what to feed[2].

Several surveys have been done regarding pet food choices. Surveys can be affected by selection bias, i.e. self selection by those interested or engaged in the topic, and by social desirability bias, a tendency to respond in a way that will be viewed favourably by others. Responses are limited to the population studied making it difficult to generalize to a wider population, but surveys provide a starting point for the veterinary profession to understand diet choices.

The percentage of owners using veterinary advice for diet information varies greatly among studies, from 9%2 to 43.6%[3]. The latter US based study included 26% animal professionals (veterinarians, veterinary nurses/technicians, breeders, trainers, petfood employees) and a highly educated demographic population with about 75% having a college degree, compared to about 38% of the total US population[4].

Studies occurring before the Covid 19 pandemic showed that friends and family provided about 10% of recommendations, similar to pet food stores, and online sources provided about 5%[5]. Use of online sources to purchase pet foods increased considerably during the Covid-19 pandemic[6]. Use of the Internet as a resource for pet diets may also have increased, especially as veterinary visits were limited or done under adverse conditions (e.g. in the car park with owners not allowed into the clinic) and pet food stores were often closed.

The percentage of compliance with pet food recommendations also varies. Compliance with veterinary recommendation has been reported at 77 to 80%, lower than breeder recommendations at 86%[5]. Compliance with online "information websites" e.g. comparison sites, and with manufacturer's websites was 68% and 66%, respectively.

Owner receptiveness to diet recommendations also depends upon whether a conventional or an alternative diet choice, e.g. homemade or raw diets, is fed, with conventional feeders more likely to follow veterinary advice. Cat owners were more likely than dog owners to feed commercial pet food rather than a home prepared diet as a main diet (96% vs 87%)[7] and (90% vs 79%)[8] and were also more likely than dog owners to consider veterinary nutritional recommendations as effective for their pets' diet[9].

Pet owners feeding alternative non-commercial diets are less likely to discuss nutrition with their veterinarian and less likely to trust veterinary nutritional advice[10.] Most owners who feed raw food diets learn about these diets online or from a friend or family member10. Unfortunately, it also appears that owners feeding non-conventional diets, include home prepared raw diets, are likely to source the diet formulation and amount from the internet or from non-veterinary books[10,11]. Diet formulations from these resources have been frequently reported to contain deficiencies and excesses[12].

Reason given for feeding unconventional diets include a desire to feed a diet closer to that of the pet's ancestors (e.g. like a wolf), the perception that these diets are healthier or more natural, a wish to avoid certain ingredients (e.g. grains, by-products/ meat derivatives, additives), distrust of large pet food manufacturers, and a lack of an appropriate and/ or palatable diet for medical conditions (e.g. chronic kidney disease plus canine chronic pancreatitis).

The Nutrition Conversation

In one study only 60% of veterinarians discussed pet nutrition during an appointment and only 12% of veterinarians recommended long term health related diet changes[13]. Another study showed only 21% of Belgian and Dutch veterinarians had prior knowledge of the WSAVA nutritional guidelines[14]. While veterinarians may recommend therapeutic diets for

disorders, diet discussion during routine appointments or wellness checks is less common. This may be due to the veterinarian's perceived limitations of their own knowledge, avoiding a potential emotionally charged discussion, a lack of time, or simply not routinely incorporating nutritional assessment into consultations.

A nutritional assessment, including diet history, should occur for every pet at every consultation[15]. It is an effective way to initiate the more detailed discussion about diet and provide fact based information. Using open ended questions such as "Tell me what you feed your dog" garners more information than " What do you feed your dog?". The latter may result in information only about the main diet16. Many dogs and cats are fed homemade or raw foods in addition to a primary conventional diet8 . Other suggested diet history taking approaches include asking everything the pet eats from morning until bedtime[16] or to describe the diet as they would to a potential pet sitter.

Owners may be reluctant to discuss treat giving or food used for medications. Using a statement such as "Tell me what treats Sam gets" or "Describe the foods you use to give Buffy her medications" can provide an approach which appears less judgemental than "Do you give treats?".

Providing Facts about Pet Food and Making Diet Change Recommendations.

What do we do when the diet history reveals a diet which is not complete and balanced or not appropriate for that pet? Before telling the owner evidence-based facts, ask them in a non-judgemental way to tell you why they have made their diet choice. If their choice is due to misperceptions about the diet's benefits, asking if it is ok to discuss it further. Some owners may not be ready for that discussion or may not wish to have it at all.

Changes in behaviour, such as feeding practices, have several stages[17]. The first is pre-contemplation. In this stage the owner has not thought about a diet change and isn't ready to consider a change.

Contemplation is the next stage, where a change may be possible in the next several months but the owner is not yet ready or sure of wanting a change.

Preparation or determination is the next stage, where an owner is getting ready to change. This is followed by action where the change actively happens, and finally, maintenance where the behaviour is maintained.

It is difficult to deal with misinformation. When a belief is based on a misperception the person has built a set mental model using that idea. They may also have confirmation bias, which is the process of selectively seeking or agreeing with information which fits a world view or idea and ignoring or refuting opposing ideas. When presented with a balanced set of facts, people gravitate toward those which reinforce their pre-existing views, especially with strongly held views.

When facts are presented which are in disagreement to established ideas, the established idea may be clung to even more fiercely. Doubt turns people into stronger advocates of their ideas[18]. This is termed cognitive dissonance and the effect is stronger if someone's identity is threatened (e.g. I'm a vegetarian so my pet should be vegetarian) or if the belief is important to them (e.g. my dog should be fed like his wolf ancestor).

Offering fact based information, e.g. handouts or website links may be useful, especially in the earlier stages of change, e.g. pre-contemplation or contemplation or if there is resistance to a discussion of diet. Written material is more effective than oral information.

The WSAVA GNC toolkit has information for owners on using internet information and on selecting a petfood[15]. Advice on using the Internet includes checking the information source, timelines, and the author's credentials, and to be wary of anecdotes, rating websites and grand claims.

You are not likely to change a set mind in one consultation, but can plant the seed that there are other potentially beneficial diets and have another conversation at future consultation. Statements based on pet health are the most effective for persuading owners to consider changing their pet's diet compared to a veterinarian's personal choice or manufacturer recommendations[19]. Ninety-three percent of owners indicated they were at least somewhat willing to change their pet's diet based on veterinary recommendation. Of the 18% who had a diet change recommended, 68% planned to follow the advice, especially if recommendations occurred within an established empathetic veterinarian-client-pet-relationship.

Overall, studies show that most owners do want nutrition advice from the veterinary healthcare team and are willing to consider a diet change. The profession needs to be more active and willing to perform nutritional assessments and provide fact based information.

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SURGICAL DECISION-MAKING IN TRAUMA

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ABSTRACT BODY:

An animal's response to injury is dictated first by the primary trauma and resulting direct tissue and organ injury. The source and velocity of the trauma play a large role in the severity of the injuries sustained. For example, high velocity blunt impact trauma can result in internal bleeding, traumatic brain injury, long-bone fractures, and visceral organ damage; whereas low velocity blunt trauma can result in extensive external injury, but perhaps limited internal trauma. With large open wounds following trauma the animal should be assessed for life-threatening injuries prior to initiating wound care. Assessment of the entire patient is critical, with priority given to the cardiovascular, respiratory, and neurologic systems. Wounds are of lower importance in the initial period following presentation, unless there is a significant arterial bleed or penetration into the abdominal or thoracic cavity.

Additional injury comes from the secondary response to the injury (endogenous) and emergency interventions aid the animal (exogenous). These are the "second hits" the concept known as the 2-hit theory. Acute traumatic cases that require surgical intervention fall into one of three categories:[1]

- Patients requiring immediate surgery to prevent imminent death
  - Arterial hemorrhage
- Patients with varying degrees of hemodynamic stability with injuries contributing significantly to morbidity requiring surgical intervention
  - Diaphragmatic herniation
- Patients that are hemodynamically stable that can go to surgery on a semiselective basis
  - Uroabdomen

The timing of surgical intervention on traumatized animals can have a strong influence on outcome, whether it is instituted too early or unnecessarily delayed. Advancements in emergency stabilization, intensive care, and general anesthesia also play a role in the decision of when to go to surgery. For example, a landmark retrospective studies found mortality associated with diaphragmatic herniation is higher when surgery is performed less than 24 hours or more than 1 year after injury.[2] However, studies performed 15 to 20 years later did not find an effect of the duration of the hernia on overall outcome, whereas the severity of the trauma and degree of concurrent injuries does affect outcome.[3-5]

Animals sustaining bite wounds should be handled with great care and caution. Often there are multiple penetrating wounds, and the degree of trauma may not be initially apparent. The iceberg principle applies here, as the superficial wound is not reflective of deeper soft tissue damage. The crush pressure of the canine jaw ranges from 150 to 450 psi, and the trauma can result in neurovascular injury. The contamination to the wound is significant and often results in polymicrobial infection. Antimicrobial coverage should include an anaerobic spectrum.

It is generally accepted that penetrating trauma to the abdominal cavity requires immediate surgical intervention upon recognition, even though the animal may not be hemodynamically stable enough to tolerate general anesthesia. This leads to a relatively new concept in veterinary trauma medicine called Damage Control Strategy. This approach has been widely accepted in human medicine to reduce mortality. Regarding abdominal injury, the goal would be an early abbreviated surgical procedure to stop bleeding and gastrointestinal contamination, with a more definitive surgery a later time when the animal is further resuscitated and stabilized.

Penetrating thoracic trauma has more challenges when deciding whether surgery is even indicated at all. In multiple studies evaluating outcomes following thoracic bite wound, only 20-30% of cases undergo exploratory thoracotomy, and regardless of surgery or not, mortality averages around 15%.[6-7]

Additional challenge in veterinary medicine center around the limitations to performing emergency surgery in a general or primary care practice. These limitations include, but are not limited to:

Surgical confidence

Staffing support and assistance

Equipment and product availability (e.g., blood products)

Anesthetic support

Postoperative support

Minimal surgical skills required for trauma patients prior to transfer or referral include achieving

hemostasis, airway capture, wound debridement and application of dressings, stabilization of musculoskeletal or spinal injuries.

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MASAS TORACICAS: CLAVES PARA CONOCER SU ORIGEN EN UNA RADIOGRAFÍA

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ABSTRACT BODY:

INTRODUCCION

Las masas de la cavidad torácica pueden tener su origen en el

- pulmón
- mediastino
- pared torácica (extrapleural)
- espacio pleural.

El objetivo de esta charla es describir los hallazgos radiológicos para poder diferenciar una masa de otra.

MASAS PULMONARES

Las masas pulmonares se localizan a nivel de los lóbulos pulmonares. Así recordad que los perros y gatos tienen dos pulmones, los cuales no son simétricos en cuanto a tamaño y localización. La anatomía lobar se basa en la división de los bronquios.

-El lóbulo izquierdo está formado por dos lóbulos:

- Lóbulo craneal izquierdo, dividido en un segmento craneal y uno caudal.
- Lóbulo caudal izquierdo

-El lóbulo derecho está formado por cuatro lóbulos:

- Lóbulo craneal derecho
- Lóbulo medio derecho
- Lóbulo caudal derecho
- Lóbulo accesorio

Los signos radiográficos que observamos cuando existe una masa pulmonar son:

- Márgenes nítidos, forma regular
- Forma un ángulo agudo son la pared torácica
- No suele desplazar otras estructuras torácicas
- No invade la pared torácica

La neoplasia pulmonar es la causa más común de una masa pulmonar (que puede presentarse solitaria o múltiple). Los adenocarcinomas representan aproximadamente el 90% de todos los tumores pulmonares primarios, le siguen carcinomas de células escamosas, carcinomas broncogénicos, broncoalveolares y anaplásicos. Los carcinomas broncogénicos se suelen localizar en la vecindad del hilio pulmonar, mientras que los carcinomas bronquiolares, generalmente se localizan en la periferia pulmonar.

A parte de neoplasias, las masas pulmonares pueden ser compatibles con otras patologías, y los siguientes diagnósticos diferenciales deben ser considerados:

1. Granulomas.
2. Nódulos/masas extratorácicas.
3. Absceso
4. Quiste
5. Hematoma.
6. Lesión cavitaria.
7. Consolidación

MASAS MEDIASTINICAS

Las masas mediastínicas son comunes, y su aspecto radiográfico es similar. Las proyecciones VD/DV son más útiles que las laterales para decidir si una masa es anormal y su localización (mediastino vs pulmón).

Se considerará que una masa es mediastínica cuando:

1.- la masa está situada en la línea media o adyacente

2.- la masa está en posición consistente con alguno de los tres recesos mediastínicos

3.- la masa desvía alguna estructura mediastínica

Las masas pulmonares no deben confundirse con una masa de origen mediastínico porque ellas generalmente se sitúan en una posición lateral al mediastino.

La diferenciación de las masas mediastínicas se basa en su localización específica en una de las cinco regiones en las cuales se divide el mediastino:

- Craneo-ventral
- Craneo-dorsal
- Hiliar y perihiliar
- Caudodorsal
- Caudoventral

En los gatos las más frecuentes son las craneoventrales. En perros las craneoventrales e hiliares. Perros jóvenes craneodorsales

Cuando se realiza la interpretación radiográfica hay que pensar en la variación anatómica, el mediastino es ancho en las razas braquicefálicas, en perros viejos y obesos. En perritos y gatitos podemos observar el timo (4-5 meses).

A excepción de los lipomas, el resto de tumores del

mediastino son malignos.

1.- Masas craneoventrales

Signos radiográficos

- Pérdida del espacio radiotransparente craneal al corazón
- Desplazamiento dorsal y hacia el lado derecho de la tráquea, también puede hacer compresión de la luz de la tráquea. La elevación de la tráquea puede también producirse cuando hay gran cantidad de líquido en el espacio pleural sin masa mediastínica, ello es debido a que los pulmones están flotando en el líquido. La elevación de la tráquea no se ve cuando hay pequeñas cantidades de líquido, al menos de que exista una masa mediastínica.
- Desplazamiento caudal del corazón algunas veces se forma el signo silueta
- Desplazamiento caudal de los lóbulos craneales.

Causas:

1. Artefactos: masa en el lóbulo craneal que puede contactar con el mediastino y parecer una masa mediastinica. Liquido pleural alrededor del lóbulo craneal.
2. Timo normal
3. Neoplasia, a menudo se acompaña de efusión pleural
    - Linfosarcoma (más común en gatos)
    - Timoma acompañado por miastenia gravis y megaesofago
    - Histiocitosis maligna. Razas grandes y de mediana edad (rotweiler, Golden)
    - Tumor de tiroides y paratiroides ectópico
    - Tumor en costilla
    - Otros, lipoma, fibrosarcoma
4. Dilatación esofágica secundaria a una anomalía del anillo vascular
5. Linfoadenopatia mediastinica craneal.
6. Linfoadenopatia esternal (opacidad dorsal a la región de la 2-4 esternebra, y se ve mejor en proyección lateral)
7. Abscesos, granulomas mediastinicos (C.E., nocardiosis, actinomicosis)
8. Hematoma mediastinico
9. Quiste mediastínico

2.- Masas craneodorsales

Son menos comunes que las craneoventrales .

Signos radiográficos

- Desplazamiento ventral de la tráquea intratorácica y el esófago craneal al corazón. Ocasionalmente desplazan la tráquea hacia la derecha. Signo silueta con la aorta descendente. En todas las masas mediastinicas craneodorsales está indicado un esofagograma, para ver si el esófago está implicado, funcionalidad del mismo y desplazamiento de este.

Causas:

1. - Esófago
    - Dilatación (megaesofago, anomalías vasculares)
    - Cuerpos extraños
    - Neoplasia esofágica (raro)
2. - Aneurisma aórtico
3. - Tumores de base de corazón (chemodectoma)
4. - Asociaciones con lesiones vertebrales (neoplasia, osteomielitis, espondilosis)
5. - Abscesos y granulomas mediastínicos (reacción a CE, nocardiosis, actinomicosis)
6. - Hematomas (hemorragias, coagulopatías)

3.- Masas hiliares y perihiliares

Estas son las más comunes en perros. Causadas por muchas enfermedades y a veces se deben a un aumento de los ganglios traqueobronquiales. Las lesiones en masa en esta área generalmente están mal definidas en la base del corazón y pueden parecer un edema pulmonar localizado.

Signos radiográficos

- Aumento de la opacidad perihiliar
- Se acentúa la flexión ventral de la tráquea
- Compresión de la luz de la tráquea
- Desplazamiento dorsal del esófago/tráquea

Las lesiones en masa de esta área incluyen:

1. - Esófago:
    - Cuerpos extraños
    - Dilataciones
    - Tumores (algunos secundarios a granuloma de Spirocerca lupi)
2. - Tumores de la base del corazón (chemodectoma= tumor arco aortico)
3. - Linfoadenopatía de los nódulos linfáticos traqueobronquiales, generalmente asociado con patología pulmonar, pleural y otros signos sistémicos (neoplasia).
4. - Estructuras cardiacas que parecen masas (ej Aumento de la aurícula izquierda/derecha)
5. - Masas bronquiales y pulmonares adyacentes
6. - Masa tiroides ectópica

4.- Masas caudodorsales/ventrales

El número de estructuras es muy limitado en esta zona

del mediastino. Las densidades se originan a partir del esófago y de órganos de la cavidad abdominal que se sitúan en esta zona entrando vía hiato esofágico o vena cava caudal.

Causas:

- Alteraciones esofágicas (cuerpos extraños, divrticulo esofágico..)
- Hernias (hernia peritoneo-pericardica)
- Granulomas, abscesos debido a CE
- Quistes pericárdicos

MASAS DE LA PARED TORACICA (EXTRAPLEURAL)

La definición de masa extrapleural, es aquella masa que se origina a lo largo de la línea o "espacio potencial" que existe entre la pleura y la pared torácica, en el tejido conectivo que constituye la fascia intratorácica. Las estructuras dentro y adyacentes a este "espacio potencial" a partir de las cuales se originan las masas extrapleurales son: tejido conectivo, grasa, costillas, esternebras, músculos, vasos sanguíneos y nervios.

El factor común denominador de las masas extrapleurales, es que están cubiertas por las dos capas pleurales (visceral y parietal), y es por ello que estas masas presentan un contorno, que contacta con el pulmón, muy liso, convexo y mejor definido que las masas pulmonares. De hecho, el término "signo extrapleural" designa esta apariencia radiográfica característica. Existen excepciones como los osteocondromas, condrosarcomas y osteosarcomas, los cuales pueden tener una superficie nítida, pero con forma irregular.

En ocasiones, las masas extrapleurales resultan indistinguibles de las masas pulmonares.

Los signos radiográficos de las masas extrapleurales son:

1. Contorno convexo bien definido
2. Bordes craneal y caudales inclinados/estrechados hacia la superficie de la pared torácica.
3. El diámetro transverso es mayor comparado con el diámetro perpendicular en el sitio de inserción de la masa a la pared torácica.
4. La parte más ancha de la masa es el lugar de su inserción a la pared torácica.
5. El espacio pleural permanece inicialmente inalterado.
6. Puede existir reacción perióstica en las costillas, o destrucción de una costilla y ensanchamiento irregular de espacios intercostales.

Posibles etiologías de las masas extrapleurales de la pared torácica:

1. Enfermedades inflamatorias y traumáticos (abscesos, hematomas granulomas y cuerpos extraños)
2. Enfermedades neoplásicas (tumores de costilla primarios (condrosarcoma, osteosarcoma, fibrosarcoma) los más frecuentes

La mayoría de las masas extrapleurales desarrollan una reacción ósea en las costillas antes o después, por ello la afección de una costilla es un signo patognomónico de una patología extrapleural.

Las masas más comunes son neoplasias primarias de costilla, metástasis en las costillas, granulomas por cuerpo extraño y abscesos debido a heridas por mordedura. Radiográficamente, todas las lesiones parecen similares y la ecografía de las masas junto con aspiraciones con aguja fina para realizar citologías (o biopsias) serán los medios que nos ayudarán a realizar un diagnóstico definitivo.

MASAS PLEURALES

Las masas pleurales suelen ir asociadas a efusiones pleurales, con lo cual se requiere su drenaje para poder visualizar la masa si se sospecha de esta.

Diagnósticos diferenciales

· Hernia y eventraciones

· Tumores pleurales (Mesotelioma es el tumor pleural primario que no se presenta como masa). Las efusiones se suelen presentan mas comúnmente con neoplasias pleurales y extrapleuralares que con tumores pulmonares.

· Carcinomatosis metastática

· Absceso y granuloma pleural

· Líquido pleural encapsulado (no se desplaza con la gravedad)

· Fibrina

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COMO FORMAR EQUIPOS DE ALTA PERFORMANCE

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ABSTRACT BODY:

Formar equipos ya es tarea ardua, compleja. De alta perfornace, sisempeño, aún más. Hay qye tener metodologia, y hay muchas en estes tiempos. Manejando el promedio de edades de los coaboradores, sus deseos, sus visiones, conflictos. Juntar las visiones del team con la del empresario, dueño de su negocio es fundamental. Ningun equipo llegará al alto disempeño si no sabe para donde caminamos, para donde queremos llegar en términos de números, la construcción de nuestra visión integrada, con proyecciones al futuro objectivado. La cosntruccion de las estrategias es metodógica, y cualquier empresa puede construirlas, con base en el liderazgo concreto de su director. El team tiene que saber como estamos, nuestros números, ingresos, lucros, etc. Es mito no se divulgar entre los colaboladores tod el financiero.

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COMMON EMERGENCY ARRHYTHMIAS- IDENTIFYING AND TREATING THEM

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ABSTRACT BODY:

An arrhythmia is a variation in the cardiac rhythm from normal sinus rhythm. Arrhythmias are commonly encountered in patients on an emergency basis and can be seen in critically ill patients. Depending on the type and underlying cause, they may be benign or malignant. Common causes of arrythmias include structural heart disease, hypoxia (seen in patients with anemia, hypotension, etc.), infectious diseases, electrolytes abnormalities and catecholamine excess (as seen in patients with a pheochromocytoma). Prompt recognition and treatment of patients with arrhythmias is crucial in those who present with malignant unstable abnormalities.

Antiarrhythmic drugs usually target two areas of the heart- the SA and AV nodal tissues which function as the pacemakers of the heart, as well as the atrial or ventricular myocardium. The SA node controls the heart rate and has autonomic rhythmicity. The AV node is a backup pacemaker and takes over when the SA node is diseased. Arrythmias originating from both locations are treated with calcium channel blockers or beta blockers. Atrial myocytes, ventricular myocytes and Purkinge cells are non-pacemaker cells but can take over as pacemakers when the SA/AV nodes are diseased or may spontaneously be aberrant pacemakers. Arrhythmias originating from these sites are treated with sodium channel blockers, potassium channel blockers or a combination of both. For patients with arrhythmias, the decision to treat or not to treat is still slightly controversial. It should be based on the type of arrhythmia (malignant or stale), clinical signs and urgency of intervention. Patients with arrhythmias should ideally be monitored with a 24-hour Holter recording or continuous ECG**Supraventricular arrhythmias** originate from the SA node, atrial tissue and AV junction. There are very many types of supraventricular tachycardias depending on where they originate from. All supraventricular tachycardias are fast and have normal looking QRS complexes, which are often tall and narrow. The P waves may be absent, present, or buried in the proceeding T waves. P waves may also have an abnormal morphology.

Atrial fibrillation is defined as a complete absence of coordinated atrial electrical activity (a bag of worms). The atria transmit multiple irregular impulses to the AV node which results in rapid irregular ventricular response rate. Heart rates of 180-220 BPM is very common. This type of arrhythmia is often associated with chronic AV valve insufficiency or cardiomyopathy. Certain dog breeds may also develop lone atrial fibrillation with no underlying cardiac causes. When the heart rate is greater than 180 BPM, the primary therapeutic intervention is to control the heart rate and any signs of heart failure. When the heart rate is less than 120, no treatment is generally needed but the patient should be closely monitored for worsening signs. Digoxin may be utilized for rate control but does not control ventricular response rate during stress or exercise. Hence it is usually used in combination with diltiazem or atenolol. Calcium channel blockers (diltiazem, verapamil, amlodipine) are also used for rate control during atrial fibrillation. Their negative inotropic and chronotropic effects are less potent than beta blockers, so they are generally safer to use when there is systolic dysfunction or heart failure. Rhythm control is uncommonly achieved to control atrial fibrillation in canine patients. Electric cardioversion has been reported in a small group of patients with mild structural heart disease or lone atrial fibrillation, however the rate of recurrence is high.

Ventricular tachycardias are arrhythmias that originate below the AV node. The QRS complexes often have a wide and bizarre biphasic morphology. Arrythmias included in the group include ventricular premature complexes, ventricular tachycardia, accelerated idioventricular rhythm, ventricular fibrillation/flutter, and torsade de Pointes. Ventricular premature complexes (VPCs) are caused by cardiac disease, altered autonomic tone, hypoxia, drugs, and idiopathic causes. With ventricular tachycardia, the heart rate is usually higher than the SA nodal rate and can lead to weakness, syncope and hemodynamic abnormalities. Animals with an accelerated idioventricular rhythm have heart rates that are about 10% faster than the underlying sinus rhythm. Those animals are not tachycardic and this rhythm is best treated with benign neglect. Ventricular tachycardias are treated with sodium channel blockers (lidocaine, mexiletine, procainamide) and potassium channel blockers (sotalol and amiodarone). Animals with ventricular fibrillation or flutter should be immediately defibrillated. IV magnesium lay lower defibrillation threshold.
Bradyarrhythmias include sinus bradycardia, sinus block/arrest, AV blocks, sick sinus syndrome and atrial standstill. An atropine response test may be performed

to determine the influence of vagal tone. Medications that can be used include terbutaline (beta agonist), theophylline and propantheline. These medications do not usually work well, and patients become refractory. Pacemaker therapy is the treatment of choice for those patients.

References available upon request

UPDATE ON THE WSAVA VACCINATION GUIDELINES

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ABSTRACT BODY:

Introduction

The next World Small Animal Veterinary Association (WSAVA) vaccination guidelines are nearing completion and are expected to be launched formally in the first quarter of 2023. Previous versions of these guidelines were published in 2007, 2010, and 2016[1]. These previous versions have been cited in the peer-reviewed scientific literature hundreds of times and downloaded by veterinarians many thousands of times. The WSAVA's Vaccination Guidelines Group (VGG) produces these guidelines. The VGG was convened to develop guidelines for the vaccination of dogs and cats intended to be helpful to companion animal veterinarians in all countries of the world. Given the breadth of this ambition, the VGG recognizes that its recommendations need to be broad. Detailed recommendations about specific vaccines and vaccination of dogs and cats that might be suitable for some countries or regions may be much less applicable elsewhere. Recommendations are based on fundamental immunological principles and good quality scientific evidence wherever possible.

The Purpose of WSAVA's Vaccination Guidelines

The purpose of WSAVA's vaccination guidelines has not changed as the latest update is prepared. They are intended to provide broad guidance in decision-making, they do not describe mandatory or minimum standards of care. They can be used by national and regional veterinary associations and individual veterinarians and veterinary practices to support development of their own vaccination schedules suitable to their own local or national conditions. Indeed, this has been done recently in multiple countries. For example, locally-authored guidelines have been produced for Israeli veterinarians that deal with vaccination of both dogs[2] and cats[3] in that country. Guidelines for the vaccination of dogs are available for Sri Lankan veterinary practitioners[4]. In some countries where national or regional vaccination guidelines have already been published, the WSAVA guidelines will be supplemental and of less specific assistance than what is already available. The American Animal Hospital Association (AAHA) and the American Academy of Feline Practitioners (AAFP) have recently updated the feline vaccination guidelines particularly relevant to North America[5]. AAHA has also produced canine vaccination guidelines particularly relevant to North America[6]. The Advisory Board on Cat Diseases (ABCD) has produced feline vaccination guidelines most pertinent to Europe[7].

A Revised Definition of "Core" Vaccines

Although plans for the next version of the WSAVA vaccination guidelines have not yet been finalised, and therefore remain subject to change, it is likely that the document will include a revised definition of "core" vaccines. The revision would be subtle, but important. If incorporated, this change would lead to redesignation of vaccines intended to protect against canine leptospirosis as core *in many but not all geographical regions*. It would lead to redesignation of feline leukaemia virus (FeLV) vaccines as core in young cats and in older adult cats with outdoor access *in many but not all geographical regions*. Local knowledge of disease prevalence (and in the case of leptospirosis, implicated serovars) would be needed to designate vaccines correctly.

Core vaccines are defined in the 2016 WSAVA vaccination guidelines as *"those which ALL dogs and cats,* regardless of circumstances or geographical location*, should receive."* Core vaccines are defined similarly in guidelines published by other large veterinary organisations. This definition is problematic because it is immediately qualified (indeed, contradicted) by stating that *"in countries where rabies is endemic, rabies vaccines are also core."* More recently, experts have suggested that vaccines intended to protect dogs against leptospirosis should also be designated as core in the many parts of the world where the disease is endemic. This is because canine leptospirosis is a zoonotic, potentially life-threatening, vaccine-manageable, infectious disease. A comparable recommendation has been made for FeLV vaccines in young cats (<1 year of age). The VGG fully supports these suggestions, especially as vaccination rates in countries where canine leptospirosis is endemic need to be improved substantially[8,9] and FeLV vaccination rates are also known to be low in some countries[10].

Another motivator is that designating a vaccine as "non-core" (i.e. using that descriptor) has been suggested to lead some veterinarians (and perhaps some owners) to view such vaccines as less important and perhaps dispensable, even when the vaccine

protects against a zoonotic and or potentially life-threatening disease. Demotivation was never an intention of vaccination guidelines authors, but may have been an undesirable, unintended consequence of this use of language.

With this background in mind, the VGG is considering removal of *"regardless of geographical location"* from the definition of a core vaccine, as it is now considered unhelpful. Core vaccines would be defined as those that ALL dogs and cats should receive, after considering the geographical areas in which they live or to which they travel. Thus, according to this proposed definition, some core vaccines would protect animals from potentially life-threatening diseases that have *global* distribution and others would protect against life-threatening diseases that are prevalent only in particular countries or regions or in animals with a particular lifestyle. The self-contradiction in the current definition would be resolved. Core vaccines for dogs *in all parts of the world* are those that protect against canine distemper virus (CDV), canine adenovirus types 1 and 2 (CAV) and the canine parvovirus type 2 variants (CPV-2). Core vaccines for cats *in all parts of the world* are those that protect against feline parvovirus (FPV), feline calicivirus (FCV) and feline herpesvirus-1 (FHV-1). In areas of the world where rabies is endemic, vaccination against rabies virus should be considered essential for both dogs and cats (i.e. rabies vaccines are **core** in those places), even if there is no legal requirement for this. Leptospirosis in dogs is another life-threatening, zoonotic disease that is widely distributed around the world including in some arid areas. In countries or regions where canine leptospirosis occurs, where relevant serovars are known, and where suitable vaccines are available, vaccination of all dogs against leptospirosis is highly recommended and the vaccines should be considered **core** in those places. In regions where FeLV is prevalent, all young cats < 1 year should all be vaccinated and older adult cats with outdoor access should be vaccinated. Thus, FeLV vaccines should be considered **core** in those regions and situations.

The 26-week Revaccination Recommendation

Plans for the next version of the WSAVA vaccination guidelines have not yet been finalised and therefore remain subject to change. Nevertheless, it is probable that the previous recommendation to bring forward the so-called "first annual booster" to 26 – 52 weeks will be revised in the next version to "26 weeks or slightly later". This is consistent with a 2020 recommendation made by AAHA/AAFP in their most recent feline vaccination guidelines, which itself built usefully upon an earlier recommendation by VGG[5].

Maternally derived antibodies (MDA) interfere with most currently available core vaccines that are administered to puppies and kittens early in life (CDV, CAV and CPV-2 in puppies, FPV, FCV and FHV-1 in kittens). MDA may prevent the young animal from mounting an active immune response. As the level of MDA varies substantially within and between litters, VGG recommends the administration of multiple core vaccine doses to puppies and kittens, with the final vaccine in the initial series being given no earlier than 16 weeks of age. Revaccination at or after 6 calendar months of age (rather than waiting until 12-16 months of age) is advised to successfully immunize the minority of animals that still had interfering MDA present at the time of their 16+ week vaccination. In situations where a pet puppy or kitten can only receive a single vaccination, that vaccination should be with the core vaccines at 16-18 weeks of age.

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CLINICAL CASES IN GENETIC TESTING AND COUNSELING: FELINE PHENOTYPE VS GENOTYPE, CANINE DEGENERATIVE MYELOPATHY, AND GENETIC DIVERSITY TESTING

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ABSTRACT BODY:

Precision Medicine, DTC Genetic Testing, & the Do's and Don'ts of Genetic Testing

Topics of the Presentation

How to use Direct to Consumer Testing (DTC)

Pros & Cons / Dos & Don'ts of DTC

What is Precision Medicine & how to use in veterinary medicine

Learning Objectives

Know when to request a genetic test

Know where to get genetic tests

Know how to use the information?

Realize the concerns with genetic tests

The first genetic tests for the domestic cat were developed over 25 years ago. Presently, 167 different mutations affecting 101 genes have been identified, producing interesting phenotypes and traits but also diseases and other health concerns. Current genetic and genomic technologies can be employed to test nearly all the known mutations in one assay (parallel genotyping) using DNA arrays, mass spectroscopy or genotyping-by-sequencing. Unlike in human medicine, where traditionally a physician had to request a genetic test, the owner of a pet can directly purchase genetic tests from a company, i.e., direct – to – consumer testing (DTC). DTC of large panels of DNA variants developed for humans and are now easily available for cats and dogs as DTC tests. Thus, no longer are genetic tests only available from the researcher who identified the mutations or a laboratory associated with a veterinary / research facility. Private companies now offer genetic tests at lower costs by using the massively parallel genotyping, however, provide little genetic counseling and education to the owner, breeder or veterinarian. These large panel tests generally do not have documented sensitivities and specificities for the mutation being tested.

How to use Direct to Consumer Testing (DTC)

Use Online Mendelian Inheritance in Animals (//OMIA.ORG) to find the researcher who found the variant – ask the researcher first! (google them!), Otherwise, preferably use a laboratory associated with a research university – invested in the science, which will provide the best education, genetic counseling and customer service.

Pros & Cons / Dos & Don'ts of DTC

Large panel DTC tests allow an effective screening method for many diseases and traits.

Not one technology can accurately genotype the various different types of mutations that cause diseases, thus, look for services that can use a second method, or use a secondary service.

Many tests are not valid in different breeds, which is not generally divulged by some services.

While some services work towards scientific standardizations, others are more profit oriented.

Don't make health & neutering decisions based on results from large panel DCT testing. Request a single gene test for a second opinion.

Precision / Genomic (P4) Medicine is an emerging approach for disease treatment & prevention that takes into account individual variability in genes, environment, and lifestyle. Precision medicine is used to understand the complex mechanisms underlying a patient's health, disease, or condition, and to better predict which treatments will be most effective – not the average or average for an ethnic group!

DNA testing is a form of Precision Medicine. Large panel genetic testing is an improvement and further facilitates the use of genetics to diagnoses and potentially treat health concerns. Whole exome and whole genome sequencing of a patient is a larger scale effort to even further advance precision medicine. Genome and exome sequencing projects are available for cats and dogs and these techniques are becoming common place for the identification of mutations that cause heritable diseases and traits.

Common inherited diseases in domestic cat breeds.

Disease / Trait (alleles) OMIA Entry (-9685)	MOI‡	Phenotype (Breeds affected)*	Gene
Acrodermatitis Enteropathica 000593	AR	Turkish Van	SLC39A4
ALPS - Autoimmune lymphoproliferative Disease 002064	AR	non-neoplastic lymphoproliferative disease (British Shorthair)	*FASL*
Centronuclear myopathy 001508	X	Neurological (Maine coon)	*MTM1*
Craniofacial Defect 001551	AR	Craniofacial Defect (Burmese)	*ALX1*
Cystinuria 002023	AR	(MCC, Sphynx, Siamese)	*SLC7A9*
Ehlers-Danlos 002165	AR	Stretchy skin (Bengal, Bombay)	*COL5A1*
Factor XI Deficiency 000363	AR	Bleeding disorder (Maine Coon)	*F11*
Gangliosidosis 1 000402	AR	Lipid storage disorder (GM1) (Korat, Siamese, S.E. Asia)	*GLB1*
Gangliosidosis 2 01462	AR	Lipid storage disorder (GM2) (Burmese)	*HEXB*
Gangliosidosis 2 01462	AR	Lipid storage disorder (GM2) (Korat)	*HEXB*
Glycogen Storage Dis. IV 000420	AR	Glycogen storage disorder (GSD) (Norwegian Forest Cat)	*GBE1*
Hermansky-Pudlak syndrome-5 002161	AR	Ocular albinism (Donskoy)	*HPS5*
Holoprosencephaly 002366	AR	Forebrain malformation (Toyger)	*GDF7*
HCM 002316	AD	Cardiac disease (HCM) (Sphynx))	*ALMS1*
HCM 000515	AD	Cardiac disease (HCM) (Maine Coon, Ragdoll)	*MYBPC*
Hypokalemia 001759	AR	Potassium deficiency (HK) (Burmese)	*WNK4*
Hypotrichosis 001949	AR	Hairless, early death (Birman)	*FOXN1*
Progressive Retinal Atropy 001244	AR	Late onset blindness (rdAC) (Abyssinian)	*CEP290*
Progressive Retinal Atropy 001613	AR	Mid onset blindness (Bengal)	*KIF3B*
Progressive Retinal Atropy 001222	AR	Early onset blindness (Persian)	*AIPL1*
Polycystic Kidney Disease 000807	AD	Kidney cysts (PKD) (Persian)	*PKD1*
Polycystic Kidney Disease 2 002525	AR?	Polycystic kidney disease (Siberian)	*PKD2*

Disease / Trait (alleles) OMIA Entry (-9685)	MOI‡	Phenotype (Breeds affected)*	Gene
Pyruvate Kinase Deficiency 000844	AR	Hemopathy (PK Deficiency) (Abyssinian, Siberian, MCC)	*PKLR*
Skeletal dysplasia 002485	AR	Skeletal dysplasia (British)	*LTBP3*
Spasticity 001621	AR	Congenital myasthenic syndrome (CMS) (Devon Rex)	*COLQ*
Spinal Muscular Atrophy 000939	AR	Muscular atrophy (SMA) (Maine Coon)	*LIX1-LNPEP*

‡ Mode of inheritance of the non-wild type variant. *The breed affected represents the breed in which the DNA variant was identified. Other breeds may be affected if they have common ancestry, especially other breeds in a breed family. Not all transcripts for a given gene may have been discovered or well documented in the cat, DNA variants presented as interpreted from original publication. A "+" implies the wildtype allele when known. In reference to the variant allele, AD implies autosomal dominant, AR implies autosomal recessive, co-D implies co-dominant, X implies X-linked. OMIA: Online Mendelian Inheritance in Animals (http://omia.org) entries provides links to citations and clinical descriptions of the phenotypes and the diseases.

CLINICAL CASES IN GENETIC TESTING AND COUNSELING: FELINE PHENOTYPE VS GENOTYPE, CANINE DEGENERATIVE MYELOPATHY, AND GENETIC DIVERSITY TESTING

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ABSTRACT BODY:

Commercial genetic testing is becoming increasingly available and utilized by owners, breeders, and veterinarians. Official documentation of genetic testing allows for verification and validation (including identification of the testing center). The following examples will discuss the clinical implications of genetic testing and their use, including genetic counseling in clinical practice.

1) Feline Phenotype versus Genotype: Pyruvate kinase (PK) deficiency

Historically, genetic testing for specific disease-causing variants has been limited to the breed of discovery and genetically related breeds. Large panel, direct-to-consumer (DTC) genetic tests are now available for cats from non-research commercial companies. DTC testing can determine allele frequencies of DNA variants in larger and more diverse populations. Thus, stronger data can be ascertained for many DNA variants, either supporting or disputing their association with a disease or phenotypic trait. In cats, DNA variants for pyruvate kinase deficiency, mucopolysaccharidosis VI, Factor 12, hypertrophic cardiomyopathy and charcoal coloration need specific attention to determine their effect on causing disease in individual cats.

Pyruvate kinase (PK) deficiency

PK deficiency is documented in Abyssinian and Somali cats. In contrast to affected dogs, PK deficient cats have an intermittent rather than chronic hemolytic anemia. Bilirubin gall bladder stones and post-hepatic obstruction are also characteristics in PK deficient cats. A single nucleotide substitution (c.693+304G>A) in intron 5 of the PKLR gene, which has evidence of impacting splicing, is associated with the manifestation of PK-deficiency. Several population studies of the PKLR intronic variant have indicated a higher prevalence of the variant in Abyssinian, Somali, Bengal, Egyptian Mau and Singapura cat breeds, which are breeds associated with the Abyssinians. More recently, Siberians and Maine Coons have been identified with the PKLR variant. Three of 10 Maine Coon cats homozygous for the PKLR variant were reported as having episodes of anemia, however, detailed clinical evaluations have not been conducted. Sanger sequencing and whole genome sequencing has not indicated a different variant within PKLR, considering full exons and introns, that may be more likely causal for disease. In addition, the 99 Lives cat genome sequencing consortium dataset indicates the variant is overall rare within cats, as does other population studies.

Recommendation to veterinarians and breeders

The suggested causal variant for PK deficiency is overall rare in cat populations around the world and most common in breeds associated with Abyssinians / Somalis. The identification of the variant in non-Abyssinian associated breeds, Maine Coons and Siberians, is likely due to cross-breeding. Cats with overt pyruvate kinase deficiency need to be whole genome sequenced to provide any additional support for the variant, however, the allele frequency data, the lack of other highly suspect variants within PKLR, and the identified transcript splice variants support the variant as causal for PK deficiency. Commercial laboratories are encouraged to conduct follow-up on homozygous cats to acquire additional data to support or dispute the candidate variant, as well as provide more thorough and proactive genetic counseling to cat owners. PK deficiency should always be considered in the differential diagnosis of anemia in cats worldwide, including genotyping of the PKLR variant. Although health concerns due to the variant and the disease are unpredictable, cat breeders are strongly encouraged to eradicate this variant from their population, particularly since the allele frequency is low and the new cat populations of concern, Siberians and Maine Coons, have large populations of breeding cats, thus, genetic bottlenecks are of lesser concern.

Clinical Case: 5 year old spayed female Abyssinian with anemia, lethargy and inappetence.

2) Canine Degenerative myelopathy and SOD1 genetic testing.

The clinical disease of degenerative myelopathy has been recognized as an infrequently encountered

disease of progressive (months to a year) hind end paresis and paralysis primarily in German Shepherd Dogs and Boxers. It is sporadically seen as a familial disease in Pembroke Welsh Corgis, Cardigan Welsh Corgis, Bernese Mountain Dogs, Rhodesian Ridgebacks, Borzoi and Chesapeake Bay Retrievers. It is rarely diagnosed in other breeds or mixed-breed dogs. In 2009, researchers at the University of Missouri USA found that all pathologically confirmed clinically affected dogs had two copies (autosomal recessive) of a SOD1 gene variant. Subsequent testing of large numbers of purebred and mixed-breed dogs showed that this variant is the most frequent variant present in all dogs. It occurs at a gene frequency of 7.77% across all mixed-breed dogs, and 5.41% across all pure-bred dogs in over 120 breeds. The frequency of the SOD1 variant is over 20-90% in more than 30 breeds, although no dogs in most of these breeds have ever been confirmed with DM.

With the rapid utilization of commercial genetic testing, owners are being provided with information that their dogs are "at risk" of developing degenerative myelopathy, regardless of their breed or mixed-breed background. This places an emotional burden on the owners of a fatal disease in their family pet that will likely never occur.

In working up older patients with hind end weakness, there are many differential diagnoses to work with – most with methods for successful treatment. As veterinarians we need to make decisions based on our clinical examination and available testing modalities (imaging, genetic testing) to arrive at a proper diagnosis.

Clinical case: 11 year old neutered male Boxer with recent progressive hind end weakness and ataxia.

3) Canine & Feline Genetic Diversity Testing

Several commercial genetic testing companies provide diversity testing in domestic cats and dogs. Each service has different genetic markers and methods, different sample databases and different statistical algorithms for their evaluations and interpretations of the genetic data from a specific individual. The genetic markers have different capabilities, and the sample database may or may not be highly representative of a particular breed or population.

Genes come on paired chromosomes – one from the sire and one from the dam. If a gene pair has two copies of the same gene this is called homozygosity. If a gene pair has two different variations of a gene it is called heterozygosity. Within a breed, there can be several variations of a gene available in each gene pair. Unless a gene variant is either desirable or deleterious then its homozygosity or heterozygosity has no effect on the quality or health of the dog or cat.

There is misunderstanding of the meaning of breed-wide genetic diversity, how it is measured, and how it can be maintained. Commercial genetic testing companies market "genetic diversity tests" based on an individual's amount of homozygosity. Homozygosity measurements do not reflect genetic diversity, but only the average type of mating being performed. If a handful of individuals are mated to the ones most related to each other, then the next generation will have higher homozygosity. If the same individuals are mated to the ones least related to each other then there will be lower homozygosity. This has not changed the genetic diversity of the population – they are the same individuals and have the same gene frequencies within the breed. Breed-wide genetic diversity means maintaining dogs and cats from different areas of the gene pool (different pedigree backgrounds) in the breeding population.

Any selection causes a loss of genetic diversity in what is being selected against. A loss of genetic diversity is not always detrimental to a breed unless its loss causes a decrease in health or quality. If a breed standard has certain requirements, or if breeders want to select against certain detrimental traits or disorders then you need breed-wide homozygosity at that gene location to reproduce the desired phenotype (what you see in the animal). It is homozygosity that allows a breed to breed true. Mars/Genoscoper data shows that purebred dogs on average have higher homozygosity (44.87%) versus mixed-breed dogs (35.98%). This indicates that it takes approximately 35.98% homozygosity to be a dog, but on average an additional 8.89% of homozygosity to be a purebred dog.

The markers used by the companies are not self-evident, nor are the population databases used to obtain the range of genetic diversity and the average homozygosity of a population. Significant biases can occur in the sample databases as they are populated by the cats and dogs who participate in the testing. Without specific and focused genetic counseling, breeders then think it best to breed their dog or cat to others with low homozygosity, thinking this will make the offspring even more genetically diverse. However, if the individuals have the same alleles at the same markers, this practice could actually increase homozygosity in the offspring. Uniformly outbreeding to create heterozygosity will cause a homogenization of the breed, lowering the differences and diversity between individuals and ability to improve health and quality through direct selection.

Some breeds have a higher average homozygosity but based on valid health surveys are relatively healthy.

Some breeds have lower average homozygosity and have considerable issues with hereditary disease. It is not the homozygosity or heterozygosity of a breed that determines its health, it is the breed-wide accumulation of disease predisposing genes.

Clinical Cases: Diversity panel reports from commercial laboratories.

NASOPHARYNGEAL DISEASE IN CATS

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ABSTRACT BODY:

Nasal Diseases

Clinical signs associated with nasal diseases in cats include nasal discharge, sneezing, epistaxis, facial deformity, and depigmentation. *Unilateral nasal discharge* is more likely to be associated with, foreign body, tumors, nasal stenosis, or oronasal fistula, whereas *bilateral nasal discharge* occurs more in cats with nasal tumors, fungal, allergic or lymphocytic-plasmocytic rhinitis, and nasal stenosis. Neoplasia is more common in older cats, whereas infectious and parasitic diseases, and allergies are more common in younger animals. Facial deformity is associated with tumors and cryptococcosis.

The first step in the clinical approach to nasal disease in cats is to classify the rhinitis as destructive or non-destructive. This is best achieved during CT examination. It easily classifies the problem as destructive or not, and provide information about cribriform plate integrity, extension of the problem, and soft tissue involvement. Nasal radiographs are of limited value when dealing with nasal disease in cats, but may show bone and turbinate destruction, as well as provide evidence of presence an intranasal mass.

Rhinoscopy are helpful if there is a history of unilateral nasal discharge, high risk for a nasal foreign body, further evaluation of a mass, and for obtaining biopsy specimens. The retropharynx should also be directly examined with a flexible scope or a mirror and a light source. Biopsy specimens can also be obtained blindly, without the need of rhinoscopy. It is important to measure the needle during blindly biopsy to prevent accidentally sampling the brain. Cryptococcus titers should be obtained in all cats with chronic nasal disease.

The most important causes on *non-destructive rhinitis* in cats are: acute feline upper respiratory tract disease, parasitic rhinitis, foreign bodies, lymphocytic-plasmacytic rhinitis, and oronasal fistula. Destructive rhinitis are usually secondary to feline chronic rhinosinusitis, fungal rhinosinusitis, or neoplasia.

Non-Destructive Rhinitis

Lymphoplasmacytic Rhinitis (LP Rhinitis)

Feline LP rhinitis is a disease with an unclear pathogenesis characterized by lymphoplasmacytic (LPC) inflammation and progressive tissue destruction. It is not as common in cats as it is in. LP rhinitis is also known as idiopathic or allergic rhinitis. Severity of signs is associated with reduction of IgA expression. The disruption of mucosal defense mechanisms generating cycles of tissue inflammation, tissue damage and repair with progressive loss of function. Disease progression is independent from viral infections.

Most affected cats have mucoid od mucopurulent nasal discharge. There are no systemic signs and nasal airflow tends to be preserved. Diagnosis is one of exclusion and is based on the persistence of clinical signs confirmed by gross and microscopical lesions in the absence of any other primary or systemic condition associated with chronic rhinitis. Nasal biopsies reveal lymphoplasmacytic infiltrates, mucosal edema with no apparent etiology. The best therapy is not known. The disease is often resistant to therapy, and control of clinical signs, rather than cure, is the goal of therapy. Nonspecific treatment includes nebulization with saline and the use of saline nasal drops to help loosen secretions, especially in dry environments. The nares should be kept free of dried discharges. Broad spectrum antibiotics (e.g., amoxicillin-clavulanate, clindamycin, or doxycycline) may be used to control secondary bacterial infections. One must be careful with chronic or repeated use of antibiotics and the potential for development of resistance. Some cats may respond to orally or inhaled glucocorticoids. Those should be used with caution as they may exacerbate viral and bacterial components of the disease. Antihistamines such as clemastine, hydroxyzine or chlorpheniramine can also help a few cats.

Feline respiratory disease complex

Feline respiratory disease complex refers to the characteristic acute presentation of a contagious respiratory or ocular disease caused by one or multiple pathogens Most commonly associated microorganisms are Feline herpesvirus 1 (FHV-1), Feline calicivirus (FCV), *Chlamydia felis, Mycoplasma*, and *Bordetella*.

Calicivirus is associated with oral ulcers, whereas herpesvirus can lead to corneal ulcers. *Chlamydia* infections are always accompanied by conjunctivitis. Bordetelliosis may occur in cats that live with dogs. It typically causes mild clinical signs

(cough, sneezes, nasal and ocular discharge) in cats that resolve within 10 days. Any cat with an acute onset of upper respiratory signs or conjunctivitis or both and a history of recent exposure to other cats should be suspected to have infectious FRDC, and the suspicion is increased in kittens and poorly vaccinated cats. Etiological diagnosis of a specific pathogen as the cause of the disease is not as simple. PCR is useful in cases of outbreaks when multiple cats can be tested. In a single individual, presence of an organism does not correlate with disease.

Nonspecific treatment includes, Supportive care, local hygiene, maintenance of hydration and nutrition, humidification, and stress reduction. Antibiotics can be used to control of secondary bacterial infection and in cases associated with *Bordetella*, *Chlamydia* or *Mycoplasma*. Most antibiotics work and doxycycline works against the 3 most common bacteria. Altering local immunity has been attempted with intranasal vaccines for FHV-1 and FCV and use of Interferon-alpha 2b. Unfortunately, there was no untreated controls in the Interferon study. Topical (Cidofovir) and systemic (Fancyclovir) antivirals appear to be safe and at least partially effective. Lysine supplementation has been advocated to reduce viral shedding with conflicting results. Maropitant and probiotics have also been used, but their efficacy have not been properly evaluated.

Destructive Rhinitis

Feline chronic rhinosinusitis

Feline chronic rhinosinusitis is a very common problem in cats associated with nasal discharge, excessive mucus production, and sneezes. It is likely multifactorial and associated with FHV-1 and secondary bacterial infection, as well as failure of the local immune response. Clinical signs include bilateral nasal discharge with no systemic signs. It is an exclusion diagnosis with lymphoplasmacytic inflammation in the histopathology. Presence of eosinophilic infiltrates suggest an FHV-1-related disease. Antibiotics can be used to treat secondary infections. Anti-inflammatory agents such as piroxicam and glucocorticoids can be helpful in some cats, whereas antiviral medications like Famciclovir can be helpful in cats with FHV-1-related disease

Cryptococcosis

Is an infectious disease caused by the Cryptococcus fungus. *Cryptococcus neoformans* is associated with pigeon's droppings and it is the most widely disseminated. *Cryptococcus gatti* is associated with bark and leaf litter from eucalyptus trees and is the most common agent in a few defined areas. Mammalian infection occurs by inhalation and cats usually are presented with chronic nasal disease. It is a destructive rhinitis that can lead to facial asymmetry. It occurs most commonly in young cats. Diagnosis can be achieved by finding the organism in cytology or histopathology. However, antigen detection by latex agglutination in blood is the test of choice because it is fast, reliable, and minimally invasive. This test can also be used to monitor therapy.

Surgery is helpful for treatment of large granulomas on the skin, oropharyngeal, and nostrils. Fluconazole and itraconazole can be used to treat nasal and systemic infections. Fluconazole appears to be more effective for infections involving the CNS and eyes. It is also better tolerated by cats than itraconazole. Amphotericin B is the treatment of choice for CNS infection or systemic disease. There is a significant risk of nephrotoxicity with this drug. Therapy should be continued until 1 month post resolution of signs or a decrease in titer by 2 orders of magnitude. Median duration of therapy is 8.5 months.

Nasopharyngeal Diseases

Nasopharynx is the cavity caudal to the choanae above the hard and soft palate The most common diseases affecting the nasopharynx are tumors, polyps, and nasopharyngeal stenosis. Nasopharyngeal tumors are usually an extension from a nasal tumor.

Nasopharyngeal Polyps

Polyps are nonneoplastic pedunculated growth that affects young cats. The growth originates in the epithelial lining of the tympanic bulla and the auditory tube. The classic presentation is a young cat (average 1.5 years) with stertorous breathing. Maine coon are at a higher risk. Additional signs include otitis, nasal discharge, sneezing, and reverse sneezing. Signs are chronic and may or may not be progressive. Removal by traction of surgery is the treatment of choice. Prognosis is good with removal, with a recurrence rate of 50% with traction alone. Recurrences are more common in patients where there is middle ear involvement on radiographs.

Nasopharyngeal Stenosis

It is a pathologic narrowing within the nasopharynx. It can be congenital or secondary to chronic rhinitis, aspiration rhinitis, surgical manipulation, trauma, tumor, and polyps. Most common cause in cats is chronic rhinitis. Median age at presentation is 2.4 years, ranging from 4 months to over 16 years. In 90% of cats, there is patency in the stenosis. Most common clinical signs are stertorous breathing, dyspnea, obligatory open mouth breathing, nasal discharge, gagging, and repeated swallowing attempts. Diagnosis can be obtained by rhinoscopy or CT examination. Thin

lesions (< 5mm) can be treated with balloon dilation with 50% success in cats. Thicker lesions or in cases where balloon dilation was unsuccessful, placement of a stent is recommended.

WHAT DRUG DO I USE FOR THIS UNSTABLE PATIENT? SEDATION AND ANALGESIA IN THE ER.

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ABSTRACT BODY:

Sedation and analgesia are important considerations for critically ill patients that present to veterinary professionals. These patients may demonstrate signs of pain, anxiety or aggressiveness. Depending on the presenting complaint, they may also have significant concurrent co-morbidities (respiratory distress, hypotension, anemia, seizures) that make the choice of drugs to utilize important.

In critically ill patients, it is importance to prioritize sedatives that have minimal effects on the hemodynamic system. The goal of sedation is to get the patient more relaxed in the safest way possible. Sedated critically ill animals should be closely monitored with ideally continuous ECG and a blood pressure while they are sedated. Whenever possible, continuous pulse oximetry should also be utilized if tolerated by the patient. Opioids are commonly used for sedation and analgesia of critically ill patients. They have minimal effect on the cardiovascular window making them excellent choices for patients with hypotension. They have a wide therapeutic window and are readily available. Pure mu receptor opioids can also be reversed if excessive sedation is a concern. Full mu receptor opioids (fentanyl, hydromorphone, methadone) are excellent for analgesia. While they do not provide as much sedation as butorphanol (a mu receptor antagonist and kappa receptor agonist), they can be used alone for both sedation and analgesia in critical patients. They may also be combined with a benzodiazepine to allow for more profound sedative effects. Buprenorphine, the partial mu receptor agonist, is good for mild to moderate pain but likely has the least sedative effect of all the opioids. Butorphanol (a mu receptor antagonist) provides excellent sedation but should not be used in painful patients because of its limited analgesic effects.

Benzodiazepines (midazolam, diazepam) provide good sedation and muscle relaxation. They have an excellent cardiovascular profile and are safe to use in animals with hemodynamic instability, cardiac disease or respiratory disease. They can cause hyperexcitement (usually seen in healthy or anxious patients) but provide good sedation in critically ill patients. Midazolam can be administered IM since it is water soluble and can be combined in the same syringe with other sedatives, unlike diazepam. Acepromazine provides good sedation in dogs and cats but is usually used in combination with an opioid when analgesia is needed concurrently. Acepromazine may affect the hemodynamic system due to vasodilation thus it should be avoided in unstable patients. Hypotension is rarely seen when administered at low doses (0.01-0.05 mg/kg IV or IM)

Alpha-2 agonists like dexmedetomidine may be considered for sedation in critically ill patients. It should be avoided in patients with degenerative valve disease, systolic dysfunction or patients with hypotension. The author prefers to use micro doses (0.5-1 mcg/kg) as an augment to other sedative drugs in critically ill patients who are hemodynamically stable and refractory to other sedatives administered. Ketamine may be used to provide sedation at low doses without causing respiratory depression. It is rarely used alone due to its ability to cause myoclonus but can be combined with a benzodiazepine or opioid to provide excellent sedation. Ketamine also provides analgesia although its analgesic effects also work more effectively when combined with an opioid or a sodium channel blocker like lidocaine. Propofol and alfaxolone are two other induction agents that can be utilized to provide sedation in critically ill patients. They are both able to cause profound respiratory depression so the practitioner should be ready to intubate any patient being sedated with either of these drugs. They can also impair the hemodynamic system and cause hypotension. Both propofol and alfaxalone are short acting so they may need to be titrated to effect when used for long procedures. They have no analgesic effects and should be used with an opioid when analgesia is desired.

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ACUTE PAIN ASSESSMENT IN DOGS AND CATS

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ABSTRACT BODY:

Cats

Despite significant developments and availability of feline specific analgesics, their pain is still undertreated. One of the main reasons for this is the difficulty in recognizing and "measuring" pain in populations that cannot "self-report." Lord Kelvin stated that "if you cannot measure it, you cannot improve it" and this approach should be taken when treating pain in our veterinary patients. To treat pain, we must first look for it, recognize it and quantify it in some way so we can assess the efficacy of our interventions. Pain is a complex multidimensional experience with both sensory and psychological components. The sensory-discriminative component is "how it feels" (type, source and intensity of pain) and the affective-emotional component is "how does it make the animal feel?" Pain is a conscious emotion and is always unpleasant. The International Association for the Study of Pain (IASP) have updated their definition of pain which now states that "*pain is a distressing experience associated with actual or potential tissue damage with sensory, emotional, cognitive, and social components.*"

In humans who can self-report, pain is what the patient says it is but in neonates, cognitively impaired people and animals, pain is what the observer says it is. As animal caregivers, we make assessments on the patient's behalf which puts an extra burden on us, to "get it right."

Acute pain assessment

There have been many attempts to correlate objective measurements such as heart rate and blood pressure with pain. In cats, no study has found a consistently reliable objective measure, which is not surprising since these parameters can be affected by many factors other than pain[1]. Cats suffer from "white coat" syndrome just as humans do; for example, fear and the stress of a journey to a veterinary hospital will alter heart rate, respiratory rate and blood pressure[2].

Mechanical nociceptive threshold testing (algometry) is a useful technique for evaluating both primary (wound) and secondary (remote areas unrelated to the wound) hyperalgesia. In research studies mechanical threshold testing differentiated between treated and control groups of cats following feline castration[3]. These findings suggest that an assessment of wound tenderness should be incorporated into the overall assessment of post-operative pain. Observation from a distance and interacting with the animal are both essential components of an evaluation. A cat may remain very still and quiet because moving hurts; without interaction these patients will be overlooked.

Currently there is no gold standard for assessing pain in cats. Basic pain scales include simple descriptive scales, numerical rating scales and visual analogue scales, these are considered unidimensional scales as they do not assess all the complex aspects of pain. Any system that is used must be valid, reliable and sensitive. Without strictly defined criteria and instructions to follow during assessment, and training of observers, many scoring systems are highly variable.

Multidimensional or composite systems are particularly important when self-reporting is not possible. However, these systems must incorporate components that have been proven as sensitive and specific indicators of pain in the species being studied. Knowledge of the normal behavior for the individual being evaluated is helpful and deviations from normal behavior may suggest pain, anxiety or fear, or some combination of stressors. Normal behaviors should be maintained post-operatively if a cat is comfortable. Grooming is a normal behavior but licking excessively at a wound or incision can be an indicator of pain. The occurrence of new behaviors or the loss of a normal behavior, should raise a "red flag" that initiates further investigation and assessment.

Acute pain assessment tools for cats

The original UNESP-Botucatu scale has been reworked to make it quicker and easier to perform in clinical practice and has been validated in cats with different causes of acute pain[4].

The Glasgow Composite Measures Pain Scale-Feline[5] has been updated to include facial expressions[6,7]. This tool was developed using cats undergoing different types of surgery or with medically related pain.

The major assessment domains in cats include:

- Vocalization
- Posture
- Attention to the wound

- Response to people
- Response to palpation of the wound or painful area
- Facial expressions
- Overall demeanor

Facial expressions of pain

Facial expressions have been studied in humans, including newborns to measure emotional states. Similar studies are now being done with a wide range of mammalian species and many "grimace" scales are now available. Each animal, including humans has different facial action units based on the anatomy of their face and facial muscles, therefore the details of a "pain face" will be different in each species but there are remarkable similarities across species that include the eyes, jaw, muzzle, ear and head position.

The Feline Grimace Scale

The Feline Grimace Scale (FGS) has been published in full[8]. There is also a dedicated website with additional information including a training manual: www.felinegrimacescale.com

The FGS also shows good agreement between different users including owners, veterinarians, veterinary students and nurses[9]. You can also download the Feline Grimace Scale Phone App from Google Play or at the APP store.

There are 5 Action Units to score:

- Ear position
- Orbital tightening
- Muzzle tension
- Whisker position
- Head position

Each is scored as 0, 1, or 2 making 10 (2 x 5) the maximum score. Intervention is recommended at a score of ≥ 4.

Using pain assessment tools in feline practice

Each clinic should choose a scoring system that fits their specific needs, and this may require some trial and error. Whichever one is chosen, it should be user friendly, quick to complete and easily performed by all veterinary care team members and it should be an integral part of the cat's evaluation. After temperature, pulse and respiration are recorded, pain, which has been coined the "fourth vital sign," should also be assessed. A scale should include both non-interactive and interactive components and rely heavily on behavior.

The severity of surgery or trauma, the patient's response to analgesic therapy and the expected duration of action of the analgesic drug(s) administered will help to determine the frequency of evaluations.

For example, if a cat is resting comfortably following administration of an opioid, it may not need to be re-assessed for two to four hours. Cats should be allowed to sleep following analgesic therapy. Vital signs can often be checked without unduly disturbing a sleeping animal. Undisturbed observations coupled with periodic interactive observations (e.g., palpation of the wound) are likely to provide more information than only occasionally observing the animal through the cage door. Routinely using a pain assessment tool enhances the care of patients in the perioperative period.

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ACUTE PAIN ASSESSMENT IN DOGS AND CATS

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ABSTRACT BODY:

Dogs

Recognition and quantification of acute pain in dogs is pivotal to adequate pain management. Unless pain is recognized it will not be treated effectively. Similarly, quantification of pain is key to ensuring that pain is not over or under treated with analgesics. Tailoring analgesic drug administration to the level of pain is vital to minimize the risk of analgesic drug side effects and this can only be achieved with the use of pain scoring tools to quantify the severity of pain. Unfortunately, recent studies[1,2] have indicated that use of pain scoring tools is not common in 1st opinion practice, and this is an area where improvements could have great benefits for animal welfare.

Detecting pain

We are currently primarily reliant on behavioural changes in order to detect pain in dogs. However in order to detect changes in behaviour it is important that the assessors are aware of normal behaviour in dogs and in that particular animal. For example, in the dog that becomes fear aggressive after surgery this could be related to pain, particularly if the dog did not show any signs of aggression and was pain free before the procedure. Abdominal pain is often characterized by a dog exhibiting a hunched appearance, with an unwillingness to settle or rest in the kennel. Cranial abdominal pain, such as pain caused by pancreatitis, is typically shown by the dog adopting a praying position, where the dog is stretching with the forelimbs on the floor and the hindlimbs standing upright. Lameness is an indicator of limb pain although bilateral lameness can be more difficult to detect clinically. When there is an obvious wound or painful area then it is important to palpate gently around the wound to see if there is a reaction e.g. turning to look at the painful area, snapping, or tensing the area being palpated. The aim of this gentle palpation is to detect secondary mechanical hyperalgesia. Hyperalgesia is when there is a heightened pain response to a normally painful stimulus and is an indicator of central sensitization. Similarly it is important to detect allodynia (a pain response to a normally non painful stimulus such as touch) in the area of injured tissue because this is also an indicator of central sensitization. Once central sensitization has occurred more aggressive pain management is needed to provide adequate pain control.

Quantifying pain

Historically, unidimensional tools such as simple descriptive scales, numerical rating scales and visual analogue scales have been used to quantify pain in dogs. These scales are simple to use but only take into account the sensory component of pain (where does it hurt and how much does it hurt?). When quantifying pain it is also important to take into account the emotional impact of pain in that animal e.g. the depression, anxiety and fear associated with the pain. This enables more comprehensive pain management and better global care of the patient. Unidimensional tools are also unvalidated and it is important to use tools that have undergone validation when assessing pain. More recently composite pain scales have been developed for quantification of pain in dogs. These have been devised using psychometric principles and are designed to measure both the sensory and the emotional component of pain. The most widely used pain assessment tool is dogs is the Short Form of the Glasgow Composite Pain Scale[3]. This tool is validated and easy to use, taking approximately 5 minutes to complete per assessment per patient. When using the tool it walks you through a logical approach to assessing a dog for pain; first looking at the dog from outside of the kennel and observing its behaviour; then opening the kennel door and interacting with the dog; then looking to see if the dog has any lameness; then applying gentle palpation around the wound and finally making an assessment of the dogs demeanor and emotional status. There are 6 questions in total with a maximum possible score of 24 (if the dog can be taken outside and walked) or 20 (if the dog is non ambulatory). Importantly the cut off scores for intervention have been validated[4]. A score of greater than or equal to 5 out of 20 or a score of greater than or equal to 6 out of 24 indicates that's rescue analgesia is needed. The main problem with the Glasgow Composite Pain Scale is that dogs that are very sedated tend to score highly on the scale so that this is a confounding factor. It is important to assess the dog's level of sedation when carrying out a pain assessment; if the dog is very sedated and therefore generates high scores on the Glasgow Composite Pain Scale consider that it may not be necessary to give intervention analgesia.

Who should carry out pain assessment and how frequently should it be carried out?

Anyone in the practice who is trained in pain assessment in dogs using the Glasgow Composite Pain Scale is suitable to assess pain in dogs using this tool. The team of people that spend most time looking after the dogs in their kennel environment are ideally placed to carry out pain assessment because they are more likely to recognize changes in behaviour from normal in the individual animal. They will also be able to detect variations in behaviour during the day in response to the administration of analgesic drugs. It is important that the scale is properly applied, with all questions completed to give a total score. In terms of how frequently pain should be assessed; this depends on the individual patient and the type of analgesia regimen that has been used. Usually starting pain assessment around 30 minutes after the end of anaesthesia is appropriate, once the patient is awake enough to make a meaningful assessment. After this the frequency of pain assessment should range from every 2 -4 hours, with the more frequent assessment being applied to dogs that are judged to be suffering from moderate to severe pain and where analgesic management is challenging. It is also prudent to assess pain frequently in dogs that have received a local anaesthetic block and are receiving further systemic analgesics on a per required need basis. Four hourly assessment is suitable for most patients receiving systemic methadone, morphine or buprenorphine as an analgesic.

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WHEN AND HOW TO ENGAGE CLIENTS ON LIFE STAGE NUTRITION FOR THEIR PETS

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ABSTRACT BODY:

Adequate nutritional management is very important throughout the whole life of dogs and cats. Different life stages have specific nutritional and energy requirements (1), which can be further affected by other factors, such as health status, neuter status, and activity level.

As a result, navigating the choice of diet for different life stages can be very complex, made even harder by the multitude of diets available for purchase, conflicting marketing messages, the high volume of information on the internet, and others. Additionally, there is not one single diet that works for all dogs/cats (the same way that one dog/cat might do well with multiple dietary choices). Pet owners need and want information, but a high proportion of them do not pursue it from their veterinary team (2), which is not necessarily in the best interests of the pet. The veterinary team, therefore, must be proactive in discussing nutrition. There are some topics that can be sensitive, and it is important that the veterinary team has both the knowledge to discuss these and a strategy to best communicate the feeding plan that would work best for the patient.

Tips

- Performing a nutritional assessment (3) in every visit gives the veterinary team the chance to bring up nutrition into the conversation and to show the pet owner that the team takes the nutritional status of their pet seriously. Moreover, the information provided by the assessment will allow for a tailor-made nutritional assessment: the veterinary team knows the patient, which places us in the position to give the best advice for that particular pet.
- Use visits to proactively discuss nutrition for each life stage: vaccination and deworming visits for growth, yearly visit for adults and seniors. The neuter appointment is the best moment to discuss obesity prevention and teach pet owners what to look out for. Pets with high obesity risk (some breeds, low activity, neuter, some diseases) should receive a specific weight management plan and be weighed and body condition scored more frequently. As the pet ages, look for milestones (4) that would signal ageing and specific issues that might be addressed with nutrition, such as body composition changes, dental disease, changes in mobility and activity, and the presence of nutrient-sensitive disease.
- Use the whole veterinary team to perform the nutritional assessment and communicate nutrition recommendations, in order to solidify them.
- Visuals are great for many situations, such as discussing weight management (like body condition score charts, pictures of pets at different body conditions). Having these available for the clients to take home is useful (or providing some links to online resources). You can also have other information to take home like growth charts (to monitor puppy and kitten growth), which is very useful to prevent future obesity (5,6) and, in larger breeds, orthopaedic disease (7)
- I recommend using forms to obtain a diet and environmental history, which helps save time and likely helps get more information (they can fill it at home, at their leisure, with direct access to whatever they feed). Once in the clinic, get even more details by the use of open-ended questions such as “walk me through a typical day of feeding your pet, morning to night”. Consider asking specifically about food items that might not be perceived as foods, such as medication vehicles. Creating a non-judgemental environment is important to properly assess the dietary management and to give useful advice.
- After the nutritional assessment is performed, instructions should be given written down for the pet owners to consult later. Diet recommendations should be specific. Some options can be given, tailoring these to pet and owner preferences, as long as the manufacturers meet quality criteria. The goal is that everyone be satisfied with the choice: the veterinary team (a safe and nutritious complete diet), the pet (a palatable diet) and the pet owner (a quality diet that adjusts to their preferences and budget).

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NONTRAUMATIC HEMOPERITONEUM: DIAGNOSIS TO OUTCOME

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ABSTRACT BODY:

New discussion regarding nontraumatic hemoabdomen centers around 1) unique causes, 2) odds of malignancy for bleeding splenic masses [do we believe the dogma?], 3) predictors of short-term survival, 4) necessary preoperative diagnostics, and 5) advanced methods for splenectomy.

Acute, nontraumatic hemoabdomen frequently occurs in dogs due to spontaneous bleeding from parenchymal organs, specifically the spleen or liver. Emergency surgical treatment is indicated these animals, however there should be awareness of other causes of spontaneous hemoabdomen (e.g., coagulopathy, anaphylaxis) where surgery is not warranted.

Splenic tumors occur far more frequently in dogs than in cats. Hemangiosarcoma, hemangioma, and hematoma are the most common lesions requiring splenectomy. Other less common splenic tumors include fibrosarcoma, leiomyosarcoma, mast cell tumor, and lymphoma. Tumors in the liver are classified as primary, hemolymphatic, or metastatic. Metastatic tumors are most common in the dog, while hemolymphatic tumors predominate in the cat. Primary hepatic tumors are generally uncommon, but hepatocellular carcinoma is most common in the dog and can become quite large, whereas cats are mostly affected with biliary carcinoma. Prediction of splenic malignancy is not as simple as once believed. The dogmatic “two-thirds/two-thirds” rule may not be accurate with more recent published information.[1] A simple risk score for hemangiosarcoma was developed based on four parameters (body weight, platelet count, total protein level, and thoracic radiographic changes).[2] The incidence of hemangiosarcoma in dogs with nontramatic hemoabdomen was 36%, 76%, and 96% for low, medium, and high-risk groups, respectively. Other factors that may influence the likelihood of malignancy include species (dog vs. cat), breed of dog, size of mass, and single versus multiple nodules.

Prior to surgical intervention for bleeding abdominal organs, the extent of preoperative diagnostics is dependent on the status of the animal and the goals of the owner. At a minimum, complete blood count, biochemistry panel, and thoracic radiographs should be performed. Thrombocytopenia is not unexpected, but if available further coagulation parameters should be evaluated. The need for abdominal ultrasound and fine-needle aspiration is debatable. A recent study found the cavitated appearance of a splenic mass not to be associated with malignancy, while another study found a low level of sensitivity for abdominal ultrasound compared to surgery or necropsy for detecting grossly identifiable lesions in dogs with nontraumatic hemoabdomen.[3,4]

If indicated, the timing of surgical intervention is controversial, with some choosing to attempt hemodynamic stabilization prior to general anesthesia while others choose to take an animal to surgery as soon as possible. The author prefers the latter but works in a high-level academic institution with broad resources and acknowledges potential limitations for emergency surgeries in other settings.

At surgery, splenectomy or large hepatic mass resections require a generous ventral midline abdominal incision. The spleen can be removed with traditional methods using suture to ligate small bundles of the splenic vessels. The main splenic artery and vein should be ligated separately, and often circumferential and transfixing sutures are used on to ligate the artery. As an alternative, a vessel-sealing system (LigasureÒ) can be used to expedite spleen removal, but the large vessels larger than 7 mm should be ligated individually. The author removes the spleen along the splenic mesenteric border regardless of technique. This avoids issues with inadvertent ligation of the left gastroepiploic vessels or technical challenges with the large splenic artery and vein. In addition, this may anecdotally lessen the risk of postoperative portal vein thrombosis. Preoperative hypercoagulability was found to be common in a small subset of hemodynamically stable dogs with splenic masses.[5] Laparoscopic-assisted splenectomy has been described in dogs. Case selection is crucial for this technique and should be avoided in cases of hemoabdomen.[6] Risk of developing gastric dilatation-volvulus syndrome following splenectomy is debatable in the scientific literature. Decision to perform this procedure is based on other risk factors for GDV and the stability of the animal under general anesthesia.

Liver tumors are best and most safely resected by removing the affected liver lob using stapling techniques. However, other techniques for removal include using vessel-sealing system, harmonic scalpel, large encircling ligature(s), and skeletonization with hemoclips. Location of the liver mass plays a role in resectability, as left divisional masses are often straight-forward, while masses of right and central division may be challenging to due proximity to the posthepatic caudal vena cava and short hepatic veins.

The perioperative prognosis for nontraumatic hemoabdomen is good to excellent. Dogs presenting with hemoperitoneum regardless of the underlying cause had a reported perioperative mortality of 16%.[7] Major perioperative complications include marked ventricular arrhythmias, disseminated intravascular coagulation, and venous thrombosis. Long-term prognosis following splenectomy or liver lobectomy is variable depending on the histopathology of the mass and whether the animal was presented with hemoperitoneum. Median survival for dogs with splenic hemangiosarcoma is only several months even with adjuvant therapy. In contrast, dogs with massive hepatocellular carcinoma have a median survival over 4 years.

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STATE-OF-THE-ART LECTURE: UTILIDAD DE LA ECOGRAFÍA PARA EL DIAGNÓSTICO DE LA PANCREATITIS AGUDA EN EL PERRO

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ABSTRACT BODY:

INTRODUCCION

La pancreatitis se considera el trastorno más común del páncreas exocrino en la especie canina, y se define como la inflamación del páncreas exocrino. Tradicionalmente la histopatología se ha considerado el gold estándar para el diagnóstico de la pancreatitis y para diferenciar la pancreatitis aguda (PA) de la pancreatitis crónica (PC). La pancreatitis aguda se define como una inflamación del páncreas exocrino que no está asociada a cambios histopatológicos permanentes, como la fibrosis y atrofia, mientras que la ausencia de tales cambios junto con una reacción inflamatoria es más indicativa de una pancreatitis aguda.

Sin embargo, aunque la histología es el gold estándar del diagnóstico de las pancreatitis raramente se emplea en la clínica debido a su naturaleza invasiva.

A pesar de las numerosas investigaciones en diversas pruebas de diagnóstico, el diagnóstico de la pancreatitis sigue siendo un reto. Normalmente se utiliza una combinación de los resultados de varias modalidades de diagnóstico como son la historia clínica, hallazgos de la exploración física, hallazgos de diversas pruebas específicos para el páncreas (lipasa pancreática canina) y los hallazgos ecográficos o de imagen avanzada para diagnosticar la pancreatitis aguda.

El objetivo de esta charla es evaluar la utilidad de la ecografía para el diagnóstico de las pancreatitis agudas en el perro.

EMPLEO DE LA ECOGRAFIA PARA EL DIAGNOSTICO DE LAS PANCREATITIS AGUDAS EN EL PERRO

La ecografía es la técnica de imagen más utilizada para el diagnóstico de las PA, aunque actualmente hay artículos donde evalúan la utilidad de la Tomografía Computarizada. La ecografía es una técnica que está disponible en la mayoría de las clínicas y tiene una gran utilidad para evaluar la cavidad abdominal. Sin embargo, tiene una desventaja que es una técnica operador dependiente, lo que hace que influya en los resultados del examne

Los primeros artículos donde se refieren a la utilidad y hallazgos ecográficos de la pancreatitis datan de los años ochenta y hacen referencia a pancreatitis experimentales, describiendo los hallazgos que observáron como masas no homogéneas y pérdida de la ecodensidad. En 1989 se describe ecográficamente la dilatación del conducto pancreático como un signo ecográfico de PA en dos perros, posteriormente este signo no ha vuelto a ser descrito en el perro.

En 1998 Hess y cols realizaron la descripción de los hallazgos ecográficos de 70 perros con PA siendo los más frecuentes un páncreas hipoecogénico con el tejido de alrededor hiperecogenico. Además, también visualizaron el páncreas con aspecto normal en casos de PA, concluyendo que la evaluación ecográfica del páncreas no es suficiente para descartar la PA en los perros. En este trabajo achacaron estos hallazgos con la experiencia y la formación del radiólogo, ya que como hemos dicho previamente la ecografía es operador dependiente.

Hasta el año 2014 las publicaciones sobre los hallazgos ecográficos de las pancreatitis han sido escasos. Sin embargo, desde entonces hasta la actualidad las investigaciones han ido aumentando, posiblemente por la mejor calidad de los ecógrafos que hace que obtengamos mejores imágenes del páncreas, y de sus alteraciones. La exploración ecográfica del páncreas no siempre es fácil debido al gas que puede haber en el estómago y duodeno y la morfología del órgano. Por lo tanto, no solo hay que pensar en la habilidad del ecografista sino también en la tecnología que tenemos actualmente.

En muchos estudios se ha evaluado la sensibilidad y especificidad de la ecografía, así en un estudio realizado hace más de 20 años describieron que la sensibilidad de la ecografía para la detección de pancreatitis mortal grave fue del 68%.

Sin embargo, en un estudio reciente se demostró que la sensibilidad y la especificidad de la ecografía para el diagnóstico de PA varía mucho según el número de alteraciones ecográficas que se tengan en cuenta. Los hallazgos ecográficos de PA descritos en el perro son aumento de tamaño del páncreas, parénquima hipoecoico (focal o difuso) y grasa mesentérica perilesional hiperecogénica. Si sólo se observa una de estas tres alteraciones, la sensibilidad y la especificidad de la ecografía para la detección del PA fueron del 89% y del 43% respectivamente. Si se visualizaban dos de

las alteraciones, la sensibilidad y la especificidad eran del 78% y el 69% respectivamente. Por otro lado, cuando se observaban las tres alteraciones al mismo tiempo la sensibilidad y la especificidad eran del 43% y el 92% respectivamente. Por lo tanto, la ecografía no permite un diagnóstico definitivo de PA y un páncreas normal no excluye la PA en los perros. Así, cuando hay una sospecha clínica de PA y en el examen ecográfico inicial el aspecto del páncreas es normal se recomienda realizar un nuevo examen entre las 24h- 4 días, ya que el aspecto del páncreas puede variar con el tiempo. Esto puede relacionarse en la fase de la fisiopatología de la pancreatitis en la cual se encuentra cuando se realiza la primera ecografía.

Además, de los hallazgos ecográficos descritos en el páncreas también puede observarse líquido peripancreático, obstrucción biliar extrahepática, quistes, pseudoquistes y abscesos pancreáticos. Y en los últimos años se ha descrito edema de la pared gástrica, además del duodeno corrugado.

La ecografía con contraste es otra técnica mejora la detección de la perfusión tisular, así como el patrón vascular de la lesión de un órgano. Se han realizado algunos estudios utilizando contrastes ecográficos para valorar las características de perfusión y los patrones de realce que presenta el páncreas de perros con pancreatitis. En los perros con pancreatitis aguda, la media de píxeles y la intensidad máxima del parénquima pancreático fue significativamente mayor que la de los perros normales. Y todos los perros con pancreatitis mostraron una disminución de la intensidad de los píxeles 10-15 días después del primer examen. Así, el uso de la ecografía con contraste puede ayudar al diagnóstico de PA.

En conclusión, aunque en los últimos años se ha producido un gran avance en la tecnología de los ultrasonidos, y se van adquiriendo cada día mejores imágenes del páncreas, y del resto de estructuras abdominales, los hallazgos ecográficos por si solos no son suficientes para realizar el diagnóstico de pancreatitis aguda. La ecografía con contraste podría ayudar, pero es una técnica que actualmente no se utiliza de forma rutinaria. Por ello, debemos seguir utilizando la combinación de signos clínicos, exploración física, análisis de lipasa pancreática y ecografía abdominal para realizar el diagnóstico de pancreatitis aguda en el perro.

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EL FUTURO DEL MERCADO PET

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ABSTRACT BODY:

EL futuro del mercado pet y vet es maravilloso, es decir, será más grande en dos puntos: calidada y cantidad. Más peros y gatos y otras mascotas, ademas que estarán cada vez más cerca de los humano, presentes en los hogares, en las camas. De hecho serán cada vez más miembros de la familia, y asi quererán tratarlos mejor, con más gastos, mejores tecnologias a suas mascotas. Los veterinarios deven se preparar para esto, y también a plan de salud, pagos mensualmente por el tutor. Telemedicina será realidad, ya es. Nuevos grupos de invertidores están se propagando en el mundo vet, comprando clpinicas y hospitales. Se llama merger and acquisition (M&A), que ya se pasa en muchos paises. El veterinario empresario deve estar atento a esta situación, prepararse para vender, hacer sua valuation (cuanto vale, cuesta su empresa) ser buena, vender por precios altos; trabajar hoy para llegar hacia este punto, poner sus KPIs en órden.

ACUTE KIDNEY INJURY: BECAUSE TIME IS OF THE ESSENCE

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ABSTRACT BODY:

The term “acute kidney injury” (AKI) has been recently adopted in human medicine to better reflect the broad spectrum of disease of the kidney that encompasses a continuum of functional and parenchymal damage from clinically undetected subcellular damage to fulminant excretory failure. It is important to note that acute renal failure is more specific to an advanced phase of AKI where abrupt decline in more than 75% of renal function leads to retention of uremic toxins and dysregulation of fluid, electrolyte, and acid-base balance.

The diagnosis of acute kidney injury still rests upon the detection of changes in serum creatinine and BUN levels, which only occur if more than 50-75% of glomerular filtration is lost and is often delayed by more than 24 hours. Plasma urea and creatinine concentrations can also be influenced by non-renal factors such as dehydration, diet, gastrointestinal bleeding, muscle mass, muscle activity and metabolism. Such diagnostic delays and limitations make the implementation of early therapy and recognition nearly impossible. In response to these limitations, there has been an increased effort to identify novel early biomarkers of AKI (e.g. NAG, NGAL, Cystatin C, KIM-1, IL-18, Clusterin). In veterinary medicine, the diagnosis of AKI is based on the identification of azotemia on biochemical analysis (elevated BUN and creatinine) along with poorly concentrated urine on urinalysis (usually isosthenuria).

The causes of AKI can be broadly grouped into three major categories: decreased renal blood flow (pre-renal causes), direct renal parenchymal damage (intrinsic renal causes), and obstructed urine flow (post-renal causes). Pre-renal or hemodynamic causes of AKI are directly associated with decrease in renal perfusion or excessive vasoconstriction. Primary renal etiologies of AKI may be caused by disease processes affecting the glomeruli, renal tubules, interstitium, or vasculature. Glomerular disease, or glomerulonephritis (GN), is a disease that most commonly affect dogs and not cats. Inflammation of the glomerulus is typically induced by immune-mediated complex deposition or due to systemic factors (e.g. thrombosis), which are often secondary to disease somewhere else in the body. Renal tubular injury can be the result of exposure to nephrotoxic drugs or toxins (e.g. aminoglycosides, ethylene glycol, lilies, heavy metals, and grapes/raisins), metabolic disease (e.g. hypercalcemia), endogenous agents such as myoglobin or hemoglobin. Interstitial nephritis is often associated with infectious diseases such as leptospirosis or pyelonephritis. Post-renal, or obstructive causes of AKI include any disease process that affects the flow of urine. In veterinary medicine, cats are most frequently affected by this type of acute renal insult. In cats, common conditions that lead to post-renal AKI include urethral obstruction from feline lower urinary tract disease –FLUTD-; and ureteral obstruction.

Treatment and Monitoring:

Identify and correct pre-renal factors: adequate fluid resuscitation is essential to the restoration of cardiac output, systemic blood pressure and renal perfusion in patients with AKI. Prompt treatment can avert or limit subsequent kidney damage. Fluid therapy in animals with AKI should be aimed to rapidly correct hydration deficits and ongoing losses. In hypovolemic and hypotensive animals, shock doses of fluids should be promptly initiated. Goal directed fluid therapy with clearly defined goals: correction of dehydration, hypovolemia, hypotension and markers of poor perfusion such as lactate; and careful monitoring of patient body weight and urine output should be instituted within the first 24 hours.

Liberal administration of chloride-rich fluid solutions (e.g. 0.9% NaCl) might be associated with an elevation in mean serum creatinine level, worsening of renal injury and an increase in the need for renal replacement therapy. The administration of large quantities of chloride anion could lead to a reduction in glomerular filtration rate via a tubule-glomerular feedback mechanism and afferent arteriolar vasoconstriction. Use of colloids such as 6% hydroxyethyl starch, for fluid resuscitation in patients with AKI has also been associated with increased in-hospital mortality and an increased need for renal replacement therapy compared with crystalloid fluids (e.g. LRS) in severely ill people.

Antibiotics: dogs are more likely to develop infectious causes of AKI compared to cats and are likely to have a better prognosis than animals with other intrinsic causes of renal injury. Once samples have been collected for urinalysis, urine culture and serology, intravenous antibiotic therapy should be instituted

until results of these testing prove no evidence of infectious causes or another cause of AKI is identified. The antibiotic of choice is Ampicillin or Ampicillin-Sulbactam (Unasyn) as it treats systemic leptospirosis and covers a good spectrum of gram positive, gram negative and anaerobic bacteria in the case of pyelonephritis. Ampicillin does not eliminate the carrier state of leptospirosis, so once confirmation of this disease has been made, doxycycline should be started.

Identify and treat acute complications: pharmacological treatment of life-threatening hyperkalemia is focused on translocation potassium into the intracellular space and none of these therapies increases the excretion of potassium out of the body. Therefore, the efficacy of these treatment is modest and transient. In cases of urinary flow obstruction, these therapies will provide a window of opportunity to relieve the obstruction. In cases of anuric or oliguric AKI, these therapies may not be enough to stabilize the animal and dialysis may be required. Pharmacological agents to treat hyperkalemia include IV dextrose administration either by itself or followed by insulin, sodium bicarbonate, or terbutaline. Calcium gluconate is used in severe hyperkalemia to increase the resting membrane potential of cardiac cells but does not change potassium concentrations. Most patients with AKI present with a degree of metabolic acidosis due to the decreased ability of the kidneys to reabsorb bicarbonate and excrete hydrogen ions, as well as the contribution of lactic acid build up secondary to fluid loss and decreased perfusion. Acidosis is partially corrected with fluid therapy and restoration of hydration and perfusion.

Review other drugs: stop nephrotoxic agents (e.g. NSAIDs, aminoglycoside antibiotics, radiocontrast media), adjust doses and monitor concentrations when appropriate.

Accurately monitor fluid balance: fluid overload is a common reason for therapeutic failure in animals with AKI. Ins and outs should be monitored at least every 6 hours with quantification of the urine production via either urinary catheterization, collecting naturally voided urine or weighing cage bedding. Using body weight to measure net fluid gain or urine output (weighing before and after urination) is a non-invasive tool commonly underutilized in veterinary medicine. Body weight should be measured at least twice daily.

Early nutritional support: ensuring the provision of optimal amount of energy, protein and micronutrients in people with AKI has proved to improve survival. The main goals of nutritional support is to preserve lean body mass, avoid further metabolic derangements, support the immune system and support gastrointestinal health. The use of feeding tubes early in the diagnosis of AKI is imperative in order to increase the chance of a good outcome in small animals.

Renal replacement therapies: renal replacement therapies (dialysis) is the most efficient means of managing uremic, acid-base, electrolyte and fluid overload complications associated with AKI. Dialysis is indicated when signs of uremia are uncontrolled, azotemia worsens despite conventional medical management or when anuria/oliguria lead to fluid overload. Despite efficacy in reducing and controlling signs of uremia, hemodialysis is expensive and not widely available.

Other therapies: the current evidence from randomized controlled trials and observational studies suggests that furosemide is unlikely to be able to improve renal function or mortality directly. First, it is important to emphasize that furosemide can increase urine output without improving the creatinine clearance and renal function. Fenoldopam administration at 0.8 g/kg/min in dogs and 0.5 g/kg/min in cats has been proven to be relatively safe but has not been associated with improved survival to discharge or renal recovery. Based on currently available evidence, in hypotensive vasodilated animals with acute kidney injury, restoration of blood pressure within autoregulatory values should occur promptly with norepinephrine and be sustained until such vasodilatation dissipates. Dopamine has no advantages over norepinephrine and is not as reliably effective in restoring blood pressure and urine output. Its widespread use cannot be supported in patients with vasodilatation and acute kidney injury, especially in cats where dopamine receptors may not be functional or not exist at all.

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QUESTIONS AND ANSWERS ABOUT THE LATEST VACCINATION GUIDELINES

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ABSTRACT BODY:

Introduction

The next World Small Animal Veterinary Association (WSAVA) vaccination guidelines are nearing completion and are expected to be launched formally in the first quarter of 2023. Once again, a selection of frequently asked questions, with answers, will be included. Previous versions of these guidelines were published in 2007[1], 2010[2], and 2016[3]. This 2022 WSAVA Congress programme includes two sessions presented by the WSAVA Vaccination Guidelines Group (VGG) chairman. These two sessions will consider aspects of the WSAVA canine and feline vaccination guidelines.

Although vaccination is a very well-established element in preventative healthcare and primary veterinary practice, it is a changing field, with new scientific evidence and novel products continuing to emerge. It is crucial that veterinarians remain up to date with the newest recommendations in order to provide the highest quality healthcare for their clients and for the animals under their care.

This second of two sessions will provide an open forum and the opportunity for attendees to ask questions of the speaker about any facet of the WSAVA vaccination guidelines. Attendees may also wish to share their own experiences about vaccines and vaccination that may be particular to their own home country or region.

The 2016 WSAVA vaccination guidelines document included 110 frequently asked questions and their answers. The VGG is in the process of updating, merging and rationalising these. A small selection of some of the most frequently asked questions, with answers, is provided below. These are provided as examples only. You are encouraged to pose whatever questions interest you most.

Selected Questions and Answers

Q. I know that maternally derived antibodies (MDA) can prevent active immunization with MLV vaccines - but can they also block immunity to killed vaccines?

Yes. MDA can block certain killed vaccines. If the killed product requires two doses, as is often the case, and the first dose is blocked by MDA, then the second dose will not immunize on its own. In this circumstance, the second dose will prime (if not blocked), and a third dose would be required to immunize successfully.

This is not true for MLV vaccines where, in the absence of MDA, it only takes a single dose to immunize successfully. Nevertheless, two doses are often recommended, particularly in young animals, to be sure one is given when MDA has waned and cannot block. That is why in the puppy or kitten series, the last dose should be given at 16 weeks of age or older.

Q. I have been told that certain canine MLV combination core products need only be given twice, with the last dose at an age as young as 10 weeks. Is that accurate?

The VGG is aware that certain canine vaccines are licensed for such an 'early finish' in order to allow puppies the benefit of early socialization. The VGG recognises and respects the high importance of puppy socialization. However, we have reservations about the immunological validity of this approach to vaccination. No combination core product currently available will immunize an acceptably high percentage of puppies, particularly not against CPV-2, when the last dose is given at 10 weeks of age. The VGG advises that wherever possible the last dose should be given at 16 weeks of age or older, regardless of the number of doses given earlier. Where the 'early finish' protocol is adopted, the VGG recommends that owners carefully control the exposure of their pup to restricted environments and only permit contact with healthy and fully vaccinated puppies and adult dogs.

Q. Are there mutants of CDV or CPV-2 in the field against which currently-available vaccines cannot provide protection?

Not as far as we know. All of the current CDV and CPV-2 vaccines provide protection from all the known isolates of CDV or CPV-2, respectively, when tested experimentally, as well as in the field. This is not controversial for CDV. However, there is a report in the literature of an outbreak of CPV-2c in Italian dogs that were vaccinated with a MLV vaccine containing CPV-2. There is another Italian report of a vaccinated, aged dog developing CPV-2c related disease.

Q. Can certain CPV-2 vaccines immunize pups with MDA at an earlier age than other CPV-2 vaccines?

Yes. Certain CPV-2 vaccines with higher viral titres (i.e. mass of virus in the vaccine ampoule) and/or with more immunogenic isolates or constructs (regardless

of variant) will immunize quite a few weeks earlier than other "ordinary" CPV-2 vaccines.

Q. When a Leptospira vaccine is used, should it be a product with two serogroups or one with more than two serogroups (e.g. three or four component products available in some countries)?

Ideally, a commercial vaccine that contains all of the serogroups that cause disease in dogs in that region should be used. Unfortunately, such vaccines are not available for many regions. In other regions there is insufficient knowledge of which serogroups are circulating in the canine population. The VGG would encourage collection and dissemination of such data.

Q. Do Leptospira vaccines give long term (e.g. years of) immunity and are they highly effective, like the core canine viral vaccines?

No. Leptospira vaccines provide short-term immunity (e.g. 12-15 months) and efficacy may be only partial. Leptospira products may prevent clinical disease but fail to protect against infection and shedding of bacteria, especially when infection occurs more than 6 months after vaccination.

Q. Can nosodes (holistic preparations) be used to immunize pets?

Nosodes are homeopathic vaccines. They do not immunize because they do not contain antigen. So, no. Nosodes cannot be used for the prevention of any disease.

Q. What does the VGG think of the use of canine enteric coronavirus vaccines?

The VGG does not recommend the use of canine coronavirus vaccines as there is insufficient evidence that these vaccines are protective, nor indeed that enteric coronavirus is typically a significant canine pathogen in adult dogs. Variant strains of this virus have sporadically been reported to cause severe disease in adult dogs and puppies in various parts of the world. However, it is unclear that the available vaccines would protect against these variants. The identification of coronavirus with a test kit does not necessarily mean it is the cause of any concurrently present enteric disease.

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DEALING WITH BRACHYCEPHALIC DOGS IN YOUR CLINIC

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ABSTRACT BODY:

Brachycephalic Dogs and Brachycephalic Obstructive Airway Syndrome (BOAS)

Brachycephalic dogs are dogs with congenital and acquired deformities resulting from selective efforts to diminish craniofacial bone morphology with no regard to preservation of function or whether the existing structures will fit in the reduced head size. Several canine breeds of various sizes are brachycephalic. This includes small and miniature breeds like: Boston terrier, French Bulldog, Lhasa apso, Pekinese, Pug, and Shih tzu; as well as medium to large breeds: Boxer, English Bulldog, Bull mastiff, and Shar pei. Several of those breeds are among the top 15 most popular AKC breeds in 2021.

Brachycephalic dogs are at an increased risk for numerous morbidities, and the potentially inability to breathe adversely affects their welfare. *Brachycephalic Obstructive Airway Syndrome (BOAS)* is a chronic, debilitating respiratory syndrome, where soft tissue and abnormal turbinates blocks the airways. All brachycephalic breeds suffer from some degree of BOAS. BOAS risk and severity increase sharply in a non-linear manner as relative muzzle length shortens. It occurs primarily in dogs whose muzzles comprises less than half their cranial length. Thicker neck girths and obesity also increase BOAS risk and severity. English bulldog, French bulldog, Boston terriers, and pugs are the breeds most commonly affected.

BOAS is a progressive disorder of the upper airway that is characterized by *primary anatomical abnormalities* (stenotic nares, elongated soft palate, distortion of pharyngeal soft tissues, nasopharyngeal turbinates, macroglossia, redundant pharyngeal folds, and hypoplastic trachea) causing resistance to airflow and restricted breathing. This leads to increased negative intraluminal pressure during inspiration, exacerbating soft tissue abnormalities, and leading to *secondary anatomical changes*: everted laryngeal saccules, pharyngeal hyperplasia, tonsillar hyperplasia, laryngeal edema and granuloma, protrusion of the dorsal tracheal membrane, bronchial collapse and eventually, laryngeal collapse. The increased inspiratory effort resulting from the airway obstruction leads to laryngeal and pharyngeal collapse, laryngeal edema, protrusion of the tracheal membrane, and bronchial collapse. The resulting decease in intrathoracic pression increases the risk of regurgitation, hiatal hernia, and aspiration pneumonia. English and French bulldog with BOAS are almost 4 times more likely to develop aspiration pneumonia than other non-brachycephalic breeds. It is important to remember that BOAS is a progressive disease, and it is better to address it earlier rather than later.

Brachycephalic dogs are also at a high risk for heatstroke. The canine nasal cavity plays an important role in the dog's ability to regulate its temperature, carrying the released heat away from the body when the dog pants. This process is hindered in BOAS-affected dogs that have an obstructed nasal cavity. As a result, BOAS-affected dogs cannot exchange heat as easily as healthy dogs when their body temperature rises during exercise. In addition, these dogs have decreased ability to loos heat through convection and evaporation and have lower body surface in relation to body mass. Thus, they are at high risk of developing heatstroke after exercise, in presence of high environmental temperatures, or when they are in the hospital. It is a real emergency that can happen very fast, triggered by stress, anxiety, fear, and tachypnea.

Functional Evaluation of BOAS

A Functional evaluation of BOAS severity was developed by the Cambridge Veterinary School (https://www.vet.cam.ac.uk/boas/about-boas/recognition-diagnosis#clinical-assessments). It is based on results prior and after Exercise Tolerance Test (ETT). The ETT consists of trotting the dog at a speed of 7 – 8 kilometers/hour for 3 minutes. Dogs fail if they refuse 3 times due to inability to exercise. Results are graded based on 3 parameters: 1) Respiratory noise, 2) Inspiratory effort, and 3) Dyspnea, cyanosis and syncope before and after the ETT.

Assessment: Grade 0 are dogs that are normal before and after ETT in all parameters. Grade 1 dogs are normal before ETT, and after ETT show mild respiratory noise with no effort to mild inspiratory effort. Grade 2 patients have mild to moderate respiratory noise or inspiratory effort before ETT. They should not have dyspnea, cyanosis or syncope. After ETT, Grade 2 dogs can show, moderate to severe respiratory noise or inspiratory effort and mild dyspnea. Cyanosis and

syncope should not be present. Grade 3 patients have moderate to severe respiratory noise, inspiratory effort, and dyspnea at rest. They may or may not have cyanosis or syncope and inability to exercise. After ETT they can show severe respiratory noise, inspiratory effort, and dyspnea and may or may not have cyanosis or syncope

Recommendations: Grade 0 patients are *BOAS free*. Annual recheck is recommended for dogs under 2 years of age. Grade 1 dogs are *clinically unaffected*, with mild respiratory signs. Annual rechecks are needed for dogs under 3 years of age. Grade 2 dogs are *BOAS affected with moderate respiratory signs.* Required management include weight loss and potentially surgical intervention. Grade 3 dogs are considered *BOAS affected with severe respiratory signs*. Management includes a thorough evaluation and surgical intervention.

Dealing with Brachycephalics

Puppies

Brachycephalic puppies should be properly socialized. Some are stubborn and can become aggressive. It is also easier and less stressful to deal with well-behaved patient in the hospital. Clients should be educated. Many don't realize that their animals are abnormal and think that the respiratory noises and the inspiratory effort are not a problem or sign of disease. Palliative surgery for elongated soft palate and stenotic nares should be considered for BOAS Grade 2 or 3 sooner, rather than later. Information about heatstroke and stress-related problems should be provided, and body weight should be kept in the ideal range for the breed and size.

Hospital patients

Brachycephalics in the hospital are always a problem. A Brachycephalic Risk Score (BRisk) has been developed to predict risk of complications in dogs presenting for surgical treatment of BOAS (https://onlinelibrary.wiley.com/doi/10.1111/vsu.13291). It can be adapted to be used in all brachycephalic dogs admitted to the hospital. Increased risk for complications occurs in underweight and obese dogs, dogs with low temperature on presentation, history of previous surgeries, and level of compromise at admission.

Excessive barking and tachypnea while in the kennel, during restrain or anesthesia increases the risk of airway edema, obstruction, and non-cardiogenic pulmonary edema. Airway edema compromises thermoregulation and increases the risk of heatstroke. Any brachycephalic with tachypnea should be considered an emergency. They should be sedated, temperature should be monitored, and immediate cooling implemented if temperature starts to raise, Brachycephalics are also difficult to restraint, and it is not easy to muzzle them. They also have poor veins and get dyspneic in lateral or dorsal recumbency.

Steps in the hospital

- Listen to the patient. If you can hear the patient breath, you need to act
- Check the temperature. Patients with low temperature at admission are at a higher risk for complications. Body temperature increasing during hospitalization is an emergency that requires immediate action
- Sedate the patient. It is important that they are calm while in the hospital. Anxiety is a risk for regurgitation after anesthesia and for heatstroke. The ideal dose is the one that calms the patient without placing them in recumbency. Several drugs can be used, and you must give preference to the ones you are familiar with. Butorphanol: 0.2 mg/kg IV every 1 – 2 h prn is a good first choice. Acepromazine: 0.02 mg/kg IV every 4 – 6 h prn can be added in case butorphanol alone wasn't enough. It is important to realize that acepromazine has been associated with increased postsurgical mortality in English Bulldogs. Gabapentin: 10 – 20 mg/kg PO each 8 h is another viable option. Avoid opioids. They decrease the esophageal sphincter tone and gastrointestinal motility. This leads to an increased risk of vomiting and regurgitation.
- Position the patient in sternal recumbency. It increases pulmonary capacity and the concentration of dissolved oxygen. The head should be kept high, if possible, to decrease passive reflux and aspiration.
- Prevent aspiration syndromes because brachycephalic dogs are at high risk for aspiration pneumonia. Prokinetics like Cisapride can be used at 0.3 to 0.5 mg/kg PO each 8h. Omeprazol (0.5 to 1.5 mg/kg PO each 24 h) can be used to decrease stomach pH. Antinausea medications (Maropitant: 1 mg/kg IV or SC every 24 h or Ondansentron: 0.5 to 0.1 mg/kg IV every 12 h). Maropitant also decreases the activity of the airway nociceptors.
- Prevent occlusion of the airways with glucocorticoids (dexamethasone: 0.15 mg/kg IV) or topical alpha-1A adrenoceptor agonist (Oxymetazoline 0,05% intranasal). Nebulization with 0.9% Na Cl solution using a well-fitted mask can also be helpful. In case of edema, 5 ml of hypertonic saline can be added, Medications should never be added to the nebulization protocol.
- Discharge as soon as it is safe.

References available upon request.

TERAPIAS DE REEMPLAZO RENAL - HEMODIALISIS Y DIÁLISIS PERITONEAL

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ABSTRACT BODY:

Las terapias de reemplazo renal se utilizan para el manejo de injuria renal aguda que no cede al tratamiento convencional, previo a esto hay que tener en claro cual es el factor agudizante de esta patologia ya que sin esta informacion ninguna terapia de reemplazo servira. Estas terapias pueden ser de tipo intra y extra corporea. Ambas terapias son poco usadas en clinicas veterinarias ya que se piensa que son complejas, sin embargo, conocerlas nos va a permitir entender la utilidad de estas.

Si bien es cierto, la terapia renal extracorporea es compleja y se necesita equipamiento y entrenamiento para realizarla, la dialisis peritoneal es simple y de facil manejo en la clinica diaria, es por eso que vale la pena compararlas y entenderlas.

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A TRANSFORMATIONAL APPROACH TO MANAGING ALLERGIES TO CATS

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ABSTRACT BODY:

Introduction

Human allergies to cats are rising in prevalence globally. In addition to the obvious health impacts on allergy sufferers, human allergies to cats can impair quality of life for both the allergy sufferer and for the cat.

Allergies to cats are the most common mammalian-origin allergy in humans; they are estimated to affect approximately 1 in 5 adults worldwide, but the prevalence is increasing. Allergies to cats frequently manifest with ocular and nasal allergy symptoms, and can compromise the allergy sufferer's quality of life due to poor sleep quality, fatigue, reduced alertness, lower work productivity and concentration, and mood changes.

This presentation provides an overview of cat allergens, and current management approaches as well as an introduction to an innovative approach that safely and effectively reduces the major cat allergen.

Fel d 1, the major cat allergen

Fel d 1 is produced primarily in the salivary and sebaceous glands, and is spread throughout the cat's hair during grooming and shed into the environment with hair and dander. Once in the environment, Fel d 1 easily becomes and remains airborne on dander and dust particles. The allergen is very "sticky" and because it is passively transferred on clothing, the allergen is ubiquitous: potentially symptom-inducing levels of Fel d 1 have been documented in private vehicles, public transportation and buildings, and even homes without cats.

All cats produce Fel d 1 regardless of breed, age, sex, housing (indoors vs outdoors), body weight, or hair color, length or pattern; there are no truly hypoallergenic or allergen-free cats. Fel d 1 production varies widely among cats, and even in the same cat over the course of the year.

Intact male cats produce 3-5 times less Fel d 1 after neutering, suggesting an influential role of testosterone. Although its biological function has not been definitely determined, a pheromone/chemical signaling role has been proposed.

A transformational approach: neutralizing the major cat allergen at its source

To date, existing available approaches focus on intervening at the environmental level (to reduce allergen load) or at the patient level (e.g., with medications to prevent or mitigate allergy symptoms).

A company that attempted to produce hypoallergenic cats through selective breeding claimed a 96% success rate, but the business shut down in 2015.

Immunization of cats to induce autoantibodies to Fel d 1 resulted in reduction of Fel d 1 in lacrimal secretions as well as clinical improvement in cat-allergic individuals and longer petting times for 10 owner-cat pairs in an open-label, non-placebo-controlled trial.

Because the biological function for Fel d 1 for the cat is currently unknown, the potential health and welfare effects of ceasing its production are also unknown

Leveraging established IgY technology

Immunoglobulin Y (IgY) is the avian counterpart of mammalian immunoglobulin G (IgG), and is naturally formed by hens in response to exposure to various antigens.

IgY is transferred to the egg and concentrated in the egg yolk, to provide passive immunity for the chick. Purina researchers investigated the possibility of utilizing IgY technology to inactivate Fel d 1 and reduce its allergenicity.

IgY neutralizes Fel d 1 in vitro and in cat saliva samples ex vivo

Initial benchtop assays demonstrated that Fel d 1 must be bound at multiple epitope sites to result in steric hindrance that prevents its binding to Fel d 1-specific IgE on the mast cells of sensitized individuals (thereby preventing Fel d 1 from triggering an IgE-mediated allergic reaction).

Anti-Fel d 1 IgY is safe for cats

All egg products that contain egg yolk also contain IgY produced against a number of antigens to which the hen has been exposed, and egg products are common ingredients in many pet foods. Specifically targeted IgY have been used safely in human and veterinary medicine for decades. For this approach to managing Fel d 1, an egg product containing anti-Fel d 1 IgY was coated on a complete and balanced dry cat diet after extrusion.

A randomized, controlled, blinded feeding trial was conducted: more than forty cats were divided into groups that received dry diets containing up to many

times the amount of anti-Fel d 1 IgY used in the efficacy studies described below. After six months of feeding, there were no clinically relevant differences between the control group and any of the feeding level groups.

Diet with anti-Fel d 1 IgY reduces active Fel d 1 in cat saliva in vivo

An initial trial group of six healthy, adult domestic shorthair (DSH) cats received a control diet (without anti-Fel d 1 IgY) for a two-week baseline period, followed by 6 weeks on a test diet (an egg product ingredient containing anti-Fel d 1 IgY coated on the control diet). Saliva samples were collected at 1, 3 and 5 hours post-feeding. The timing of Fel d 1 collection did not significantly affect the levels of Fel d 1, and the average decrease in active Fel d 1 over the 6-week test feeding period compared to baseline was 29.57%.

In a second trial, saliva was collected from twenty healthy, adult DSH cats 5 hours after their morning feeding, 5 days per week. Following a one-week baseline period on the control diet (a complete and balanced dry cat diet), 11 of the 20 cats were fed the test diet (as described above) and the remaining 9 cats were fed the control diet for 4 weeks. Beginning in the third week of feeding the test diet, salivary active Fel d 1 levels were reduced by an average of 24% compared to baseline, while the control group reduced by only 4% (which can be explained by normal variation in Fel d 1).

Diet with anti-Fel d 1 IgY reduces active Fel d 1 on cats' hair and dander

To evaluate if this new technology works in a real situation, 105 cats healthy DSH cats were fed a control diet for 2 weeks, followed by the test diet (same as above) for 10 weeks. Hair and dander were collected by brushing the cats; hair was collected four times during the baseline (control diet) period, then weekly during the test diet period. Active Fel d 1 levels in the hair and dander were significantly reduced starting in the third week of the test period, and remained at reduced levels for the remaining time. The reduction in active Fel d 1 on the cats' hair and dander ranged from 33-77%, with an average reduction of 47% compared to baseline.

Human clinical impact of a feline diet with anti-Fel d 1 IgY

Eight healthy, adult DSH cats were fed either a control diet (n = 4) or the test diet (same as described for previous studies) for 8 weeks. During the last four weeks of feeding the diet, blankets used by the cats as bedding were collected and used to load individual environmental chambers.

Eleven adult human participants completed the study.

Following a high-Fel d 1 priming exposure, subjects were randomly assigned to either the control exposure (blanket from control diet-fed cat) or test exposure (blanket from test diet-fed cat) the next week and exposed to the opposite condition two weeks later.

Compared to the high-Fel d 1 priming exposure at baseline, subjects exposed to the test diet-fed cat blankets showed significant reduction in the severity of several nasal and ocular allergy symptoms.

Conclusion

An innovative approach was introduced, that incorporates anti-Fel d 1 IgY antibodies into the cat's food, safely neutralizing Fel d 1 at its source without altering Fel d 1 production by the cat. This provides an additional tool for allergy sufferers that easily inserts into their daily routine of feeding their cat, and does not interfere with the continuation of other methods to reduce allergen load and mitigate allergic symptoms.

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NEW DISCUSSION REGARDING GASTRIC DILATATION-VOLVULUS

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ABSTRACT BODY:

New discussion regarding gastric dilatation-volvulus syndrome (GDV) centers around methods for emergency stabilization, delayed versus immediate surgical intervention, perioperative and postoperative management, and methods and complications of elective gastropexy.

Dogs with gastric dilatation-volvulus syndrome (GDV) require immediate medical and surgical care to improve the odds of survival. The etiology of GDV is still generally unknown, although numerous conformational, dietary, and environmental risk factors have been investigated. There is a wide spectrum of presentation of dogs with GDV. Astute and educated owners may pick up on subtle clinical signs, such as mild abdominal distension and increased salivation. However, often the dog is not brought in until more overt signs are present. Diagnosis and emergency treatment of GDV are often performed based off of a high index of suspicion from signalment, presentation, and physical examination findings (i.e. a large or giant deep-chested dog with high anxiety and restlessness, non-productive retching and hypersalivation, signs of cardiovascular compromise, and abdominal distension or tympany).

Circulatory support is paramount. This is commonly initiated before attempts at gastric decompression. Crystalloid fluid therapy (90 ml/kg) is instituted with baseline parameters checked after every quarter-dose. These parameters include heart rate, pulse quality, capillary refill time, packed cell volume, and total protein. Gastric decompression can be achieved through orogastric intubation or gastric trocharization. For orogastric intubation, a large bore gastric tube is selected and a length is measured from the dog's nose to the last rib. If an orogastric tube is unable to be passed, gastric trocharization may be performed. This should be performed with extreme care to avoid inadvertent laceration of the spleen. Point-of-care abdominal ultrasound can be used to minimize this risk.

Recently, two novel techniques (ultrasound-guided, temporary, percutaneous gastropexy and gastrostomy catheter) were successfully evaluated in dogs with GDV for providing sustained gastric decompression.[1]

In anticipation of surgery, a minimum database should be performed. Evaluation of coagulation parameters may be indicated in dogs that are unstable, have platelet counts less than 100,000, or have evidence of petechiae or ecchymosis. Serum lactate concentration has been evaluated as a preoperative predictor of mortality. Increased survival and fewer complications are associated with lactate levels < 4.0 mmol/L and decrease in lactate level greater than 40% following fluid resuscitation.[2]

Abdominal radiographs can be used to definitively diagnose GDV. A right lateral positioning provides the best view to diagnose GDV. If this positioning isn't diagnostic, a ventrodorsal projection and left lateral projections should be performed as gastric malpositioning other than the typical 180-degree clockwise volvulus can occur. Thoracic radiographs should also be considered, particularly in older patients. A recent study found a negative association between survival and the presence of cardiomegaly seen on preoperative thoracic radiographs.[3]

In addition, a preoperative electrocardiogram (ECG) should be obtained. ECG allows for easy monitoring of response to shock therapy as well as screening for preoperative arrhythmias. Ventricular arrhythmias most commonly occur in dogs with GDV 24 to 48 hours postoperatively. However, the presence of preoperative arrhythmias has been shown to be associated with an increased mortality rate across multiple retrospective studies.

Dogs with GDV are surgical emergencies, although delayed surgery with prolonged medical stabilization has been recently evaluated.[4,5] In the author's opinion delayed surgical intervention should be chosen with caution and reserved for unique circumstances.

In most abdominal surgeries, an exploratory should be performed first as a general assessment of the abdomen and to make sure other possible problems are not missed. This is often not possible in dogs with GDV as the stomach bloated and malpositioned. Derotation is performed by reaching to the left side of the abdominal cavity to find the pylorus. The pylorus is pulled ventrally and over to the right while pushing the body of the stomach dorsally. If this cannot be performed easily, the stomach can be decompressed via orogastric intubation or by direct needle

trocharization.

An exploratory is performed following derotation as this gives the stomach and spleen some time before assessing tissue viability. Subjective parameters are most reliable in assessing organ viability. These include color, thickness, peristalsis, arterial pulses, and capillary (or cut surface) bleeding. Large splenic vessels can be palpated for the presence of a pulse or thrombosis. A necrotic stomach will rapidly lose the characteristic mucosal slip. If indicated a splenectomy or partial gastrectomy should be performed. The decision to perform either of these procedures should not be taken lightly, as both have been showed to be associated with increased mortality.

Advanced hemostatic devices and stapling equipment is extremely beneficial at this point to minimize anesthetic time. Bipolar vessel-sealing devices can be used to assist removal of the spleen, while a large thoracoabdominal (TA) stapler or gastrointestinal anastomotic (GIA) stapler can be used for gastric resection. Up to 70 % of the body and fundus of the stomach can be resected without significant postoperative complications. Significant complications are associated with resection of the cardia and often dogs with necrosis in this area are euthanized. Necrosis of the pylorus and antrum is uncommon. Without stapling equipment, great care should be taken to minimize gross contamination of the abdominal cavity during partial gastrectomy. The stomach should be closed using a two-layer inverting pattern, such as a simple continuous followed by a Lembert, using 3-0 absorbable monofilament suture.

Gastropexy is performed before closure to prevent recurrence. Recurrence rates following gastropexy range from 5 to 10% compared to an 80% recurrence rate without a gastropexy. Belt-loop, incisional, or circumcostal gastropexy should be performed and the choice is often based on surgeon preference. The author prefers to use an incisional or belt-loop gastropexy. An incisional gastropexy should not be confused with a midline, incorporating gastropexy, which the author does not recommend. For an incisional gastropexy, a 3 cm longitudinal incision is made through the seromuscular layer of the pyloric antrum. A matching incision is made in the transversus abdominus muscle on the right side of the abdomen caudal to the last rib. These incisions are then sutured together using 2-0 monofilament absorbable or nonabsorbable suture.

Following surgery for GDV, dogs should be monitored for cardiac arrhythmias, DIC, sepsis, and other systemic complications. Gastric mucosal protectants and motility modifiers are often prescribed. Feeding is typically begun 12 to 24 hours postoperatively with small meals of a highly digestible bland diet. There are multiple causes of death in dogs with gastric dilatation-volvulus syndrome (GDV) including shock, peritonitis, DIC, sepsis, and cardiac dysfunction. Overall mortality for dogs with GDV is approximately 10 to 18%. The animal's state at presentation has also been shown to be a highly predictive risk factor with dogs that present comatose 35 times more likely to die than dogs that walk in. The mortality rate is also increased with the presence of preoperative arrhythmias, the presence of gastric necrosis, or the need to perform a splenectomy or a partial gastrectomy.

Prophylactic/Elective Gastropexy. The effectiveness of prophylactic gastropexy in preventing an acute episode of GDV is unknown. Prophylactic gastropexy is most commonly performed due to a request from an owner who owns a high-risk breed dog (e.g., Great Dane) or has had other dogs suffer or die from GDV. To avoid a large midline incision, prophylactic gastropexy can be performed by laparoscopy or with laparoscopic guidance, endoscopic guidance, or through small grid incision on the right side of the animal. Complications following elective gastropexy have included incisional issues, undocumented delay of gastric emptying, and incidents of gastric dilatation due to inadvertent gastric malpositioning.

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EXAMINANDO EL PULMÓN: PATRONES PULMONARES Y ALGO MAS?

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ABSTRACT BODY:

INTRODUCCION

Las alteraciones del parénquima pulmonar se manifiestan la mayoría de las veces en una radiografía por aumento de la opacidad. Este aumento de opacidad puede deberse a una alteración de las vías áereas, o del intersticio, así según la parte del pulmón afectada se habla de un tipo de patrón pulmonar. Pero para poder realizar los diagnósticos diferenciales de las patologías pulmonares además de los patrones otros factores importantes son la localización y la distribución, además de otros órganos afectados.

En esta charla vamos a ir viendo los hallazgos radiográficos que nos van ayudar a definir los patrones pulmonares, así como la localización y distribución de la lesión para realizar el diagnostico diferencial.

ANATOMIA

Los perros y los gatos tienen dos pulmones, los cuales no son simétricos en cuanto a tamaño y localización. La anatomía lobar se basa en la división de los bronquios.

-El lóbulo izquierdo esta formado por dos lóbulos:

- Lóbulo craneal izquierdo, dividido en un segmento craneal y uno caudal.
- Lóbulo caudal izquierdo

-El lóbulo derecho esta formado por cuatro lóbulos:

- Lóbulo craneal derecho
- Lóbulo medio derecho
- Lóbulo caudal derecho
- Lóbulo accesorio

ANATOMIA RADIOGRAFICA

Los pulmones están compuestos principalmente por aire. El resto de estructuras son los vasos pulmonares, bronquios, bronquiolos, alveolos, tejido intersticial, nódulos linfáticos y la pleura. Los vasos pulmonares principales y los bronquios suelen verse en la zona central cerca del hilio. En la zona media solo suelen verse los vasos. El resto de estructuras proporcionan una opacidad de fondo general.

Los vasos pulmonares y los bronquios que se pueden observar tienen que ser paralelos y han de ramificarse y terminar en punta hacia la periferia.

La opacidad aire del pulmón puede verse alterada por varios factores que no estén relacionados con procesos patológicos, como son:

- Factores de exposición incorrectos
- Fase respiratoria
- Edad del animal
- Decúbito prolongado

Vascularización pulmonar

Los vasos pulmonares deben de verse con claridad en la zona media y central del campo pulmonar, disminuyendo de tamaño hacia la periferia.

En la radiografía lateral, la arteria pulmonar es dorsal al bronquio y la vena ventral a éste. La arteria y vena lobulares craneales deben ser aproximadamente del mismo tamaño y su diámetro a nivel del cuarto espacio intercostal no debe de exceder el diámetro menor de la porción proximal de la cuarta costilla. En la proyección DV los vasos se visualizan mejor que en la VD, pueden identificarse las arterias y venas lobulares caudales derecha e izquierda. Las arterias discurren laterales a las venas y deben ser del mismo tamaño, no deben exceder el diámetro de la novena costilla en el punto que la cruza.

EVALUACION DE LA RADIOGRAFIA

Los diagnósticos radiológicos de las alteraciones pulmonares se basan en:

1.- Modelo pulmonar

2.- Localización y distribución de la lesión

3.- Tamaño del pulmón

3.- Aspecto de otras estructuras torácicas.

Los **Modelos pulmonares,** es un modo de describir el incremento de opacidad del parénquima pulmonar. Los pueden clasificarse en:

- Modelo Intersticial
- Modelo Alveolar
- Modelo Bronquial
- Modelo Vascular

PATRÓN INTERSTICIAL

- **INTERSTICIAL NO ESTRUCTURADO O DIFUSO**

Aumento de opacidad general del parénquima pulmonar.

No se visualizan con claridad los vasos pulmonares.

Diagnósticos diferenciales

- Artefactos (radiografía blanda, radiografía realizada al final de la espiración, animales obesos...)
- Pulmón de animales viejos
- Tumores (Linfosarcoma, metástasis pulmonares difusas...)
- Pneumonías (vírica, micótica, parasitaria, metabólica, alérgica, tóxica...)
- Procesos en transición (broncopneumonía, edema...)

2. INTERSTICIAL ESTRUCTURADO/NODULAR

Se aprecian estructuras nodulares solitarias o múltiples de diferentes tamaños, Pueden ser cavitarias (opacidad tejido blando y gas) o no cavitarias (opacidad tejido blando).

Diagnósticos diferenciales NO CAVITARIOS

- Tumores (primarios, metástasis pulmonares)
- Granulomas (micóticos, parasitarios, de cuerpo extraño...)
- Abscesos
- Hematomas
- Quistes

Diagnósticos diferenciales CAVITARIOS

Capsula fina:

- Bullas pulmonares
- Neumatocele. Esto son cavidades causadas por neumonía crónica
- Quistes congenitales pulmonares y bronquiales
- Quistes parasitarios: hidatidosis

Capsula gruesa y generalmente irregular:

- Tumores pulmonares primarios o metastáticos. La cavitación es debida a necrosis del centro del tumor. Se observa principalmente en carcinomas pulmonares.
- Abscesos
- Quistes parasitarios: Paragonimiosis

PATRÓN ALVEOLAR

Refleja una alteración a nivel de los ALVÉOLOS. Se observa un aumento de opacidad del parénquima pulmonar y no se visualizan los vasos pulmonares.

Hay una sustitución total o parcial del aire alveolar por fluidos, detritus celulares, infiltrados neoplásicos, o bien, colapso alveolar.

Signos radiográficos

Se observa la presencia de "BRONCOGRAMAS" y/o "ALVEOLOGRAMAS" AÉREOS. Si afecta a un lóbulo pulmonar y el lóbulo de al lado esta normal observaremos el, SIGNO LOBAR (todo el lóbulo esta radiopaco), y también el SIGNO SILUETA.

Diagnósticos diferenciales

Generalizado:

- Pneumonía grave
- Edema pulmonar cardiogénico
- Edema pulmonar de origen alérgico, tóxico, urémico ...
- Ahogamiento

Focal o multifocal:

- Pneumonía (bacteriana, por aspiración)
- Edema pulmonar
- Hemorragia pulmonar
- Torsión lobar (lóbulo medio derecho y parte craneal del craneal izquierdo)
- Colapso pulmonar o Atelectasia
- Pneumonía parasitaria
- Neoplasias (excepcionalmente)

PATRÓN BRONQUIAL

Refleja alteraciones a nivel de los BRONQUIOS.

Signos radiográficos

Se observan las paredes bronquiales engrosadas como RAILES DE TREN, ANILLO, "DONUTS".

Diagnósticos diferenciales:

- Bronquitis (vírica, alérgica, bacteriana...)
- Engrosamiento de la pared bronquial
- Infiltración peribronquial
- Bronquiectasias (bronquios dilatados, no disminuyendo de tamaño hacia la periferia)
- Calcificación de la pared bronquial

PATRÓN VASCULAR

Refleja alteraciones a nivel de los vasos pulmonares, relacionado con problemas cardiacos.

Signos radiográficos

- Modificación del diámetro vascular
- Hiperperfusión pulmonar
- Hipoperfusión pulmonar
- Tortuosidad vascular

Patrón hipervascular

Aumento de venas y arterias

- Congestión pulmonar pasiva – fallo cardiaco izquierdo
- Shunts de izquierda a derecha
  - Conducto arterioso persistente
  - Defectos del septo interventricular y del septo interatrial
- Sobrehidratación yatrogénica

Venas más grandes que arterias

- Fallo cardiaco izquierdo

Arterias más grandes que venas

- Dirofilariosis
- Tromboembolismo pulmonar
- Hipertensión pulmonar
- Angiostrongilosis
- Aelurostrongilosis
- Fistula arteriovenosa periférica

Patrón hipovascular

Disminución generalizada de la vascularización pulmonar

- Hipoperfusión pulmonar (choque, deshidratación, hemorragia, Addison)
- Enfermedad pericárdica
  - Efusión con taponamiento
  - Pericarditis restrictiva
- Fallo cardíaco derecho
- Enfermedades cardiacas congénitas con shunts de derecha a izquierda
- Estenosis pulmonar grave

Disminución localizada de la vascularización pulmonar

- Tromboembolismo pulmonar
- Enfisema lobar comprimiendo los vasos

La LOCALIZACIÓN de la lesión es uno de los signos radiográficos más importantes para realizar el diagnostico diferencial. Las lesiones se categorizan en cinco distribuciones en el campo pulmonar:

- Craneo-ventral (precardial)
- Caudo-dorsal (postcardial)
- Ventral –medio (se superpone con la silueta cardiaca)
- Perihiliar (central)
- Periférico

La **DISTRIBUCIÓN** puede ser:

- Difusa
- Focal
- Multifocal.

Los DIAGNÓSTICOS DIFERENCIALES basados en la **DISTRIBUCIÓN**

Los DIAGNÓSTICOS DIFERENCIALES basados en la **LOCALIZACIÓN Y PATRÓN PULMONAR**

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ENFERMEDADES ÓSEAS DEL CRECIMIENTO

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ABSTRACT BODY:

ENFERMEDADES ÓSEAS DEL CRECIMIENTO

Hay una serie de patologías óseas que aparecen durante la fase de desarrollo del animal. Algunas siguen siendo de etiología desconocida, otras relacionadas con desórdenes alimenticios , otras van ligadas a la raza del animal ,otras relacionadas con parasitemias, y por último las que podríamos clasificar como raras o poco frecuentes

Etiologia desconocida

Panosteitis. También se la denomina enostosis y se relaciona con una disfunción de la osificación endóstica unida a procesos inflamatorios. Relativamente común en razas grandes y gigantes con cojera intermitente afectando diferentes extremidades, más frecuentemente las anteriores, de forma alterna sin relación con trauma previo que afecta a animales jóvenes de 5 a 12 meses de edad con especial incidencia en P. Alemanes y Basset-Hound. Los animales presentan dolor a la palpación y fiebre. El diagnóstico es radiológico evidenciando una densidad heterogénea en forma de “nube” a nivel de los huesos largos. Los más afectados son el cúbito-radio, húmero y fémur. Es auto limitante y suele desaparecer al completar el crecimiento

Osteodistrofia Hipertrofica. También llamada síndrome de Barlow. Antiguamente se relacionaba con un déficit de Vitamina C (escorbuto) aunque su origen sigue desconocido. Suele producir dolor, pirexia, anorexia y rechazo al movimiento. Radiológicamente se observa una línea radiolúcida paralela a la fisis a nivel sobretodo del radio. En estadios más avanzados se produce un crecimiento radiopaco, subperióstico a nivel de metáfisis. Es autolimitante

Osteopatía craneomandibular. Conocida como “cara de león” es una lesión osteoproliferativa que afecta las ramas mandibulares, huesos occipitales y temporales provocando dificultades para abrir la boca. En ocasiones afecta las bullas timpánicas. Predisposición racial en los terriers aunque también ha sido descrita en otras razas: Doberman, Labrador,etc… .De etiología desconocida, generalmente es autolimitante.

Hiperflexión- hiperextension carpal. La hiperflexión del carpo se debe a una contractura del músculo carpo-radial. Se supone que se debe a un desarrollo asincrónico entre el esqueleto y el aparato musculo-tendinoso.Se clasifica en 3 grados en función de la desviación en varo del carpo. La hiperextensión del carpo, también de etiología desconocida se relaciona con una falta de magnesio o una excesiva actividad en cachorros así como a un desequilibrio entre masa muscular y hueso. El animal presenta plantigradismo con pseudovalgo.Suelen ser autolimitantes.

Desórdenes alimenticios

Los cambios alimenticios actuales de los animales de compañía (sustituyendo dietas caseras por dietas comerciales) han hecho que patologías óseas que hace unos años eran relativamente frecuentes, se vean raramente en la actualidad. Entre ellas, el raquitismo, la osteoporosis y la hipervitaminosis A.

Raquitismo. Consiste en la falta de mineralización de la matriz condroide debido a un déficit de aporte o asimilación de calcio y/o fósforo y vitamina D. Clínicamente se presentan como deformaciones simétricas de huesos largos a nivel yuxta-articular o en la zona condrocostal (rosario raquítico) que son indoloras. Radiológicamente se detecta osteopenia, con un incremento de la anchura de los cartílagos de crecimiento, y la zona metafisaria adopta una forma de hongo.

Osteoporosis como consecuencia a un hiperparatiroidismo nutricional secundario ocurre cuando hay un desequilibrio de calcio/fósforo en la dieta (por ejemplo, animales con excesiva ingesta de carne, rica en fósforo y pobre en calcio). Radiológicamente se observa una osteopenia generalizada y corticales débiles con posibilidades de fracturas por plegamiento.

Hipervitaminosis A , típica de gatos alimentados excesivamente con productos ricos en vitamina A, (bazo, hígado) que produce una saturación de la misma a nivel de la grasa corporal, y secundariamente una hiperplasia del tejido sub-perióstico que invade vértebras, codo y rodillas, produciendo una anquilosis.

Relacionadas con la la raza del animal

Osteocondrodisplasia de Scottish, especifica de esta raza de gatos,consiste en un trastorno hereditario que produce un desarrollo defectuoso del cartílago, con afección de las zonas mas distales de las articulaciones: carpo, tarso y la formación de exóstosis óseas. El pliegue del cartílago de la oreja va ligada a esta patología.

Relacionadas con parasitemias

Lesiones óseas producidas por leishmaniosis. En zonas endémicas de leishmaniosis se puede afectar cualquier animal independientemente de la edad y se debe incluir siempre como diagnóstico diferencial en casos de sinovitis, artritis y lesiones óseas en general. El patrón óseo más frecuente es el articular.

Enfermedades óseas raras o poco frecuentes

En Medicina Humana se considera una enfermedad rara cuando su incidencia es de un caso por cada 2000 individuos. En pequeños animales este concepto no está establecido pero sí que existen una serie de patologías óseas que se pueden clasificar como poco frecuentes por su escasa incidencia en la clínica diaria. Vamos a destacar: la exóstosis cartilaginosa múltiple, la epifisiolisis del calcáneo, la epifisiolisis femoral y el síndrome de Osgood-Schlatter .

Exostosis cartilaginosa múltiple (ECM) consiste en la aparición de protuberancias óseas que pueden aparecer durante el periodo de crecimiento. Los huesos afectados son los formados mediante osificación endocondral siendo las vértebras, costillas y huesos largos los más propensos. A pesar de ser histológicamente iguales se denomina ECM, cuando hay múltiples lesiones y osteocondroma si la lesión es única. La etiología se desconoce pero se considera una mutación genética como la causa más probable. Los signos clínicos dependerán del número, tamaño y localización de las exostosis ya que pueden provocar compresión medular o daño mecánico.

Epifisiolisis del calcáneo. Las epifisiolisis consisten en la separación de la fisis de su unión con la diáfisis sin una causa traumática a diferencia de las fracturas Salter-Harris en las que si hay implicado un traumatismo. Hay pocos casos descritos de epifisiolisis a nivel del calcáneo en Medicina veterinaria. En Medicina humana se denomina enfermedad de Sever. La etiología no se conoce con exactitud y en el perro se considera una patología con pronóstico grave.

Epifisiolisis femoral consiste en la separación no traumática de las epífisis femorales. En un 30% de los casos se presenta de forma bilateral. La etiología es desconocida pero se mencionan factores mecánicos y bioquímicos. La fisis femoral permanece abierta hasta aproximadamente los 9 meses de edad. Un exceso de estrés en torsión y flexión puede dañar la placa de crecimiento por lo que es importante evitar el crecimiento rápido, el sobrepeso y el ejercicio excesivo. La clínica, la radiografía y el estudio histopatológico nos llevan al diagnóstico definitivo. La artroplastia o la prótesis total de cadera son el tratamiento de elección.

Síndrome de Osgood-Schlatter, es una apofisitis secundaria a la tracción del ligamento rotuliano produciendo una separación de la tuberosidad tibial, evidenciada mediante radiografía. En casos leves el reposo es curativo mientras que en los casos más graves se debe recurrir a la fijación mediante agujas. Es relativamente frecuente en Medicina humana en deportistas adolescentes.

PLEURAL EFFUSION AND PERICARDIAL EFFUSION. WHY THEY ARE THERE AND TIPS FOR MANAGING THEM

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ABSTRACT BODY:

Introduction Thoracic effusion (including pleural and pericardial effusions) are a common reason for dogs and cats to present to the veterinary practitioner. The goal of this session is to get the practitioner more comfortable with managing thoracic effusion as well as some helpful diagnostic tools. A small amount of fluid is present in the body cavity of dogs and cats.[1] This fluid provides lubrication that allows adjacent organ surfaces and body cavity walls to move without friction. Effusion is the abnormal or increased accumulation of fluid in any body cavity lined by mesothelial cells. This session will focus on thoracic effusion that includes pleural and pericardial effusions. Effusion is not a disease in itself, it results from alterations in fluid production, lymphatic drainage or a combination of both.[2] Some factors that affect production and absorption of fluid include changes in capillary hydrostatic pressure (heart failure), plasma osmotic pressure (hypoalbuminemia) and capillary permeability (vasculitis, infectious/inflammatory diseases)

Clinical History and Presentation Dogs and cats with thoracic effusion usually present with clinical signs of respiratory distress. Other historical and physical exam findings that might be noted includes orthopnea, open-mouth breathing, tachypnea, increased abdominal effort. Cyanosis may also be present. Some animals (cats especially) may have non-specific signs of anorexia and respiratory signs may not be noted by the owner until their signs are severe. The most common historical signs associated with pericardial effusion is weakness and lethargy, however some owners may have vomiting or sudden collapse at home.[3] The veterinary team may notice muffled heart and lung sounds on the physical exam. In animals with pericardial effusion, signs of shock (including collapse, altered mentation, weak peripheral pulses, pale mucous membranes, tachycardia) may be presented.**Diagnosis** It is helpful that the diagnosis of thoracic effusion can be performed quickly in a veterinary hospital. This is important because animals with thoracic effusions may decompensate (or die) if immediate diagnosis is not performed OR therapeutic intervention is not carried out quickly enough. The easiest and most helpful tool for diagnosing thoracic effusion is the ultrasound. [4] The thoracic FAST scan (TFAST) is useful for the rapid detection of pleural effusion, pericardial effusion and pneumothorax in dogs and cats. The advantage of using the ultrasound is that it is readily available but probably more importantly, it is SAFER to use as a diagnostic tool than radiographs. Due to the stress of obtaining radiographs (restraint as well as positioning challenges), dogs and cats may actually die during the process of obtaining radiographs. In some cases- due to the volume of fluid present- radiographs may also not reveal the cause of the animal's problems (really acute pericardial effusion may not cause cardiomegaly, pleural effusion may hide neoplasm in the chest). The use of TFAST has extended beyond trauma and should be utilized in any patient who presents with signs of collapse (thorax and abdomen) and/or respiratory distress.

Radiographs are also a very helpful tool in animals with thoracic effusions but as much as possible, radiographs should be obtained after a TFAST and after the animal has been stabilized.Other diagnostic tools that may be helpful include lab work (CBC, Chemistry, coagulation times), abdominal ultrasound (to detect neoplasia), cytological evaluation of the pleural and pericardial effusion and an echocardiogram.

Stabilization

Many animals with thoracic effusion present unstable due to poor cardiac output and/or respiratory distress. Hence it is extremely important that the veterinary practitioner considers initial stabilization before discharge or referral. In general, pleural and pericardial effusions HAVE to be removed to make the animal more comfortable. There are no pharmaceutical agents that minimize pleural effusion or pericardial effusion. The only exception is the resolution of hemorrhage after trauma or rodenticide ingestion.

Thoracocentesis and pericardiocentesis are relatively safe procedures, even for the practitioner that has never attempted one before. They are also truly life-saving procedures. Details of the procedures are described in the lecture but the main highlights is to utilize the ultrasound to identify where you would go. The author has also done both procedures blind when ultrasound was not commonly available in veterinary medicine. It is estimated that it takes 20-60 mL/kg of fluid in the pleural space to cause respiratory distress in cats and dogs. This can be used as a guide to determine if enough effusion has been removed from the pleural space, in conjunction with rechecking

remaining fluid volumes with the ultrasound. Pericardial effusion is almost always hemorrhagic. A sample should be saved in a preservative-free tube and observed for about two minutes to ensure that there is no clotting. This provides the confidence that the needle is not in a cardiac chamber. In some cases, the pericardiocentesis may not produce a large volume of fluid because the fluid leaked into the pleural space. This pleural effusion generally does not need to be removed.

Other considerations for animals with thoracic effusion include hospitalization (pleural effusion rarely returns quickly but pericardial effusion may) and diagnostics to try to determine the cause of the effusion.

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MANAGING PAIN WITHOUT DRUGS

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ABSTRACT BODY:

TREATING PAIN WITHOUT DRUGS

When it comes to treating pain in any species, it is an emerging realization that a combination of pharmaceutical and non-pharmaceutical tools must be utilized. For example, each of us bipeds who has ever been injured will realize the things like ice, massage and physical therapy are commonly applied to aide in our return to function. Physical medicine modalities that have been used in cats include acupuncture, stretching, therapeutic exercise, massage, joint mobilization, ultrasound, laser, shock wave therapy, ice, heat and environmental enrichment.

Physical medicine at it most simple, is interacting with the analgesic systems provided by an organism and attempting to activate these systems. This occurs along the entire pain sensing system: from skin to brain, and also involves muscle, tendon, fascia, periosteum, etc. Physiology provides a complex, multi-faceted tapestry for both pain sensing and modification of the transmitted signal. The overlap between the nervous system and other endogenous, autonomic systems (such as immune state, GI motility, etc.) that has long been recognized in osteopathic medicine is now becoming main-stream and evidence base is ballooning.

Understanding the physiology of nervous system wiring is the first step toward understanding methods to intervene in these endogenous systems and help modify them towards homeostasis. The relationship of many of these modalities with vagal nerve stimulation and interactions opens up a growing field of evidence to help describe the mechanisms for these interventions. While pain is a major target, several other conditions applicable to veterinary practitioners are also gaining an evidential basis, including neurological injury and return to function, GI motility modification, and immune and homeostatic modulation. In addition to modalities to modify the parasympathetic nervous system, physical medicine techniques exploit endogenous physiology in numerous ways:

- Temperature related modalities
- Tissue mobilization and mechanotransduction
- Peripheral neuromodulation
- Spinal neuromodulation
- Central neuromodulation
- Kinematic modulation
- Modulation of metabolism and blood flow
- Muscle spasm and myofascial trigger points
- Immune modulation

A number of non-pharmacological treatments are available in veterinary medicine. Those discussed today include:

Acupuncture is understood to work through neuro-modulation along the majority of innate pain-modifying and immune modifying pathways. In trained hands, acupuncture has virtually no negative side-effects, and can be a potent tool for promoting analgesia, gastro-intestinal motility and immune modulation. We will go over the basic technique of acupuncture needling with a focus on the points that might be used in trauma cases, and the neurophysiologic rationale for use of these points.

Laser therapy is a safe and non-invasive tool for increasing local blood-flow, providing local and regional analgesia, and increasing the rate of tissue healing.

Tactile input such as bandaging, touch and massage have been shown to provide comfort and analgesia across species, while improving local blood flow and relieving secondary sources of pain from medical conditions, recumbency, and procedures.

Thermal support options will be discussed, with an emphasis on the physiological effects of each, the optimal timing and methods to provide.

-Heat improves local tissue blood flow and helps to release soft tissue restrictions.

-Cooling decreases pain sensation, edema formation, and peripheral hypersensitivity

Motion such as walking, supported standing, range of motion, etc. have been shown to augment analgesia; improve fluid dynamics in soft tissues such as intervertebral disks, joints, tendons, ligaments, muscles and fascia; reduce soft tissue contracture and speed recoveries. Exercise is recognized as hypo-algesic, within strict confines. Exercise has also been shown to improve functional recovery from various injuries that can be seen with trauma (soft tissue, bone, neurologic).

Clearly, correct application of motion is the hinge between safe and unsafe use. At the same time, you may practice these tools without needing full-fledged certification (just like the limited acupuncture techniques discussed above). Most of these modalities require a good understanding of anatomy and physiology, sensitive hands to find problems, and a careful, attentive mind, to anticipate and prevent problems, and to detect when a particular plan is not being received as expected by the patient. Examples include:

-Assisted standing on a stable surface- with supportive equipment

-Assisted standing on an unstable surface

-Prioritization of functionally appropriate end-points (what you practice is what you become)

-Rhythmic stabilization for engagement of spinal stabilizers and provision of analgesia

-Safe support equipment (harnesses, peanuts, etc.).

-Utilization of routine and functional tasks – pottying and eating...

Nursing and supportive care to provide regular rotation during recumbency, warmth, touch, interaction and comfort are also critical to recovery and should be considered a chartable form of therapy, similar to any medication.

Other interventions:

ECSWT: non-healing wounds, tendon and ligament injuries

HBOT: inflammatory conditions (pancreatitis), extensive soft tissue damage (burns, wounds), smoke inhalation, nerve injury (brain, spinal cord, peripheral nerves)

Biologicals: plasma (eyes); platelet rich plasma (tendons, ligaments, skin); stem cells (enough data?)

GI support: Deserves a category, as it is as ubiquitous of a problem as pain in the ICU. This too, requires a synthesis of drug and non-drug modalities.

Drug modalities include: metoclopramide, cisapride, ranitidine, anti-emetics (but caution with reducing motility with these- maropitant for example) appetite stimulants

Non-drug modalities include:

Feeding,

Routine activity (exercise)

Stimulation of neurophysiologically rich regions for GI motility, appetite (with acupuncture, laser, massage)

Therapeutic abdominal massage

Reducing stress/ catecholamines (increasing vagal tone)

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MANAGING PAIN WITHOUT DRUGS

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INSPIRATORY DYSPNEA: INTERACTIVE CASE DISCUSSION

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ABSTRACT BODY:

Clinical Approach: Basic concepts

Dyspnea is the sensation of having difficult or laborious breathing. It is a subjective phenomenon that needs to be inferred by the clinician in companion animals. Dyspnea, tachypnea (rapid breathing), and hyperpnea (increased ventilation) are not synonymous, but are frequently grouped together in veterinary patients. Dyspnea occurs when the demand for ventilation is out of proportion to the animal's ability to respond to this demand. Thus, breathing becomes difficult, uncomfortable, or labored. Dyspnea may result from alterations in any portion of the respiratory system or be due to abnormal mechanics of the lung and chest wall.

Look: When does the Dyspnea occur?

Inspiration x Expiration x Both

The timing and pattern of respiration helps to determine the structure most likely responsible for the dyspnea. Dyspnea may occur during inspiration, expiration or both (mixed). Clinically, pure inspiratory dyspnea implies a lesion in the respiratory tract outside the thorax, whereas expiratory and mixed dyspnea occur in patients with thoracic or metabolic disease.

Look: What is the pattern of respiration?

Obstructive x Restrictive

Mixed or expiratory dyspnea should be further classified as obstructive or restrictive based on the ventilatory pattern. *Obstructive diseases* are associated with increased airway resistance in the tracheobronchial tree. Obstructive diseases may also occur in the upper respiratory tract, but those patients have inspiratory dyspnea. Patients with obstructive disease have decreased expiratory flows and hyperinflated lungs (e.g., cats with asthma). *Restrictive diseases* are those in which expansion of the lungs is restricted (e.g., pulmonary fibrosis, pleural effusion). Lungs of patients with restrictive disease operate at smaller volumes and the patient has a rapid shallow breath. In patients with a non-pulmonary cause of restrictive, residual volume is normal or increased.

Listen: Can you hear Respiratory Sounds?

Normal sounds? Abnormal sounds

The physical examination assists in identifying the cause for the dyspnea. In patients with restrictive pulmonary disease, absence of respiratory sounds indicates a pleural cavity disease, whereas presence of pulmonary sounds occurs in patients with parenchymal pulmonary disease, metabolic diseases, or abdominal distention

Inspiratory Dyspnea

Inspiratory dyspnea occurs with extrathoracic lesions in the respiratory tract (table 1). Patients with isolated pure nasal problems can breathe normally when the mouth is open. Presence of abnormal respiratory sounds may help to localize the problem. *Stridor* is a loud musical inspiratory sound of constant pitch associated with laryngeal (and occasionally tracheal) alterations. *Rhoncus* (stertorous breathing) is a rattling in the throat associated with pharyngeal or proximal tracheal diseases. *Cough* may occur in patients with inspiratory dyspnea. Cough receptors are in the larynx, pharynx, and large airways and disease in any of those locations may be associated with cough. In patients with extrathoracic disease, cough is usually paroxystic and loud. Direct visual inspection or bronchoscopy is necessary determine its cause.

Table 1. Causes of Inspiratory Dyspnea

Nostrils Nasal Cavity
Stenotic nares Foreign bodies
Nasal Mass
Rhinitis
Redundant Turbinates
Larynx/Pharynx Cervical Trachea
Elongated soft palate Tracheal collapse
Caudal palate Tracheitis
Laryngeal paralysis Parasites
Laryngeal edema Extraluminal compression
Everted laryngeal saccules Foreign body
Laryngeal laceration/trauma
Laryngeal neoplasia
Hyoideal fracture
Retropharyngeal polyps

Clinical Cases

Cases will be presented during the lecture to apply the concept described above.

References available upon request

NUTRICIÓN DEL GATO GERONTE, CUÁNTA PROTEÍNA DEBE RECIBIR?

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ABSTRACT BODY:

INTRODUCCIÓN

En el año 2021, AAHA (American Animal Hospital Association) en conjunto con AAFP (American Association of Feline Practitioners) desarrollaron las Directrices para estadificar las etapas de vida en los felinos. En estas clasificaron al gato como Cachorro (Kitten en inglés) desde el nacimiento hasta el primer año de edad; adulto joven desde 1 a 6 años de edad; adulto maduro desde 7 hasta 10 años de edad y "Sénior" al gato mayor de 10 años. Las empresas de fabricación de alimento balanceado para mascotas, generalmente recomiendan, que los gatos desde los 7 años cambien a una dieta llamada "Sénior". Erróneamente se considera que a partir de esta edad debemos reducir la proteína de la dieta en los gatos.

PROCESO DE ENVEJECIMIENTO

Durante el paso de los años, los órganos envejecen, se deterioran, y son menos eficientes. La velocidad y grado de envejecimiento depende de varios factores: genética, medio ambiente, nutrición (calidad y tipo de alimentación) y la posible aparición de patologías y/o eventos adversos (traumatismo, intoxicaciones, etc.). Se sabe que la reducción del stress ambiental influye favorablemente en la calidad de vida y por lo tanto el tiempo de vida del individuo.

Existen ciertos cambios asociados con el envejecimiento en la especie felina como son disminución de la hidratación, lo que puede favorecer a la aparición de ciertas patologías como Enfermedad del tracto urinario inferior, estreñimiento, enfermedad renal crónica, entre otras. Debido a esto es de vital importancia asegurar la hidratación del gato sénior, que desde el punto de vista alimenticio se puede lograr con uso de alimento húmedo (enlatado). Se debe buscar alimentos altamente palatables pues pierden el sentido del gusto disminuye.

Otros cambios que aparecen en los gatos sénior son: mayor índice de problemas orales, disminución de la respuesta del sistema inmune, disminución de la tolerancia a la glucosa, disminución de la función renal, variaciones en peso corporal y disminución de la digestibilidad.

DIGESTIBILIDAD Y CAMBIOS EN EL PESO CORPORAL EN EL GATO GERONTE

La digestibilidad de las proteínas y grasas disminuye en los gatos sénior. Existe una relación inversa entre la edad y la absorción de estos nutrientes.

Se sabe que la digestibilidad de las proteínas en un gato adulto sano es de alrededor del 85 a 90%. Aproximadamente un 20% de gatos geriátricos tiene menor capacidad de absorber proteínas, pudiendo bajar a menos de 77% (Camargo, 2004). Esta disminución va de la mano con pérdida de masa muscular y contribuye a que los gatos tengan un balance negativo de nitrógeno. Es por esto que muchos gatos sénior sufren de sarcopenia (Sparkes, 2011).

La digestibilidad de la grasa en un gato adulto sano es de alrededor del 90 a 95% y en un gato sénior puede bajar a menos de 80%. Esto puede ocurrir hasta en un tercio de los gatos geriátricos La capacidad de absorberla disminuye conforme avanza la edad, en algunos gatos mayores de 12 años puede ser tan baja como 30% (Camargo, 2004). Debido a que las grasas son el principal macro nutriente que provee energía, su menor absorción contribuye a la disminución de peso y pérdida de la condición corporal, además que afecta negativamente al balance energético.

Los gatos entre los 7 y 12 años tienen mayor riesgo de presentar obesidad. Sin embargo, debido a la disminución en la digestibilidad, los gatos mayores de 12 años empiezan a perder peso, por lo tanto, condición corporal. Los índices bajos de condición corporal están relacionados a mayores tasas de mortalidad (Teng, et al. 2012). Las principales causas por las que un gato viejo disminuye de peso son disminución de proteína en algunas dietas senior, dietas de prescripción (por enfermedad) y disminución de actividad por enfermedad degenerativa articular lo que contribuye a la sarcopenia (Zoran, 2018).

¿CUÁNTA PROTEÍNA DEBE RECIBIR UN GATO VIEJO?

A diferencia de los perros y humanos, los gatos viejos experimentan un aumento en sus requerimientos energéticos. La pérdida progresiva de masa muscular, debe ser mitigada alimentándolos con altos niveles de proteína. Esta debe ser altamente digestible y energética. Si la cantidad de proteína en la dieta no es mayor a 40% de energía metabolizable (EM), los gatos utilizarán su propio músculo para mantener los requerimientos proteicos (balance de nitrógeno). La

mayoría de dietas secas felinas, tienen entre 30 a 38 % de proteína EM. Esto puede significar que a pesar de que el gato coma una dieta que supuestamente es completa y balanceada, pierda masa muscular. Esta pérdida será mayor si el gato ingiere dietas económicas con proteína poco digestible o en bajos porcentajes (Zoran, 2018).

En resumen, un gato geriátrico requiere una dieta con mínimo de 12 g/100 kcal o mayor a 40% de proteína EM para preservar la masa muscular y proveerlo de una nutrición óptima. Incluso algunos gatos sénior, pueden requerir mayores cantidades de proteína altamente digestible, pudiendo llegar hasta 15 g/100 kcal o alrededor de 50% de proteína EM.

Además de los altos niveles proteicos, los gatos geriátricos necesitan mayor energía proveniente de la grasa (mínimo 20%), aumento de ácidos grasos omega 3, vitamina E, betacaroteno y vitaminas del complejo B, especialmente cobalamina (Cupp, et al. 2009). Los alimentos balanceados que se comercializan para gatos mayores o sénior, en la actualidad no contienen menor cantidad de proteína y grasa como anteriormente se pensaba. Las diferencias entre estas dietas y las de adultos, están en la mayor cantidad de fibra cruda y adición de suplementos. Su uso, de debe ser evaluado según cada caso en particular.

En los gatos con Enfermedad Renal crónica (ERC), aún existen discrepancias si realmente existe una necesidad de restringir los niveles de proteína. Las dietas de prescripción para enfermedad renal tienen en promedio 23 a 27% de proteína. Esta reducción hará que reduzca el Nitrógeno ureico en sangre y la creatinina. Sin embargo, el gato puede empezar a perder mucha masa muscular, tener un balance de nitrógeno negativo y se podría tener una falsa sensación de mejoría. Algunos autores recomiendan iniciar la restricción proteica únicamente en pacientes con estadíos IRIS 3 ó 4. En todos los estadíos es de vital importancia asegurar la hidratación (Polzin et al. 2016).

En gatos con Hipertiroidismo se debe mantener un alto nivel proteico debido a la alta tasa metabólica y balance negativo del nitrógeno, que produce la enfermedad. Idealmente una dieta para gatos con hipertiroidismo debería contener un porcentaje alto porcentaje de proteína y bajo de carbohidratos. Las mismas características corresponden a dietas que se pueden usar en gatos con Diabetes Mellitus.

CONCLUSIONES

Los gatos de edad avanzada tienen menor capacidad de utilización de nutrientes, específicamente grasas y proteínas, debido a la disminución en su digestibilidad. No se debe reducir el porcentaje proteico en la dieta, idealmente mantenerlo por arriba de 40% EM para evitar pérdida de masa muscular. Se debe evaluar cada paciente independientemente para poder hacer una recomendación apropiada del tipo de comida que debe ingerir, contemplando posibles patologías existentes.

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HOW TO FACILITATE COMPLIANCE WITH DIET ELIMINATION TRIALS

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ABSTRACT BODY:

HOW TO FACILITATE COMPLIANCE IN DIET ELIMINATION TRIALS

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This lecture will focus on implementing successful diagnostic diet trials on individuals that would benefit from them: advances in dietary options and updated information on adverse food reactions.

Cutaneous Adverse Food Reaction (CAFR) has been described as an intolerance or allergy to one or more ingredients in a diet, foods, or additives; resulting in a range of clinical manifestations: dermatological presentation such as pruritus, otitis, erythema, papules, secondary infections, secondary lesions due to trauma. Other rare dermatological presentations can occur. Non-dermatological symptoms include gastrointestinal signs, such as diarrhea, vomiting, borborygmi, flatulence, increased bowel movements in a day, and other more uncommon non-dermatological respiratory manifestations. The current consensus is that the diagnosis of CAFR is made by exclusively feeding a diagnostic diet for eight weeks, followed by a provocative challenge.

Key points to focus on:

- Diagnostic process-
- As in any veterinary, consult **a detailed clinical history** should be. Performing a thorough review of the patient's medical history, the evolution of the disease, and all previous treatments and outcomes are essential. Assessing the clinical history asking the right question correctly, and identifying relevant information is an art and requires skill and practice.
- Nutrition and gastrointestinal questions must be a part of this process. Having a routine or a questionnaire helps improve the quality of the clinical history avoiding missing questions or being led by the owner away from important issues. Feeding and nutrition can be very personal for our clients and knowing how to ask and approach the issue can. Always asking open questions being positive and non-judgmental, and being a good listener are excellent skills.
- Understanding that clinical manifestations do not help differentiate between environmental and food allergies, Adverse food reactions can be challenging to diagnose correctly because a food trial is required to confirm or rule out food involvement
- Using serological, intradermal, prick, saliva, and patch testing to diagnose food allergens is not recommended as they have not been proven reliable or practical diagnostic tools. The combination of prick and patch testing has been recently evaluated for diet selection.
- Correct patient selection:
- The following are important markers gathered during the conversation with the owner or the physical exam that help us move forward with a food trial.
- Dogs with the usual Canine atopic dermatitis dermatological symptoms that are non-seasonal have their typical clinical signs aggravated or caused by the ingestion of dietary ingredients.
- Dogs started with clinical symptoms at a year old or younger with one or more of the following symptoms: perennial Itching, acute or chronic, CAD symptoms, recurrent pyoderma pruritic or non-pruritic, recurrent “hot spots.” conjunctivitis, asthma, anaphylaxis, or sneezing associated with eating. Dorsal lumbar involvement in one of the more severely affected areas may be a clue. Reverse sneezing related to food exposure may be a clue. These dermatologic problems occur along with other gastrointestinal signs such as; more than three bowel movements per day, eating grass, vomiting, belching, and borborygmus. These elements are based on cutoffs for sneezing and gastrointestinal signs based on a study that reported prevalence that was determined in apparently healthy dogs. Older dogs that present CAD symptoms later in life with or without ear involvement and gastrointestinal problems will also be high on the candidate list of patients for a food trial.
- Another group of suitable candidates will be adult dogs with CAD symptoms and any gastrointestinal or respiratory disturbances mentioned above. Dogs that present with recurrent otitis with no other abnormalities and where other causes have been

ruled out should go into a food trial

- Identify limiting factors related to diet implementation
    - Home related factors

Homes with many pets, small children, or many people feeding the animals will make a strict and successful food trial unlikely.

- Owner related factors

An owner that is unable to follow a strict diet follows directions or is unable to understand the need for the diet to be performed a certain way.

Food ideology can make it challenging to implement a successful diet trial.

Patient-related factors

- Finicky eaters, hunters, dogs that are very sensitive to diet changes, GI issues.

It is not useful to start a diagnostic diet trial in situations that are sure to fail and will result in the loss of clients. In these cases, the food trial will not be implemented, and the patient will start multi-modal treatment to control Itching and secondary infections. Other diagnostic tests can be performed, such as environmental allergy testing for immunotherapy.

- Diet selection for a food trial
- Currently, there are a few prescription diets available for use in diagnostic diet trials, and the options are as follows
- A new elemental amino-acid-based diet. The prescription commercial elemental diet for dogs is not made from a parent protein but free amino acids and purified carbohydrates.
- The next group of diets is hydrolyzed diets. These diets have been used for many years in diagnosing and managing CAFR.
- The next option is commercial diets containing novel ingredients. Over-the-counter diets with novel proteins have been demonstrated to contain non-declared ingredients. Because of this, using over-the-counter diets for food trials is not recommended.
- Home prepared diets
- Some veterinary dermatologists still consider home-prepared diets the first choice for food trials. Proper nutrition should be a factor when using home-prepared diets; it is complicated to achieve a balanced and complete combination of ingredients, and a nutritionist must be used. Allergen cross-reactivity when choosing ingredients
- Diet trial duration has also been controversial, but a consensus is that it should last eight weeks.
- Another important consideration is that reaction time from ingesting the offending ingredient can vary greatly. Many dogs present symptoms in the first 24-48 hours, but some can take 14 days to develop a reaction.
- Performing a correct food trial requires that the pet owner understands the process's goal, the restrictions, and the changes involved. Some patients will need more than one food trial, which can be lengthy. The lecture will cover strategies to improve client compliance with diets and move forward in the diagnostic process. I have found that many owners will get stuck with diagnostic diet trials for lengthy periods, and some clients will be lost in the process. We must consider that extensive owner education and communication are needed to succeed and perform a dietary challenge to confirm the diagnosis. The clinician must also decide when the diet should be challenged, the diet trial extended, or a second or third dietary option. Several details go into those decisions, which will be covered in the lecture.

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THE ABDOMINAL EXPLORATORY: SURGERY AS A DIAGNOSTIC TOOL

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ABSTRACT BODY:

Surgical exploration of the abdominal cavity can be a valuable diagnostic tool, particularly when imaging is equivocal in helping discover what is wrong with an animal. Gastrointestinal obstruction is one such clinical example, where surgery can rule-in or rule-out obstruction when less invasive methods are unrewarding. Abdominal exploratory and subsequent organ biopsies are also used to assist in diagnosis of chronic disease and to differentiate between benign and malignant processes. Stomach and proximal intestinal biopsies are commonly performed using endoscopy. However, biopsies are limited to the mucosal surface and are often only representative of those areas. Abdominal surgery allows for complete exploratory and examination of the entire length of the gastrointestinal tract Full-thickness samples can be retrieved from the duodenum, jejunum, and ileum to diagnose diseases such as inflammatory bowel disease, lymphangiectasia, and lymphoma. Routine sampling of the liver and lymph node should be considered when in the abdomen for removal of a possible malignant process (e.g., splenic mass, focal intestinal mass).

What should the length of an abdominal incision be for complete abdominal exploratory?

What should be done with the falciform fat?

What is the value of a surgical safety checklist?

Similar to a physical examination, a routine for abdominal exploration should be developed such that nothing will be overlooked. An exploratory can be performed by system or by quadrant; one approach is “cranial-right-left-caudal”:

- Starting cranially, the diaphragm is observed and then the liver is evaluated. Each lobe of the liver can be gently manipulated such that all surfaces are examined. From left to right, the lobes of the liver are left lateral, left medial, quadrate, right medial, right lateral, and caudate. The gallbladder sits between the quadrate and right medial lobes and can be gently palpated; some prefer to express the gallbladder to ensure patency of the common bile duct, but this is often unnecessary if hepatobiliary disease is not suspected. The stomach is then examined and palpated, starting at the terminal esophagus to the fundus, body, pyloric antrum, and pylorus.
- Heading to the right side of the abdomen, the descending duodenum can be lifted gently out of the abdomen to examine the structures within the mesoduodenum (right limb of the pancreas, common bile duct, and portal vein), and then the mesoduodenum is used to hold back the small intestine to examine the right kidney, caudate lobe of the liver, and the caudal vena cava, often referred to the right gutter. A normal right adrenal gland is not readily visible as it sits under the liver alongside the caudal vena cava. It is difficult to continue to follow the small intestine oral to aboral as the duodenum is tethered at the duodenal flexure by the duodenal-colic ligament.
- Heading to the left side of the abdomen, the spleen is gently examined and palpated from the tail to the head, which is tethered to the greater curvature of the stomach by the short gastric vessels. The descending colon is then lifted to allow the mesocolon to hold back the small intestine and the left kidney and left adrenal gland can be examined (left gutter). The left adrenal gland is identified by the phrenicoabdominal vein coursing back to the caudal vena cava, bisecting the gland into cranial and caudal poles. The gastrointestinal tract is then evaluated from aboral to oral, from the colon to cecum and to the ileum, which is identified by the antimesenteric vessel. Continuing orally, the jejunum is palpated back to the duodenal flexure, taking note of the mesenteric lymph nodes. To examine the left limb of the pancreas, a hole is made in the superficial leaf of the omentum to enter the omental bursa and find the pancreas just dorsal to the greater curvature of the stomach.
- Caudally, the urinary bladder and distal ureters examined. In male dogs, the prostate is palpated by passing a finger dorsal to the urethra. In intact female dogs, the entire reproductive tract is examined from the ovaries to the cervix. In spayed female dogs, the uterine stump can be found dorsal to the bladder on the ventral surface of the distal colon. Medial iliac lymph nodes are found at the bifurcation of the aorta.

Abdominal Organ Biopsy

LIVER BIOPSY

There are multiple methods for sampling the liver. If there is a distinct lesion that needs to be sampled, its size and location often dictate the biopsy method. For diffuse disease or routine sampling, the liver can be biopsied along the edge or margin. Marginal biopsies can be performed using a guillotine method, multiple mattress sutures, or stapling equipment. Lesions away from the edge can be sampled using Baker skin biopsy punch or Tru-cut biopsy instrument. Hemorrhage can be controlled using digital pressure, electrocoagulation, or hemostatic agents such as GelfoamÒ or SurgicelÒ.

Laparoscopic liver biopsy safely can be performed through a single port or, more commonly, a two-cannula approach with the animal in dorsal or left lateral recumbency.[1] A 5 mm oval cup biopsy forceps is inserted through the instrument portal. The forceps are pushed into the edge of a liver lobe. The jaws are closed, and the sample held in place for 10-30 seconds before pulling it off the lobe. The site is observed for excessive hemorrhage.

LYMPH NODE BIOPSY

During abdominal exploratory, lymph nodes can be found around the pylorus and right limb of the pancreas, at the mesenteric root, along the descending colon, and at the aortic bifurcation (medial iliac lymph nodes). Lymph nodes can be sampled by fine-needle aspiration, wedge biopsy, or complete excision. The decision on method of sampling is based on involvement of surrounding structures and vascular supply to abdominal organs. Impression smears can be made from biopsy samples for immediate cytological examination to assist in ruling in or out lymphoma.

PANCREATIC BIOPSY

Due to risk of causing pancreatitis, veterinarians often avoid handling much less sampling, the pancreas. However, pancreatic biopsy is indicated to diagnose pancreatitis in cats, or differentiate benign versus neoplastic disease in dogs. If diffuse disease is present, the pancreas is best sampled from the left lobe to avoid damage to pancreatic ducts, the common bile duct, and the pancreaticoduodenal blood supply.

GASTRIC BIOPSY

Biopsies of the stomach are most commonly obtained through endoscopy. However, sampling by this method only retrieves pieces of the gastric mucosa. Surgical sampling retrieves full-thickness sections that may be beneficial in diagnosing schirrous gastric neoplasia that typically infiltrates deeper layers. Routine surgical biopsy is performed either in the ventral aspect of the body of the stomach between the lesser and greater curvature or in the left lateral aspect of the stomach if a gastrostomy tube is going to placed.

SMALL INTESTINAL BIOPSY

Like the stomach, intestinal biopsies are commonly performed using endoscopy. However, biopsies are limited to the mucosal surface of the duodenum. Segmental disease or abnormalities in the deeper layers of the intestine may be missed with this method, which is of particular concern in cats as the jejunum and ileum are thought to be the most common locations for gastrointestinal lymphoma. Full-thickness samples can be retrieved from the entire length of the small intestine. The large intestine is rarely surgically biopsied due to the poor healing characteristics of the colon and the risk of peritoneal contamination. A recent study examined complications associated with full-thickness biopsies of the small intestine.[2] Twelve percent of dogs died or were euthanized postoperatively due to biopsy site breakdown. Unfortunately, no predictors for risk of dehiscence were identified.

The most conventional method to biopsy the intestine is to make 2, 1 cm, parallel, longitudinal, full-thickness incisions in the antimesenteric aspect of the bowel using a scalpel blade. An alternative to this technique is to use a 4 to 6 mm Baker's biopsy punch to take a full-thickness sample from the antimesenteric border.

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QUE HAGO ANTE UNA OBSTRUCCIÓN INTESTINAL: ECOGRAFÍA O RADIOGRAFÍA

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ABSTRACT BODY:

INTRODUCCIÓN

La obstrucción intestinal (OI) es una patología frecuente en la clínica de pequeños animales. Los signos clínicos son similares a los que presentan otras patologías gastrointestinales. Cuando se sospecha de OI en base a la historia y al examen físico, las técnicas de imagen están indicadas para establecer el diagnóstico y aportar información sobre las posibles causas, localización, severidad de la obstrucción y diferenciar los casos que requieren cirugía inmediata de aquellos casos no quirúrgicos.

OBSTRUCCIÓN INTESTINAL

La OI o íleo se define como en un fallo del paso del contenido intestinal a través del tracto intestinal. Se debe diferenciar el íleo mecánico del funcional, ya que sus signos clínicos son similares. El íleo mecánico u obstructivo se produce como consecuencia de una obstrucción física del contenido intestinal mientras que el íleo funcional o no obstructivo es el resultado del cese del peristaltismo intestinal debido a alteraciones vasculares o neuromusculares a nivel de la pared intestinal. En ambos casos se produce un aumento del tamaño de las asas intestinales y éstas se visualizan dilatadas con gas, líquido o ambos. Sin embargo, cuando la obstrucción es mecánica podemos encontrar asas con un diámetro normal y otras dilatadas, mientras que si estamos ante un íleo funcional habrá distensión generalizada de todas las asas intestinales. Las técnicas de diagnóstico por imagen juegan un papel muy importante en el diagnóstico y manejo de la OI.

Actualmente en la mayoría de las clínicas se dispone tanto de radiología como de ecografía, y la pregunta que nos debemos de hacer es, ante un paciente con sintomatología compatible con OI que técnica debemos de utilizar o que técnica es más sensible para alcanzar el diagnostico.

Aunque se ha comprobado que la ecografía es más sensible que la radiografía para el diagnóstico de obstrucción intestinal, la radiografía sigue siendo en la mayoría de los centros la primera técnica de imagen a realizar. Esto se debe a su mayor disponibilidad y a que la ecografía es una técnica operador dependiente.

Por lo tanto, la pauta que se recomienda cuando se nos presenta un paciente con sintomatología compatible con OI es realizar una radiografía abdominal y si la radiografía no es suficiente para proporcionar un diagnóstico seguro de obstrucción intestinal, entonces puede ser necesario realizar otra técnica de imagen adicional, siendo la ecografía la más utilizada.

Radiografías simples

Se deben realizar tres proyecciones, lateral izquierda, lateral derecha y ventrodorsal que incluyan el diafragma, ya que pueden ser útiles para confirmar la sospecha de OI y en casos de neumoperitoneo.

El *signo radiológico* compatible con obstrucción de tipo mecánico es la presencia de dos tipos de poblaciones de asas de intestino delgado. Otros hallazgos radiográficos asociados con OI son: la posición o apariencia anormal del intestino delgado, la acumulación focal de material granulado (signo de grava) y la presencia de un cuerpo extraño o masa. El signo de grava es debido a una OI parcial crónica de localización distal.

Hay diferentes criterios descritos para el diagnóstico radiológico de OI. Cuando el diámetro de un asa intestinal supera 1.6 veces la altura de la L5, se considera que existe un 60% de probabilidad de que haya una OI, cuando el ratio es 2 existe una probabilidad del 90 %, y si es > a 2.4 es muy probable que exista OI y se recomienda o ecografía o directamente cirugía, y si esta entre 1.4-2.4 necesita un examen ecográfico para evaluar el intestino. Cuando el ratio es < de 1.4, es poco probable que haya OI mecánica.

Generalmente, la causa de la obstrucción no puede establecerse mediante radiografías simples excepto en los casos de hernias o cuerpos extraños radiopacos.

Hallazgos ecográficos

Los signos ecográficos de OI mecánica incluyen dilatación gastrointestinal, motilidad anormal, cambios en el contenido intestinal y en ocasiones líquido peritoneal. La ecografía es más sensible para diagnosticar la causa de la OI.

HALLAZGOS RADIOLOGICOS Y ECOGRAFICOS DE LAS DIFERENTES CAUSAS DE OBSTRUCCION INTESTINAL.

Las causas más comunes de OI son cuerpos extraños (CE), intususcepciones y neoplasias, mientras que las menos comunes incluyen el vólvulo y la obstrucción

intestinal debido a la incarceración de las asas intestinales. **Causas intraluminales**

Cuerpos extraños

Si el CE es radiopaco puede visualizarse en radiografías abdominales simples. Sin embargo, la mayoría de los CE son radiolúcidos y requieren de un estudio de contraste para visualizarlos o ecografía. Algunos de los CE radiolúcidos presentan un patrón de gas característico que permite identificarlos en radiografías simples, ej las mazorcas de maíz, las nueces y los huesos de melocotón. Los trozos de tela o tejidos a veces pueden ser reconocidos porque queda aire atrapado en el interior de estas. Si el CE produce una perforación intestinal, puede observarse gas libre en la cavidad abdominal y/o pérdida de visualización de serosas.

Los signos radiológicos de CE lineal incluyen la plicación de asas en forma de acordeón, acortamiento del intestino y el aumento de las burbujas de gas intraluminales en forma de lágrima o geométrica.

Ecográficamente la mayoría de CE producen una línea hiperecogénica asociada con sombras acústicas fuertes, aunque va a depender de las características físicas del mismo. En los CE lineales el intestino presenta un aspecto plegado u ondulante y la presencia de una interfase lineal brillante dentro de la luz intestinal que puede presentar sombra acústica.

Intususcepción

La intususcepción se produce cuando una porción del intestino, llamado intussusceptum, se introduce en la luz de un segmento intestinal adyacente, denominado intussuscipiens. En lo animales jóvenes suelen ser secundarias a enteritis por parásitos, infecciones bacterianas o víricas, mientras que animales adultos ocurren como consecuencia de cuerpos extraños, neoplasias o por proximidad a ganglios linfáticos aumentados de tamaño o pseudoquistes .

El aspecto radiográfico las intususcepciones a nivel distal, se produce una distensión severa y generalizada del intestino delgado. En algunos casos, se puede visualizar una estructura de opacidad tejido blando alargada ("forma de salchicha"). En la intususcepción ileocólica, el gas en el interior de colon delimita el final del segmento invaginado, lo que se denomina "signo de menisco".

Las intususcepciones tienen un aspecto ecográfico característico que en la mayoría de los casos permite establecer con confianza, un diagnóstico definitivo. El patrón ecográfico más frecuentemente observado en las secciones transversales del intestino es el de múltiples anillos concéntricos hiperecogénicos e hipoecogénicos alrededor de un centro hiperecogénico que representa al mesenterio atrapado.

Causas intramurales

Las masas proliferativas de la pared intestinal incluyen neoplasias, pólipos e infiltrados granulomatosos. El desarrollo de una obstrucción a causa de una masa depende del tamaño de la lesión y de si su crecimiento es hacia el interior de la luz.

Neoplasias

Los adenocarcinomas y linfomas causan engrosamientos focales de la pared intestinal, aunque el linfoma puede ser también difuso. En el perro el tumor más común es el adenocarcinoma, mientras que en el gato es el linfoma.

En la mayoría de los casos las radiografías simples no muestran alteraciones. Si la masa causa una obstrucción parcial crónica es posible visualizar el "signo de grava".

Las características ecográficas más frecuentes de las neoplasias intestinales son el engrosamiento de la pared intestinal y pérdida de capas.

Lesiones extraluminales

Adherencias, hernias y masas

Las lesiones que se originan fuera de la pared intestinal también pueden ser causa de obstrucción parcial o completa, y se incluyen las adherencias posquirúrgicas, hernias y masas abdominales de origen no intestinal.

La ecografía nos puede ayudar a observar el aspecto de las masas y el contenido del saco herniario.

Vólvulos

Los signos radiológicos son una dilatación de moderada a severa del intestino delgado con presencia de líquido y gas libre. La efusión peritoneal suele ser concurrente en estos casos.

En estos casos debido a la gran cantidad de gas la ecografía no sería la mejor técnica para el diagnóstico.

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COMO TRATAR FRACTURAS ABIERTAS

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COMO TRATAR FRACTURAS ABIERTAS

Se considera una fractura abierta (F.A.) cuando hay comunicación del hueso con el exterior a través de la piel. Toda fractura abierta se considera una urgencia ortopédica. En función de la gravedad se clasifican en 4 grados (Gustilo-Anderson 1976):

Grado I. Herida punzante del hueso que perfora la piel pero los tejidos blandos lo cubren y el hueso no se ve externamente. Herida < 1 cm. Ocurren de dentro a fuera. Generalmente acontecen en fracturas simples.

Grado II. La perforación de la piel ocurre de fuera a dentro y también van acompañadas de fracturas simples. Herida > 1 cm. El hueso emerge externamente.

Grado III. Daño importante de los tejidos blandos asociada a fracturas conminutas. Son producidas por traumatismos de importantes como accidentes por vehículos a motor o arma de fuego. En función de la lesión de los tejidos blandos y del daño nervioso y vascular se clasifican en IIIa, IIIb y IIIc.

Grado IV. Algunos autores incluyen en este grupo las fracturas que debido al daño neurovascular requieren amputación.

Manejo de una fractura abierta:

Estabilizar al paciente. Las F. A. son consecuencia de traumatismos muy severos que liberan mucha energía cinética y frecuentemente los animales son presentados en estado de schock y a veces politraumatizados. Se tiene que evaluar y estabilizar al paciente, con especial atención al sistema cardiopulmonar: ABCD´s (Airway, Breathing , Circulation, Drugs) y al estatus neurológico.

Evaluación de la lesión. La lesión vascular y nerviosa de la extremidad afectada nos indicará si es recuperable o no. La función vascular la valoraremos por la presencia o ausencia de sangrado, temperatura de la extremidad, pulso periférico (Doppler). La función nerviosa debe evaluarse con cuidado ya que la analgesia transitoria resultante del traumatismo nos puede inducir a error y en ocasiones se debe recurrir a la electromiografía pasados unos días del traumatismo para una valoración neurológica objetiva.

Tratamiento intrínseco del área afectada .Control de la hemorragia. Mediante la colocación de un apósito ejerciendo presión que además nos protegerá la herida de una posterior contaminación. Evitar el uso de torniquetes ya que la isquemia favorece la infección.

Limpieza de la herida. Se debe limpiar la herida y protegerla lo antes posible para evitar infecciones nosocomiales . Retirar los elementos contaminantes: pelos, suciedad....Evitar el contacto directo con agua del grifo ya que es citotóxica y puede provocar deshidratación celular. La con povidona yodada o clorhexidina sin diluir son irritantes. La mejor opción es el lavado copioso con suero fisiológico dejándolo caer por gravedad o a baja presión.

Desbridamiento quirúrgico. Debe retirarse todo el tejido necrótico no viable cada 24-48 h. La viabilidad del músculo se basa en las 4C´s : Color , Consistencia, Contractibilidad y Capacidad de sangrado. Todo el tejido que consideremos inviable se debe retirar así como fragmentos de hueso desperiostizados ya que serían origen de secuestros óseos. Una vez tengamos limpia la herida se puede recoger una muestra para cultivo bacteriano ya que son mas fiables que las muestras recogidas antes del lavado.

Antibioterapia.Toda fractura abierta se considera contaminada (presencia de microorganismos) y el “periodo de oro” se establece en unas 3 - 6 horas que es el tiempo que necesita para convertirse en una fractura infectada (replicación de microorganismos). El lavado, desbridamiento y antibioterapia dentro de estos límites pueden revertir una herida contaminada en una herida limpia. El control de la infección y el dolor son esenciales en el manejo de las F.A. El porcentaje de infección en las de tipo III puede llegar al 50%.

La mayoría de gérmenes aislados en fracturas abiertas corresponden a Gram +: Staphylococcus spp y Streptococcus spp y Gram - : Pseudomonas spp, Pasteurella spp.

Se ha comprobado que la administración intravenosa de antibiótico en las tres primeras horas post-lesión reduce drásticamente el desarrollo de osteomielitis. En espera del antibiograma se utilizan cefalosporinas de primera generación: cefazolina frente a Gram + asociada aminoglucósidos frente a Gram -. El uso de quinolonas es controvertido .Los cultivos bacterianos precoces tienen poco valor predictivo: sólo un 18% se correlacionaron con el microrganismo causante de la infección.

La administración local de antibiótico es muy eficaz en fracturas abiertas reduciendo la tasa de infección del

20% al 6% en un estudio de M. Humana. Se aplican esferas de cemento acrílico – PMMA- mezclado con aminoglicósidos que se dejan “in situ” 10-12 días que es el periodo que van liberando antibiótico de forma local. Después se deben retirar las esferas ya que se comportarían como un cuerpo extraño. El antibiótico seleccionado debe ser termoestable, hidrosoluble y disponible en forma de polvo. Las ventajas de la antibioterapia local es que se consigue una concentración local de antibiótico 20 veces superior a la obtenida por vía sistémica evitando los efectos secundarios de los aminoglicósidos ya que la concentración sistémica es baja. Se pueden sustituir el uso de esferas por esponjas bioabsorbibles que liberan el antibiótico gradualmente y se evita tener que retirarlas

Control del dolor. Los AINES combinados con analgésicos como la morfina, buprenorfina, fentanilo... disminuyen la inflamación y proporcionan un alivio excelente del dolor en las fracturas abiertas. Debe considerarse su uso después de la exploración neurológica ya que nos podría alterar el resultado del mismo.

Estabilización de la fractura. Las F.A. deben estabilizarse rápidamente, ya que se ha demostrado una clara relación entre inestabilidad biomecánica y osteomielitis. Para que la fractura consolide es indispensable la estabilidad mecánica. Las uniones retardadas son frecuentes debido a la lesión vascular. Un apoyo precoz después de reparar la fractura mejora la circulación a nivel del foco de fractura y favorece la osteogénesis. Las fracturas tipo I y la mayoría de tipo II se pueden estabilizar como si fueran fracturas cerradas

La fijación externa es una buena opción en fracturas abiertas de Grado III ya que biológicamente es menos agresivo que otros medios de fijación.

Cierre de la herida. Una vez estabilizada la fractura podemos optar por tres procedimientos para el cierre posterior de la herida:

Cierre primario inmediato que solo es recomendable en las de Tipo I con la condición de que la piel no sufra tensión y que se intervenga precozmente.

Cierre primario retardado, pasados unos días del accidente y cuando se ha vencido la infección.

Cierre secundario por segunda intención aprovechando la gran capacidad de granulación que tienen los animales. En casos extremos y una vez controlada la infección se puede recurrir a los implantes cutáneos.

Las fracturas por arma de fuego constituyen un apartado especial dentro de las fracturas abiertas y suelen ser consecuencia de accidentes fortuitos durante la caza

El potencial destructivo de un proyectil viene determinado por la energía liberada al producirse el impacto: $Ec = ½ mv^2$, por lo que la velocidad contribuye más en el efecto letal que la masa.

En general se distinguen 2 tipos de proyectil: Los de alta velocidad que alcanzan 600 m/seg. o más y los que no alcanzan dicho límite son considerados de baja velocidad.

Las escopetas de caza (perdigones) , las armas cortas, pistolas, revólveres ... son de baja velocidad , mientras que los rifles militares y los de caza mayor son de alta velocidad.

La gravedad del impacto también está en relación con la densidad y la elasticidad del tejido afectado. El hueso suele presentar fracturas conminutas debido a su alta densidad.

Si el proyectil es de alta velocidad se pueden producir fracturas lejos del sitio del impacto debido a las ondas de choque. Por otra parte se producen fragmentos óseos que se comportan como proyectiles secundarios causando daño tisular y destrucción vascular. La cavitación es un fenómeno consistente en la formación de una cavidad temporal producida por la onda de paso de un proyectil de alta velocidad, produciéndose una presión “sub-atmosférica” que arrastra por succión aire, pelos, restos de piel... dentro de la herida. Una vez ha pasado el proyectil la cavidad se colapsa i se favorece el desarrollo de infecciones anaerobias.

STATE-OF-THE-ART LECTURE: THE INTERTWINE OF FEAR AND PAIN: CRITICAL TO TREAT BOTH

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ABSTRACT BODY:

Introduction

Pain can be a powerful and unrelenting amplifier – or trigger – of fear, anxiety and stress (FAS). This is probably obvious if you have ever delayed a dental appointment because of fear of procedural pain or felt anxious that your knee pain would keep you from completing your favorite outdoor activity, like a long hike or a round of golf. What might not be so obvious is that FAS can also exacerbate pain. This is well-documented in human medicine and is often called 'anxiety-induced hyperalgesia'. The examples are numerous, here are a few: Patients with pain of osteoarthritis (OA) and higher anxiety scores had significantly lower pressure pain detection thresholds at sites local to - and distant from - the initiating pain source and high anxiety scores predicted increased risk of pain onset.[1] Children and adolescents with higher level of perioperative anxiety had a higher level of postoperative pain.[2] Children with chronic pain and high anxiety had greater pain-related functional impairment and were less improved with interdisciplinary rehabilitation, both at the time of admission to rehab and at a one-month recheck, compared to children with lower anxiety.[3] In the same study, decreased anxiety improved functional outcome.[3] Patients with more 'anxiety-like states' had higher pain scores on a numerical rating system (visual analog scale).[4] And the list goes on!

One theory on the patho-physiologic development of anxiety-induced hyperalgesia is based on the Gray-McNaugton theory, which states that, in situations that create conflict, the hippocampus amplifies the neural representation of the aversive conflict-causing event.[5] If pain is the aversive event, that pain could be amplified to a 'worst case scenario' as a survival mechanism that drives the animal to avoid the cause of the pain, but which also creates FAS regarding the cause of the pain, even if the pain was seemingly minor.[6] This is a crucial concept for the individual (person or animal) that is experiencing pain because pain is not **what** you feel, it is **how it makes** you feel and even presumably mild pain can have a large negative effect. These concepts are largely based on humans since humans can self-report both their pain and FAS levels. Treatment of both pain AND anxiety is fairly common in human medicine.

What does this have to do with veterinary medicine? Simple physiology – and science. The pain pathway is well-preserved across mammalian species so for a scientific certainty, if a stimulus is painful to a human, it is painful to an animal – at least to a mammal (and many other species, but we are focusing on mammals). In addition, like humans, animals suffer from fear/anxiety/stress (FAS). So the assumption that FAS could exacerbate pain in animals, and vice-versa, is scientifically valid. Potential evidence in veterinary species includes the fact that dogs with chronic musculoskeletal pain are more likely to experience fear-related noise sensitivity.[7] Stress suffered by cats with idiopathic cystitis[8] are likely to have worsening pain as a component of the stress, as has been reported in women with the same condition.[9] As occurs in human medicine, there are likely numerous other examples of the pain/fear relationship in dogs and cats – it is just a matter of identifying them, which can be difficult in species that don't self-report their pain/FAS levels. However, even in absence of identified examples, animals with pain – especially those that have behavioral changes associated with that pain – are predicted to need both analgesics and anxiolytics, at least in the early stage of treatment (ie, until the pain is controlled) or intermittently when stress-triggers occur (eg, extra humans in the house).

Treatment Options/Ideas

As an example, the painful patient with FAS should no longer receive only NSAIDS or grapiprant, but NSAIDS or grapiprant PLUS benzodiazepines or fluoxetine or other anxiolytics. Interestingly, some of the anxiolytics directly provide analgesia as well as decreasing the pain response by decreasing FAS. This analgesic effect occurs via the descending limb of the pain pathway, which is a feedback loop from the brain back down to the spinal cord. The modulation phase of the pain pathway in the spinal cord is most often responsible for amplification of the pain sensation, but reduction of the pain sensation can also occur through this feedback loop. Serotonin-norepinephrine reuptake inhibitors (SNRIs) are likely the most effective anxiolytic analgesics. Duloxetine is an example of a SNRI analgesic in human medicine that is used to treat a variety of pain conditions including pain from fibromyalgia, diabetic neuropathy, diabetes (diabetic

neuropathy) and or chronic muscle or joint pain. Unfortunately, no SNRIs are approved in veterinary medicine and the selective serotonin reuptake inhibitors (SSRIs) like fluoxetine do not seem to work as well. Amitriptyline, a tricyclic antidepressant, may be useful for treating neuropathic pain in dogs[10] and cats. N-methyl-D-aspartate (NMDA)-receptor antagonists may also play a role in both analgesia and behavior modification. Ketamine is used commonly in human medicine at subanesthetic dosages to treat a variety of behavioral conditions like anxiety and depression. [11] Ketamine at subanesthetic dosages also provides analgesia for both acute[12] and chronic[13] pain. Amantadine, which is an orally-administered NMDA-receptor antagonist is proven effective as part of a multimodal protocol for treatment of OA pain in dogs[14] and cats[15] and could potentially play the same role, as could memantine (no data). Gabapentin is used both for relief of chronic pain and for decreased stress and has some proven efficacy in both dogs and cats. Pregabalin is used for more pain conditions than gabapentin in human medicine and may also be more effective in veterinary patients. Currently, only PK data are available. Perhaps CBD products could be beneficial since there is evidence that they may provide both decreased stress and analgesia. Alpha-2 agonists decrease cortisol release and provide analgesia. Injections of the drugs have a fairly short duration of action but could be useful for immediate pre- or post-procedural FAS and pain. And perhaps the longer lasting oral mucosal gel, with its longer duration, would be practical for use at home prior to transport of the pet to the veterinary clinic. The drug (Sileo®) is FDA-approved for treating noise sensitivity. Perhaps the oral alpha-2 agonist used in human medicine for blood pressure control, clonidine, could play a long-term role in these patients. Other drugs could be adapted from human medicine for use in dogs and cats. For now, we are utilizing only a few drugs in these categories, but perhaps the intertwine of pain and FAS should prod us to use these drugs more often, as valid pharmacokinetic/pharmacodynamic information supports, of course. In addition, in humans, acute procedural pain intensity can be alleviated using verbal preparation of the patient.[16] Although we cannot verbally prepare our patients, behavioral modification techniques that decrease FAS prior to painful procedures should be strongly considered. In addition, Fear Free and gentle handling techniques should be a major component of patient care for all patients, and should be a major focus for those with both FAS and pain.

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EXPIRATORY DYSPNEA: INTERACTIVE CASE DISCUSSION

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ABSTRACT BODY:

Clinical Approach:

Patients with expiratory dyspnea have an intrathoracic disease. The next step to better define its origin is to determine the pattern of ventilation (Figure 1). *Obstructive diseases* are associated with increased airway resistance in the tracheobronchial tree. Patients with obstructive disease have decreased expiratory flows and hyperinflated lungs (e.g., cats with asthma). *Restrictive diseases* are those where expansion of the lungs is restricted (e.g., pulmonary fibrosis, pleural effusion). Lungs of patients with restrictive disease operate at smaller volumes and the patient has a rapid shallow breath. In patients with a non-pulmonary cause of restrictive, residual volume is normal or increased.

Obstructive Expiratory Dyspnea

Obstructive expiratory dyspnea occurs in patients with intrathoracic airway diseases (table 1). Patients may have wheezes and cough. *Wheezes* are continuous musical sounds generated by air forced to pass through a narrow region abruptly into a wider region in the larger airways. Good quality chest radiographs, CT, and tracheal wash are necessary to rule in or rule out specific diagnosis.

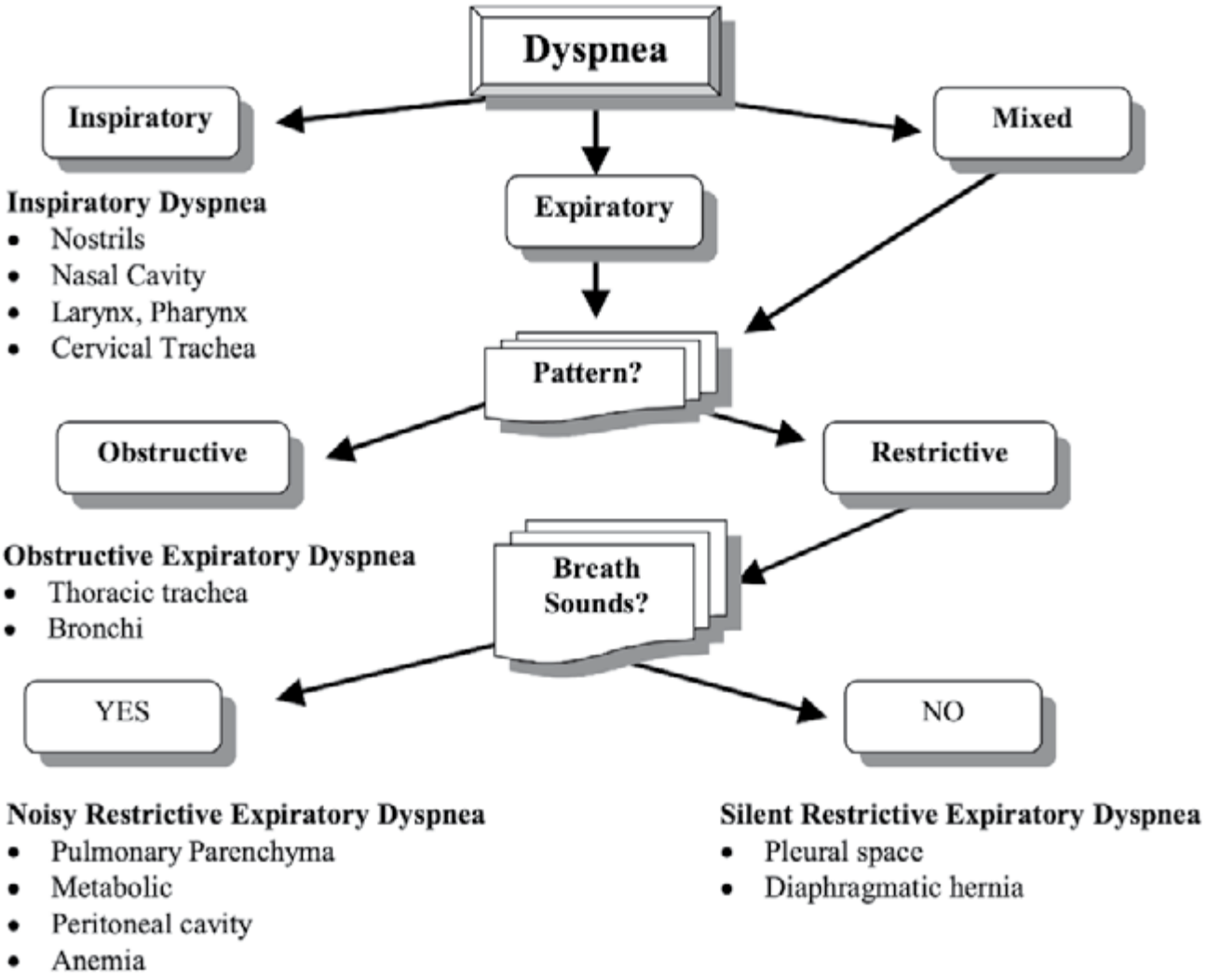

Figure 1. Localizing the origin of the dyspnea

Table 1. Causes of Obstructive Expiratory Dyspnea

Thoracic Trachea Bronchial tree
Tracheal collapse Bronchitis
Tracheobronchitis Asthma
Extraluminal compression Extraluminal compression
Foreign body Neoplasia
Neoplasia Lymph nodes
Enlarged left atrium

Noisy Restrictive Expiratory Dyspnea

Noisy restrictive expiratory dyspnea occurs in patients with parenchymal pulmonary diseases (table 2). Pulmonary sounds are audible and abnormal sounds like crackles may also be heard. *Crackles* are short, explosive, non-musical sounds that are a non-specific sign of small airway disease. Cough may occur if small airways are also involved and is usually not loud. Chest radiographs and tracheal wash or bronchoalveolar lavage are necessary to rule in or rule out the differentials.

Table 2. Causes of Noisy Restrictive Dyspnea

Pulmonary Parenchyma Abdominal distension
Edema Ascites
Pneumonia Pregnancy
Fibrosis Organomegaly
Neoplasia Gastric dilatation-volvulus
Hemorrhage/contusion Neoplasia
Embolism
Metabolic
Anemia
Anxiety/Fear
Metabolic acidosis?

Silent Restrictive Expiratory Dyspnea

Silent restrictive expiratory dyspnea occurs in patients with pleural cavity disease (table 3). Pulmonary sounds are absent or may be heard at specific locations (e.g., dorsal lung fields in patients with pleural effusion). Cough is usually absent. Chest radiographs, thoracic ultrasound, CT, and thoracocentesis may be necessary to rule in or rule out the differentials.

Table 3. Causes of Silent Restrictive Expiratory Dyspnea

Pleural Cavity
Pneumothorax
Pleural effusion
Diaphragmatic hernia
Chest tumors

Careful interpretation of the information obtained in the history and physical examination determining the timing and pattern of the dyspnea allow the veterinary practitioner to anatomically locate the origin of the dyspnea in most cases. Direct inspection visually or endoscopically, radiographs, CT, and cytology are necessary to determine the cause of the dyspnea.

Clinical Cases

Cases will be presented during the lecture to apply the concept described above.

References available upon request

HEMOPLASMOSIS FELINA, QUÉ ROL CUMPLEN REALMENTE LAS PULGAS?

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ABSTRACT BODY:

INTRODUCCIÓN

La hemoplasmosis felina puede ser causada por 4 tipos de Mycoplasmas: *Mycoplasma haemofelis* (MHf), *Candidatus* Mycoplasma haemominutum (CMhm), *Candidatus* Mycoplasma turicencis (CMt) y *Candidatus* Mycoplasma haematoparvum like. Se sugiere que tienen distribución mundial, aunque la prevalencia varía según la distribución geográfica.

Diferentes estudios hechos a nivel mundial reportan al MHf y CMhm como los dos Mycoplasmas con mayor prevalencia, siendo el *Candidatus* Mycoplasma haematoparvum like el menos prevalente. La patología puede encontrarse tanto en felinos domésticos como silvestres y produce anemia por destrucción del glóbulo rojo. El micoorganismo se adhiere a la pared celular del eritrocito debilitandola, lo que puede conllevar a hemólisis intravascular. El bazo, generalmente fagocita estos eritrocitos, pudiendo ocurrir tambien, hemólisis intravascular. Se sabe que los gatos pueden ser portadores a hemoplasmas y no necesariametne desarrollan la enfermedad clinica. Generalmente la presencia de algun evento estresante o inmunosupresión pueden ser los detonantes para que se inicie la hemólisis.

El diagnóstico citológico carece de sensibilidad y especificad, por lo que se debe hacer pruebas moleculares (PCR) en sangre. Los signos clínicos de la enfermedad son los asociados a la anemia y hemólisis extravascular (esplenomegalia). Los hallazgos de laboratorio pueden ser diversos, siendo la anemia la única constante. El tratamiento requiere el uso de antibióticos, como doxiciclina, marbofloxacino o idealmente pradofloxacino. Para frenar la reacción inmunomediada se pueden usar cortocoides y es necerario hacer un tratamiento de sosten, según cada caso en particular (alimentación asistida, transfusión sanguínea, etc.).

TRANSMISION DE LOS HEMOPLASMAS FELINOS

Siempre se ha considerado a la pulga del gato (*Ctenocefalides felis*), como el principal transmisor de los Mycoplasmas hemotrópicos felinos. Sin embargo, diversos estudios, generan mucha duda sobre su real rol en la transmisión.

Las Ctenocefalides felis ingieren MHf y CMhm, cuando se alimentan de los gatos infectados. Sin embargo cuando se alimentaron gatos con pulgas infectadas con MHf y CMhm, no se documentó la infección (Lappin, 2011). En otro estudio, expusieron gatos gatos positivos y negativos a hemoplasmas, a pulgas infectadas con Mhf y CMhm (PCR positivas); ninguno de los gatos negativos se contagió (Woods, et al. 2006). Un estudio hecho en Canada (Kamrani, et al. 2008) demostró que a pesar de que hasta un 50% de las pulgas encontradas en gatos callejeros contenían ADN de CMhm, sólo un 13% de los gatos fueron PCR positivos. Por otro lado, se ha documentado la infección en gatos libres de ectoparásitos.

Estos estudios permitieron concluir que la ingestión de Ctenocefalides felis no es una forma importante de transmisión de MHf y CMhm en los gatos. Se ha demostrado la presencia de MHf, CMhm y CMt en garrapatas (Duplan, et al, 2018), aunque aún no se determina la posibilidad de contagio al ingerirlas. Se puede concluir que falta evidencia para determinar la transmisión por vectores, sugiriendo otras formas de contagio.

Se ha demostrado la presencia de CMt en la saliva y sangre de gatos infectados. Un estudio inoculó sangre vía oral y saliva vía nasal a gatos negativos, (semejando contacto social), no logrando documentar la transmisión. Al mismo tiempo inocularon saliva y sangre contaminada, vía subcutánea a gatos negativos (semejando una conducta agresiva – mordida -), y únicamente los gatos inoculados con sangre dieron positivos a CMt. Esto sugiere que el contacto social no es fuente de contagio, pero si la exposición con sangre contaminada. (Museux, et al. 2009).

De lo anterior se puede concluir que la transmisión principalmente ocurre con el ingreso de sangre contaminada al organismo del gato negativo. Es por lo tanto una posibilidad la transmision via transplacentaria. Adicionalmente puede ocurrir la transmisión vía iatrogéncia, a través de material contaminado (agujas, material quirúrgico, etc.) y vía transfusiones sanguíneas. Se sugiere que los gatos donantes deben ser hemoplasma negativos (vía PCR) para prevenir infecciones en el gato receptor, ya que el donante puede ser portador asintomático.

CONCLUSIONES

La forma real de transmisión de los hemoplasmas esta aún en debate. Los vectores (pulgas y garrapatas) son una posiblilidad. Debido a esto se recomienda el uso de antiparasitarios externos regularmente en los gatos. La transmisión vía agresiones es otra posibilidad, por lo que debemos evitar las peleas entre gatos. El contacto con sangre infectada esta demostrado, por lo que se debe tener especial cuidado en el manejo del material e instrumental veterinario, y durante las transfusiones sanguíneas.

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HOW I TREAT MANDIBULAR FRACTURES

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ABSTRACT BODY:

Mandibular fractures can be treated by several methods. Among them, plates and screws, external fixation should rarely be used, once they can damage the mandibular canal or teeth roots. Avoid them! Some caudal fractures can be fixed with plates, wire cerclage, but always avoinding roots and the canal. Radiograph is mandatory. When rostral fractures are present, acrylic resin is very useful, and non-invasive. It is applied over the prepared teeth, fixed, and remain until bone consolidation. The teeth are acid etched, dryed, and the resin applied. Avoid contact between resin and the gingiva, protecting the periodontium. The resin is hard enough to support masticatory forces. The volume of resin is important to mainting occlusion as normal as possible. Antiseptic is used daily as oral wash. The resin is removed after 4 to 6 weeks, in most cases, under general anesthesia, with burs and forceps. Teeth are cleaned, and polished. Radiographs are taken before removal of the resin.Other methods exist, however, this is the most simple, and cheap, and protects the mouth tissue in a way.

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HOW I TREAT CAUDAL STOMATITIS

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ABSTRACT BODY:

Caudal Stomatitis

This is another relatively recent disease process in cats that is frustrating us at present. The best description is a severe immune mediated reaction to dental tissues, but we really don't know. Some feel that this may be a group of disease processes that look the same clinically which is why they can be very frustrating to treat.

The history will generally include anorexia, drooling, gagging, and pain during mastication. Physical exam will typically include a thin pet with unkempt fur. The oral exam will reveal severe stomatitis usually over all teeth. The inflammation will most commonly be worse on cheek teeth than canines and incisors. However, faucitis is the key clinical finding. Severe hyperplastic inflammation to the gingiva can result from periodontal disease, however caudal stomatitis will not be present.

Medical Therapy*:* Most medical therapies will work for a while, however in general resistance will start within a year or less. In addition, most therapies have side effects worse than the disease process in and of itself. In general, medical therapy is very frustrating to the practitioner and client.

Corticosteroids are the mainstay of most medical therapy today. It is generally very effective at first and is relatively inexpensive for the client. In my experience, injectable (depomedrol 10 mg IM) is much more effective than oral preparations in my experience. However, they will typically loose effectiveness after a year or so requiring higher and higher doses at shorter increments. This generally results in significant deleterious effects. About 10% of stomatitis cases we treat are already diabetic!!!

Surgical Therapy*:* Extraction is currently the ONLY effective long-term treatment for this disease process in cats. In our experience, the sooner this is done, the better that cats do both post-operatively as well as long term.

For extractions to be successful, the teeth must be COMPLETELY removed. Therefore, post-operative radiographic confirmation of complete extraction of the tooth roots is recommended. Following the insurance of complete removal of the teeth, perform aveloplasty to remove the periodontal ligament and smooth rough bony edges. This is typically performed do this with a rough diamond bur.

Studies report a 60% success rate when all teeth caudal to the canines are extracted, however our experience has not been as good. However, whole mouth extractions have a success rate of approximately **90-95%** for clinical remission. Slight faucitis may remain, but pets are comfortable. In addition, the rare cases that don't completely respond are generally much more responsive to medical therapy.

If there is NO inflammation to the canines or incisors (which is rare), then the owner is given the option of leaving the canines. However, if these are inflamed, all teeth should be extracted. If the teeth are ankylosed, complete root pulverization may be necessary.

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WSAVA ZOONOSES GUIDELINES IN DOGS AND CATS - ENTERIC, BITES AND SCRATCHES

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ABSTRACT BODY:

Zoonotic diseases are defined as being common to, shared by, or naturally transmitted between humans and other vertebrate animals. Humans are infected with zoonotic agents from direct contact with the infected pet, contact via contaminated food or water, from shared vectors, and from the shared environment. Direct contact with animal feces (enteric zoonoses), respiratory secretions, urogenital secretions, or infected skin and exudates, as well as bites and scratches can result in human infections.

Pets that are healthy, being dewormed, and on flea and tick control are unlikely to be dangerous for their owners. Owners should bring their pet yearly to the veterinarian to assess for zoonotic risk and to appropriately provide preventative health care. Veterinarians should work closely with the family physician on pet ownership issues.

Some zoonotic agents are transmitted between animals and man by shared vectors like fleas, ticks, or mosquitoes. *Rickettsia rickettsii* (ticks), *Ehrlichia* spp. (ticks), *Borrelia burgdorferi* (ticks), *Rickettsia felis* (fleas), *Bartonella* spp. (fleas, ticks), *Anaplasma phagocytophilum* (ticks), *Dirofilaria immitis* (mosquitoes), *Dipylidium caninum* (mosquitoes), and West Nile virus (mosquitoes) are examples of vector borne zoonoses. The pet brings the vector of the organism into the environment resulting in exposure of the human. *Bartonella* spp. are notable examples as some species like *B. henselae* survive in flea feces for days. Flea and tick control should always be maintained on our client's animals and infested animals that are seen in the clinic should be treated immediately. Use of flea control products have been shown to block transmission of *B. henselae* amongst research cats and so theoretically could lessen transmission to humans. Neurobartonellosis (headaches, blurred vision) in veterinary health care providers is now recognized as an important zoonotic disease syndrome related to *Bartonella* spp. in flea frass.

Some zoonotic agents including *Histoplasma capsulatum*, *Coccidioides immitis*, *Blastomyces dermatitidis*, *Cryptococcus neoformans*, and *Aspergillus* spp do not usually infect humans from direct with the infected pet but are acquired from the same environmental source. In contrast, *Sporothrix* spp. and dermatophytes are direct zoonoses.

Most of the agents discussed in this lecture can infect and cause disease in anyone, but disease is generally more prevalent or more severe in those that are immunodeficient. Humans with AIDS are discussed most frequently, but there are many more immunodeficient individuals including the very old, the very young, and those receiving chemotherapy for immune-mediated diseases, organ transplantation, or neoplasia. Humans are unlikely to contract zoonotic diseases from contact with their pets and so in most cases do not need to relinquish their animals. The Centers for Disease Control of the United States online site, Healthy pets Healthy People (http://www.cdc.gov/healthypets/index.html) is a great resource for veterinarians and owners. The American Association of Feline Practitioner's Zoonoses Guidelines states 'All human or animal care providers should provide accurate information to pet owners concerning the risks and benefits of pet ownership so that an informed decision about acquiring and keeping pets can be made' (Lappin et al, 2019). The Companion Animal Parasite Council also provides great information to help with decision making about zoonotic parasitic diseases (www.capcvet.org; www.petsandparasites.org/). The WSAVA One Health Committee encourages veterinarians and physicians to work closely together to determine the risks associated with pet ownership for individuals and their families.

Enteric zoonoses. There are multiple infectious agents of the gastrointestinal tract that potentially can be shared between pets and humans. Since some enteric zoonotic agents are infectious when passed with feces (*Campylobacter* spp., *Salmonella* spp., *Giardia* spp., *Cryptosporidium* spp. and others), direct contact with infected animals can result in human infections. However, it is felt that most enteric zoonoses result from ingestion of the infectious agent in contaminated food, water, or other environmental sources. *Giardia* spp., *Cryptosporidium* spp., *Toxocara* spp., and *Toxoplasma gondii* are notable examples. *Toxoplasma gondii*, hookworms, and roundworms require a period outside the host prior to becoming infectious. While on the zoonoses

list, *Giardia* and *Cryptosporidium* spp. of dogs and cats are rarely detected in people and human strains have not been associated with illness in pets. The minimal diagnostic plan to assess for enteric zoonoses in pets with diarrhea includes a fecal flotation, *Giardia* spp., screening procedure, and fecal wet mount. Fecal culture should be considered if *Salmonella* spp. or *Campylobacter* spp. are on the list of differential diagnoses. In the United States, heartworm preventatives that control hookworms and roundworms are recommended year round (http://www.capcvet.org). Dogs and cats with normal stool are not considered human health risks.

Bite, scratch, or exudate exposure zoonoses. Approximately 300,000 emergency room visits per year are made by people bitten by animals in the United States. Local infections are most common but 28% to 80% of cat bites become infected and severe sequelae can develop. Immunodeficient humans or humans exposed to *Pasteurella* spp., *Capnocytophaga canimorsus* (DF-2), or *Capnocytophaga cynodegmi* more consistently develop systemic clinical illness. Splenectomized humans are at increased risk of developing bacteremia. *Mycoplasma* spp. and L-form bacteria infections of people has been associated secondary to dog or cat bites.*Bartonella* spp. *Yersinia pestis*, and *Francisella tularensis* infections of humans can be associated with bites and scratches but these agents are also vector-associated zoonoses. Of the many fungal agents that infect both humans and animals, only *Sporothrix* spp, and the dermatophytes have been shown to infect humans upon direct exposure. *Histoplasma*, *Blastomyces*, *Coccidioides*, *Aspergillus*, and *Cryptococcus* infections of humans and animals can occur in the same household, but infection of humans generally results from a common environmental exposure rather than by direct contact with an infected animal. Rabies is still the only significant small animal viral zoonosis in the United States. Psuedorabies is a herpesvirus that infects pigs; dogs and humans can develop self-limiting pruritic skin disease following exposure. Recently, a presumptive case of monkeypox in a dog from exposure to its owners was reported. Feline retroviruses are not zoonotic.

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PSYCHOLOGICAL SAFETY: WHY IT MATTERS FOR PERFORMANCE AND HOW TO CREATE IT

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ABSTRACT BODY:

Imagine working in a team that embraces diversity, where you and your unique skills and talents are valued. A team where it feels safe to ask for help, where mistakes are not talked about as personal failures, and it is safe to take risks. If this description sounds familiar, you can assume that you work in a "psychologically safe" work environment.

The term psychological safety was first coined in 1990 by William A. Kahn as "being able to show and employ oneself without fear of negative consequences to self-image, status or career." [1] Later on organizational behavioural professor Amy Edmondson of Harvard, described psychological safety as "a belief that one will not be punished or humiliated for speaking up with ideas, questions, concerns, or mistakes."[2]

Edmondson first introduced the construct of "team psychological safety" and defined it as "a shared belief held by members of a team that the team is safe for interpersonal risk taking."[2]

Timothy R. Clark, an international authority in the fields of psychological safety & innovation, describes psychological safety as a condition in which human beings feel included, safe to learn, safe to contribute and safe to challenge the status quo. All without fear of being embarrassed, marginalized, or punished.[3]

A question that inevitably arises is: how is psychological safety different than trust? According to Amy Edmondson, psychological safety is experienced at a group level, while trust refers to the interactions between two individuals or parties. For example, you might trust one colleague, but not another.[2]

A two-year study by Google, Project Aristotle, discovered five components found in high-performing teams[7]:

- Psychological safety – team members feel safe to take risks and be vulnerable in front of each other.
- Dependability – team members deliver high quality outcomes in a timely manner.
- Structure and clarity -team members have clear roles, responsibilities, and action plans
- Meaning of work – work is personally important to team members.
- Impact of work – team members believe that their work is meaningful and that it generates positive change.

Of the five, psychological safety was deemed the most important. The Google researchers found that individuals on teams with higher psychological safety are less likely to leave their job, and more likely to embrace diverse ideas, increase sales, and receive high performance ratings by their managers.[7] The researchers found that how the team worked together mattered more than who was in the team. [7]

What is the impact of a workplace environment with low psychological safety? A toxic environment significantly impacts performance, because people need psychological safety before they can focus on performance.[3]

Here are some potential signs that team members do not feel psychologically safe[8]:

- They are more likely to hold up than speak up during important conversations.
- They progressively become quieter and share fewer ideas.
- They rarely, if ever, admit weaknesses or mistakes.
- They tend to avoid difficult conversations.
- Leaders typically dominate the conversation during meetings.
- They rarely give or request feedback.
- They don't ask one another for help.
- There are hardly any disagreements within the team.

Timothy R. Clarke in his book "The Four Stages Of Psychological Safety" describes a conceptual model of four stages of psychological safety that teams can move through progressively:

- **Inclusion Safety** – members feel safe to belong to the team. They are comfortable being present, they feel included and appreciated. This relates to the human need to belong and gain membership.
- **Learner Safety** – This means being able to ask questions, give and receive feedback, experiment, and even make mistakes. Humans instinctively look for learner safety before they engage in the learning process.
- **Contributor Safety** – members feel safe to participate using their talents and abilities to make

a difference and contribute their own ideas without fear of embarrassment or ridicule.

- **Challenger Safety** – members can question others' ideas or suggest significant changes to ideas, plans or ways of working. Challenging the status quo without fear of retaliation paves the way to innovation.

Successful organisations have identified that cultivating a sense of trust and safety in the workplace is a key factor in engaging with and retaining their workforce. It does not mean that team members always agree with each other, nor that they are merely polite to each other and never engage in conflict. To the contrary, psychological safety is about honesty, productive disagreement, and the free exchange of ideas. These elements are essential for learning and innovation. [2,3]

A team of social psychologists recently verified the link between psychological safety and creativity.[5] According to Timothy R. Clark, "innovation is the process of connected people connecting things".[3] If innovation comes from connecting different things, the leader's job is to stimulate differences in the first place. Peter Drucker said, "Disagreement is needed to stimulate the imagination.[6]

How can we create psychological safety in the workplace? Amy Edmondson offers three simple things individuals can do to foster team psychological safety:

- Frame the work as a learning problem, not an execution problem.
- Acknowledge your own fallibility.
- Model curiosity and ask lots of questions.

What can you do both as a team member and a leader to create psychological safety in the workplace?

- Solicit input and opinions from the group.
- Share information about personal and work style preferences and encourage others to do the same.
- Refrain from criticizing, condemning, or complaining.
- Show respect for the other person's opinion.
- Use encouragement. Provide constructive feedback and propose a way forward.
- Show sincere appreciation for people's unique contributions.
- Listen more, speak less.

Research shows that the key drivers of psychological safety include familiarity among team members and the quality of those relationships based on prior interactions [9].

As research from the MIT Human Dynamics Lab attests, the faster you get to know each other on a deeper level, the more effectively you can work together. More contact and context tend to create more empathy .[10]

Make a conscious effort to get to know the members of your team. Spend time with them, acknowledge their individual attitudes and listen to what they have to say. In fact, if you listen long enough, people will often reveal what they would otherwise conceal. [3]

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ACTUALIZACIÓN SOBRE ENFERMEDADES CRÓNICAS GASTROINTESTINALES EN GATOS

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ABSTRACT BODY:

Introducción y presentación general

Las enfermedades gastrointestinales crónicas, o enteropatías crónicas, comprenden un grupo de enfermedades de ocurrencia frecuente en la clinica medica felina. Forman un grupo de enfermedades poco comprendidas, que causan manifestaciones persistentes o intermitentes del tracto gastrointestinal, además del infiltrado inflamatorio asociado, así que la enfermedad inflamatoria intestinal (EII) y el linfoma de células pequeñas son sus principales presentaciones.

Estas son las enfermedades gastrointestinales más frecuentes en gatos mayores de 10 años, incrementándose su incidencia en la última década. Sus principales manifestaciones clínicas incluyen vómitos, pérdida de peso, cambios en el apetito y diarrea. Los animales con síntomas a largo plazo pueden quedarse demacrados, con su intestino engrosado, linfadenopatía mesentérica y malestar abdominal craneal (muchas veces relacionado con enfermedad hepática y/o pancreática).

Debido a que los síntomas más comunes son inespecíficos, el abordaje diagnóstico comienza con la exclusión de enfermedades extraGI y/o sistémicas que generan manifestaciones clínicas similares. Las endocrinopatías como el hipertiroidismo, la enfermedad renal crónica, la hipercalcemia, los retrovirus y los parásitos son problemas importantes que deben excluirse antes de realizar exámenes diagnósticos más específicos en el triaje de estos pacientes.

Exámenes de imagen

El uso del examen de ultrasonografía es de fundamental importancia en los casos de enfermedad gastrointestinal crónica. Además de ayudar a descartar causas que puedan presentar síntomas similares, permite la evaluación de capas intestinales, estratificación y engrosamiento, además de permitir la recolección de material de los ganglios linfáticos y líquido libre. El examen de ultrasonografía también ayuda en la toma de decisiones sobre la modalidad de recolección de biopsias más apropiada.

Lo principal hallazgo en las enteropatías crónicas es el engrosamiento intestinal, siendo común el engrosamiento de la capa muscular y una relación muscular:mucosa > 1,0 es indicativa de un segmento intestinal anormal.

El hallazgo de dos (o más) segmentos del intestino delgado con espesor de la pared intestinal superior a 2,8 mm, o un segmento superior a 3 mm, tiene correlación histológica con enteropatías crónicas en más del 95% de los casos. Sin embargo, los datos del examen de ultrasonografía no ayudan a diferenciar la EII del linfoma de células pequeñas.

Diagnóstico y obtención de biopsias

El diagnóstico de enteropatías crónicas se obtiene tras excluir causas extragastrointestinales en gatos con mala respuesta a las terapias convencionales, incluido el uso de una dieta hipoalergénica. En estos animales, la obtención de biopsias intestinales sigue siendo el estándar de oro para el diagnóstico definitivo. La evaluación histopatológica permite identificar los infiltrados celulares predominantes en la mucosa y otras capas intestinales, diferenciando la EII (generalmente un infiltrado predominante de linfocitos +/- plasmocitos) del linfoma de células pequeñas. Además, permite excluir otras causas de enfermedades infiltrativas, como linfomas de células grandes/granulos grandes, otras neoplasias (carcinomas, mastocitomas) y enteritis neutrofílica/granulomatosa.

Cada metodología de biopsia (endoscópica x laparotomía) tiene ventajas y desventajas, y no existe una metodología “perfecta”.

Las biopsias endoscópicas son mínimamente invasivas y tienen menos morbilidad, permiten la visualización directa de la mucosa (y la elección de los mejores sitios de recolección), pero tienen un alcance limitado del tracto gastrointestinal, con poco o ningún acceso al yeyuno (especialmente en las regiones media y distal).

Por otro lado, las biopsias quirúrgicas, aunque invasivas, permiten la recolección de material de cualquier segmento intestinal, además de sitios extraintestinales (hígado y páncreas). Sin embargo, permite la recolección de un número limitado de fragmentos, además de presentar mayor morbilidad.

Cada caso debe ser evaluado individualmente, teniendo en cuenta la ubicación de los segmentos más afectados mediante ultrasonografía y el estado general del paciente, para una mejor toma de decisiones.

¿EII o linfoma?

En algunos casos, la diferenciación entre enteritis

linfoplasmocítica y linfoma de células pequeñas es difícil, pudiéndose aplicar la inmunohistoquímica y la prueba de clonalidad (PAAR) en la diferenciación de las enfermedades. Sin embargo, la sensibilidad de la clonalidad en el diagnóstico del linfoma oscila entre el 50-70%, con una baja especificidad (alrededor del 33%). Son necesarios más estudios sobre los efectos beneficiosos reales del uso de estas tecnologías en las enteropatías crónicas.

Estudios recientes que utilizan espectrometría de masas guiada por histología han demostrado una sensibilidad y especificidad superiores a la clonalidad en la diferenciación de la enteritis linfoplasmocítica y el linfoma de células pequeñas, que pueden ser de gran valor, cuando están disponibles comercialmente.

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GESTACIÓN EN LA PERRA

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Ha escrito el libro Reproducción y neonatología felina y canina publicado en el 2011

Ha coordinado y escrito el libro, Medicina Pediátrica en Pequeños Animales publicado en 2012 por Editorial Servet

Es miembro de la EVSSAR y miembro del GERPAC.

Ha sido miembro del Comité científico de AVEPA en el periodo 2002 al 2010 en la Especialidad de Reproducción, tesorero del Grupo de Estudio en Reproducción y Pediatría de Animales de Compañía (GERPAC), Vocal de AVEPA por Madrid y coordinador de vocalías de la Junta de AVEPA. Ha realizado los capítulos de reproducción en el Manual de medicina interna de pequeños animales publicado por Improve en 2015.

Ha sido ponente en el Congreso AVEPA-SEVC realizado el 20-22 de Octubre de 2017 en Barcelona.

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ABSTRACT BODY:

Introducción. Mantenimiento de la gestación

El que se mantenga a gestación hasta el final de la misma va a depender principalmente de que se mantenga la secreción de progesterona por encima de 15ng/ml. Si realizamos la ovarioectomia durante la gestación, se va a producir reabsorción fetal y aborto dependiendo del momento en que la realicemos[1].

La prolactina se incrementa cuando empieza a disminuir la progesterona, aproximadamente en la segunda mitad del diestro, y además otro dato a tener en cuenta es que las concentraciones de prolactina son mucho más altas en perras gestantes al final del diestro, que en hembras no gestantes en ese mismo periodo[1]

El útero no parece que tenga influencia en el mantenimiento del cuerpo lúteo, pero en cambio, las prostaglandinas que se van a producir en la unión feto placentaria, son probablemente las que den lugar a la luteolisis previa al inicio del parto y que va a dar lugar al nacimiento de los cachorros[2,3].

Cambios fisiológicos durante la preñez

Durante la gestación se producen cambios de tipo fisiológico, que se pueden confundir con patologías clínicas y que van a dar lugar a cambios en los protocolos anestésicos habituales en e caso que tengamos que realizar una cesárea o administrar fármacos como por ejemplo los antibióticos.

Se va a producir una anemia normocítica normocrómica, reflejo del aumento del volumen plasmático sobretodo después de la séptima a la novena semana de gestación[1,4,5]

Un aumento de los niveles de colesterol en plasma entre la tercera y la octava semana de la gestación[1]. A partir del día 55 de gestación se incrementa la resistencia a la insulina[1] y a partir del día 56 disminuyen los niveles de calcio en sangre hasta el parto[1].

Diagnóstico de la gestación

Palpación abdominal

Podemos palpar las vesículas embrionarias en la perra a partir del día diecisiete al veintidós después de la ovulación[1], en general, el momento mas adecuado para realizar la palpación de las vesículas es entre los días 35 a 45 después de la ovulación. La palpación, si es fácil en perras de unos 20 Kg, puede ser mucho mas complicada realizarla en perras que contraen la prensa abdominal o de razas grandes, o aquellas que tengan pocos fetos o estén estos situados muy craneales.

Diagnóstico laboratorial

Se basa en la detección en el plasma de niveles altos de relaxina, la cuales producida en la placenta a partir de las 3 semanas de gestación, aproximadamente entre los días 20 a 30 de gestación[1,5], podemos encontrar falsos negativos si realizamos la prueba alrededor del día 21[1], por lo que se aconseja repetir 7 a 10 días después.

Diagnóstico radiológico

A partir del día 21 desde la ovulación, podemos detectar signos de aumento del tamaño del útero, pero hasta el día 43-46 después del pico de LH no vamos a poder detectar radiologicamente la columna vertebral y el cráneo[1,5]. Mientras que los dientes vamos a poder verlos en radiografías a partir del día 58 a 63del pico de LH[1,5], aproximadamente entre 3 a 8 días antes del parto. La radiología nos aporta datos importantes previos al parto, como pueden ser la posición de los fetos, si hay algún cachorro muerto, o so tienen posiciones distócicas, lo que nos va a permitir decidir en muchos de los casos en la conveniencia de realizar una cesárea programada.

Diagnóstico ecográfico

Hoy en día podemos considerar a la ecografía como el método más fiable para diagnosticar la gestación, pero no va a ser un método exacto para determinar el número de fetos en la perra. Nos va a permitir detectar patologías fetales como el anasarca y realizar medidas fetales como el diámetro biparietal craneal o el diámetro externo de la cavidad coriónica, que nos van a decir con bastante exactitud los días que quedan para el parto.

Si aproximadamente sobre el día 21 desde el Pico de LH podemos detectar la vesícula embrionaria[1,5], hasta el día 23-25 no podremos detectar el latido cardiaco[1,5], los movimientos fetales son detectables a partir del día 30 a 39 de gestación[1,5]y el intestino entre los días 57 a 63 después del pico de LH[15]

Cálculo de la edad gestacional

Los primeros estudios para calcular la duración de la gestación de un modo más exacto se publicaron en 2003 por la **Michelle A. Kutzler,** en los cuales se valoró que:

100% 62 a 68 días (65 +/- 3) a partir del pico de LH.

90% 63 a 67 días (65 +/-2) a partir del pico de LH

67% 64 a 66 días (65 +/-1) a partir del pico de LH

El Pico de LH lo podemos determinar cuando la progesteronemia se situa entre 1,5 - 3 ng/ml o cuando se doblan los niveles basales

Debido a que existen ocasiones en las que no siempre se conoce el día exacto de la ovulación, existen ciertos métodos por los que podemos calcular el momento gestacional; ya que su conocimiento es de elevada importancia para saber cuando se va a producir el parto y así reconocer situaciones patológicas.

Uno de los métodos más fiables de determinación de la edad gestacional en la perra es la medida ecografica de estructuras extrafetales; utilizando unas u otras en función del día aproximado de gestación en el que nos encontramos[10].

Hasta 25 días antes del parto, la medición ecográfica de los diámetros interiores de la cavidad corionica, es el método de medición ecográfica mas preciso. Este detalle se detecta desde los 45 días antes del parto, es una estructura anecoica con márgenes claramente definidos esféricos. Para realizar la medición de los dos diámetros debemos situarnos en ángulo de 90 grados desde un lado de la reacción decidual trofoblastica a la otra.

Existen ecuaciones aplicadas tanto a razas pequeñas, medianas o gigantes. La precision del calculo no varia con el sexo o el tamaño de la camada pero si por el tamaño de la raza: En razas pequeñas con ± 1 día de margen, el cálculo se cumple en un 77%; en medianas y en grandes como margen se utilizan ± 2 días y se cumple en un 88% y un 85% de los casos respectivamente[10].

Parámetro	Tamaño raza	Fórmula
Diámetros cavidad coriónica	Pequeña	Días antes del parto= (mm - 68.68)/1.53
	Mediana	Días antes del parto= (mm – 82.13)/1.8
Diámetro biparietal	Pequeña	Días antes del parto= (mm - 25.11)/0.61
	Mediana	Días antes del parto= (mm - 29.18)/0.7

Cuando el parto se va a producir en los siguentes 25 días aproximadamente, la medida más adecuada para predecir el parto es el diámetro biparietal. Se toma en la misma sección longitudinal del LCR, en este momento la cabeza fetal ya es fácil de identificar, pero debemos tener cuidado y tomar la medida cuando los huesos parietales del cráneo estén paralelos, midiendo en este momento la distancia entre ellos. La precisión en el cálculo del día del parto (± 1 día) fue del 75% y 63% en las razas pequeñas y medianas; y con ± 2 días

de margen fueron del 88% y 81% respectivamente[10].

Otros de los detalles observables por ecografía en función de la edad gestacional son[7]:

Detalles ecográficos fetales	Días a los que se visualiza
Vesícula uterinas	19 días
Detección del embrión	23 días
Latidos cardíacos fetales	24 días
Miembros	33-35 días
Ojos riñones e hígado	39-47 días
Cráneo y columna vertebral	47-49 días
Intestino	57-63 días

Ecografía de gestación a los 24 días del pico de LH

Cuidados de la hembra gestante

Es importante examinar a la perra a las 4 semanas de gestación, realizando en ese momento el primer control ecográfico para diagnosticar la gestación y la viabilidad fetal determinando la existencia del latido cardiaco. Es interesante también realizar algunos controles clínicos como urianálisis o análisis coprológicos, con objeto de detectar agentes infecciosos y parásitos.

Proporcionar una alimentación adecuada es muy importante para prevenir la toxemia en la madre, así como disponer de una paridera adecuada que evite lesiones tanto en los cachorros como en la madre y ante todo evitar en lo posible la administración de fármacos durante la gestación

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UPDATES IN DIAGNOSTIC IMAGING OF URETERAL DISEASE IN DOGS AND CATS

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ABSTRACT BODY:

UPDATES IN DIAGNOSTIC IMAGING OF URETERAL DISEASE IN DOGS AND CATS

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Diagnostic imaging is commonly used in the workup of ureteral disease in dogs and cats. The most common indications for diagnostic imaging include ureteral obstruction and congenical anomalies such as ureteral ectopia.

Ureteral obstructions are all too common problems in small animal practice, particularly in cats. Ureteral obstruction results in decreased GFR and progressive renal damage and therefore its presence should be treated as an emergency. Ultrasound and radiography have proven to be sensitive diagnostic modalities for ureteral obstruction, but in select cases contrast-enhanced CT has the advantage of superior anatomic depiction of for example retrocaval ureter location with subsequent stricture or ureteral ligation after ovariectomy. The risk of administering contrast medium with potential nephrotoxicity in already compromised patients with reduced renal function causing contrast medium excretion to be reduced and inconsistent, as well as the need for sedation or general anesthesia have to be considered. The main imaging finding in any modality for detection of ureteral obstruction is the presence of renal pelvic dilation and hydroureter with presence of intraluminal ureteral debris such as calculi, blood clots, or cellular and mucoid debris. Degree of renal pelvic distension, though not useful as a prognostic indicator, can help in determining presence of ureteral obstruction. A renal pelvic diameter of more than 13mm has been shown to definitively be associated with ureteral obstruction in one publication, whereas obstruction was likely with renal pelvic dilation over 7mm. However, ureteral obstruction cannot be excluded if the renal pelvis measures less than 7mm in diameter. Minimal pelvic and proximal ureteral dilation which tapers quickly, within 1-2cm distal to the renal pelvis has been associated with non-obstructuive disorders. Depending on the clinical presentation, in these cases ultrasound- or fluoroscopic guided pyelography can be helpful to determine presence or absence of obstruction.

Ectopic ureters are the most common ureteral anatomic anomaly and the main cause for urinary incontinence in female dogs. It can also cause incontinence in male dogs, but this is much less common given the anatomy and length of the urethra. Female dogs typically present with clinical signs of incontinence at a young age, less than a year old, whereas males tend to present a little later. Delayed onset urinary incontinence, however, can occur in female dogs with ectopic ureters as well, and a combination of ectopic ureters with urinary sphincter mechanisms incompetence may be present. Ureteral ectopia is not uncommonly associated with other anatomic anomalies. Hydronephrosis and hydroureter is also commonly associated with ectopic ureters.

Normal ureters should insert in the bladder trigone after a characteristic cranial “hook” turn in their course. Ectopic ureters most commonly present bilaterally, however the location of the orifices may differ between the left and right side. There are many variations of displaced ureteral orifices. The most common variant is the intramural type where one or both ureters insert in the urethra after tunneling in the bladder and urethral wall. This type of ectopic ureter has been reported in up to 68% of patients with ectopic ureters. Extramural ureters extend along the bladder wall without attaching themselves to or entering the bladder wall, and open directly into the urogenital tract caudal to the urinary bladder trigone. Occasionally, they may enter into the vagina or vestibule.

Several diagnostic imaging modalities may be used to diagnose ectopic ureters, and often more than one imaging modality has to be applied for a full assessment. Survey radiographs are usually low yield, but can provide some answers about kidney size and morphology, or unilateral renal agenesis. It is helpful to assess the position and shape of the urinary bladder to determine if it is in an intrapelvic position and if there are other congenital anomalies such as urachal diverticula. It is also essential to acquire survey radiographs prior to intravenous pyelography procedures in order to determine if there are excessive amounts of ingesta and fecal material present that could obscure the ureters, and if there are any mineral opacities in the urinary tract that could be obscured by contrast media.

The first method used in veterinary medicine to

investigate normal anatomy and pathology of the ureters is excretory urography or intravenous pyelography. Iodinated contrast medium is injected intravenously orthogonal radiographs of the abdomen are acquired immediately and at regular intervals, at least 3 minutes and 10 minutes after contrast medium administration. The 3-minute time interval typically captures the nephrogram phase, whereas the pyelogram phase is captured at 10 minutes. In the following, multiple projections including oblique views are usually necessary to image the entire length of the ureters. The absence of the normal curved shape of the terminal ureters has been used as a diagnostic criterion for the diagnosis of ectopic ureter and has been shown to have a sensitivity of 83% and accuracy of 76%. An issue that is difficult overcome is superimposition of the ureters with abdominal structures and the pelvis. Retrograde vaginocystography has the advantage of the catheter being placed outside the urethral lumen, and has been shown to be useful. Overall, contrast radiography allows correct diagnosis of ectopic ureters in 62-77% of cases.

Ultrasonography is commonly used for the diagnosis of ectopic ureters. Contrast radiography and ultrasound have similar results in terms of sensitivity. The main criteria used for ultrasonographic assessment of the ureteral openings into the urinary bladder is presence or absence of ureteral. Spontaneous ureteral jets, however, are often sporadic and weak, and it is time-consuming and sometimes impossible to determine if ureteral jets are present and normal. Adding diuretics such as Lasix has been shown to markedly increase strength, visibility and frequency of ureteral jets and is routinely used in the assessment of patients suspected to have ectopic ureters at our institution. Ectopic ureters are often dilated and can be followed past the bladder trigone into the urethra.

Contrast-enhanced computed tomography can be used to assess patients with suspected ureteral ectopia. Its main advantage is the fact that there is no superimposition with the pelvis. The diagnosis of ureteral ectopia is made by demonstrating the ureter either continuing past a partial opening into the urinary bladder, or completely bypassing the urinary bladder and inserting into the urethra or, vagina. Multiple image series are usually necessary to demonstrate urine flowing into the urinary bladder through the ureteral papillae, as ureteral peristalsis is often asynchronous.

An anatomic variant not uncommonly associated with ureteral ectopia is ureterocele. Ureteroceles are cystic dilations of the terminal ureter and are classified as orthotopic or ectopic. Orthotopic ureteroceles are also called intravesicular ureteroceles and are dilatations of the submucosal portion of the distal ureter. Ectopic ureteroceles on the other hand are associated with ectopic ureters and located in the bladder neck or urethra. Ureteroceles are readily identified with ultrasound as thin walled, smoothly margined fluid filled cystic structures.

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CANINE ORAL PATHOLOGY

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Introduction

Canine oral pathologies comprise a set of pathologies that can occur in isolation or together. Many of them are chronic and deserve special attention from the veterinarian and the owner of the patient. The joint work between doctor and owner is essential for the success of medical treatment. Canine oral pathologies can affect the patient from birth to old age and an accurate diagnosis is essential at all stages of the patient's life

Primary and secondary cleft palate: Congenital defects. In the first it is a failure of the mesenchyme of the lateral palatine process to fuse with the intermaxillary segment and in the second it is a failure of the palatine processes to fuse with each other. IF the animal survives the first months of life due to careful feeding by the owner, the corresponding repair surgery can be performed after 3 and a half months

Malocclusion of deciduous teeth: This occlusal disorder can generate torsion of the mouth, alteration in mandibular growth and injuries to adjacent tissues. In many cases, the therapeutic response would be interceptive orthodontics.

Impacted teeth: A condition in which a tooth fails to fully appear in the gums. The cause of impacted teeth is usually a lack of space in the bone. Surgical removal of the impacted tooth is sometimes recommended, although it may be optional, unless problems develop

Persistence of deciduous teeth: The persistence of teeth from the deciduous dentition leads to the presence of definitive dental malocclusion and difficulties in dental hygiene. Its extraction is recommended before generating definitive occlusal disorders

Dental fractures: Dental fractures usually occur with some frequency and we can classify them as non-compromised and compromised fractures. The first is when we do not have pulp exposure, and the second is when there is. Treatments for compromised fractures range from direct pulp sealing to root canal treatment.

Adamantine hypoplasia: Enamel hypoplasia is defined as a defect in the development of the hard tissues of the tooth that occurs before the eruption of the tooth as a result of a disorder in the formation of the enamel. We can appreciate it in patients who have suffered clinical or subclinical distemper or other viral, parasitic or cachexia diseases that have affected the enamel organ of the definitive pieces.

Tumors: Odontomas, cementomas, ameloblastomas can appear at an early age of the patient. early diagnosis coupled with adequate surgical treatment would be the correct therapy for these cases. Many of the tumors we see in dogs tend to be benign- these include odontogenic tumors that arise from tooth-associated tissues, such as odontomas and fibromas, as well as other types of tumors. We can also find a non-cancerous growth- Gingival Enlargement or gingival hyperplasia. But there is also a high incidence of malignant tumors such as melanoma, fibrosarcoma, squamous cell tumor, osteosarcoma and others.

Craniomandibular osteopathy: Craniomandibular osteopathy is a rare inflammatory disease of unknown etiology and characterized by bilateral, irregular, non-neoplastic bone proliferation that affects the skull bones of young dogs. It occurs mainly in the body and mandibular branches, tympanic bullae, parietal bone, occipital bone and, on some occasions, in the metaphysis of long bones. Patients with this pathology usually have difficulty eating, pain when opening the mouth, intermittent fevers, excessive salivation. In advanced cases, there is a considerable increase in the size of the jaw, a decrease in the range of movement of the temporomandibular joint, a decrease in food intake and weight loss.

Periodontal disease: Periodontal disease is by far the number one health problem in animal patients. At two years of age, 80% of dogs have some form of periodontal disease. Small and toy breed dogs are particularly susceptible. Periodontal disease is infection and inflammation of the periodontium (the tissues that surround and support the teeth) due to plaque bacteria and the host's response to the bacterial insult. Gingivitis is common in dogs and refers to inflammation of the gingiva in response to plaque antigen. Periodontitis is a more severe disease that involves inflammation of the periodontal ligament and alveolar bone, eventually causing loss of attachment (periodontal pocketing, gingival recession, bone resorption). Periodontitis is much more common in certain dog's breeds, but it can affect any individual. Despite its prevalence, periodontal disease is highly

underdiagnosed. This is due in part to lack of education, but mostly because there are few or no outward clinical signs. Therefore, the therapy usually comes very late in the course of the disease, if ever. In consequently, periodontal disease may also be the most undertreated disease in our patients.

Chronic Ulcerative Paradental Stomatitis (CUPS: Canine stomatitis is commonly described as Chronic Ulcerative Paradental Stomatitis (CUPS). Chronic ulcerative stomatitis in dogs has a greater predisposition in breeds such as the Maltese Bichon, Cavalier King Charles, Labrador Retriever, Greyhounds and Terriers. It is not as common as in cats, but it is characterized by being very painful. Although its etiopathogenesis is unknown, it is related to an abnormal response of the immune system, producing an exacerbated reaction to bacterial plaque, causing an abnormal ulcerative inflammatory response. Patients who suffer from its present halitosis, anorexia, excessive drooling, dermatitis in the folds of the lips and pain. In general, they present moderate periodontal disease.

Disorders of the salivary glands: The main disorders of the salivary glands in dogs are: Sialoceles or mucoceles, are characterized by an accumulation of saliva in the subcutaneous tissue, surrounded by mesenchymal cells without secretion capacity due to a defect in the salivary glands or duct.

Pathological bone fractures: Pathological fractures are due to a deterioration of the bone due to present pathologies that weaken or deteriorate the mandibular or maxillary bone. It is usually due to the presence of advanced periodontal disease or tumors. They can be spontaneous, trauma-induced, or iatrogenic.

ENTIENDIENDO EL COMPORTAMIENTO ANIMAL Y POR QUÉ DEBEMOS REDUCIR EL MIEDO, LA ANSIEDAD Y EL ESTRÉS

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INTRODUCCIÓN

Aunque sean términos que puedan confundirse, es importante entender las diferencias entre los conceptos de estrés, miedo, fobia y ansiedad.

Estrés

Con frecuencia, y de forma incorrecta, el término estrés se usa como sinónimo de la palabra anglosajona *distress*. Esta hace referencia a la respuesta de estrés que tiene consecuencias negativas sobre la salud física o mental del animal o las personas. Sin embargo, esto no debería ser así, ya que la respuesta de estrés es necesaria para la supervivencia de los animales. Algunos autores sugieren el uso de las palabras *eustrés* y *distrés* para diferenciar el estrés "bueno" del "malo". (En estos proceedings utilizaremos el término estrés y el contexto determinará si hace referencia al "bueno o al malo")

El estrés se define como la respuesta fisiológica de estrés se da cuando un animal se enfrenta a una situación que representa una amenaza real o potencial para su homeostasis. Desde el punto de vista fisiológico, el estrés se divide en dos grandes grupos de respuesta:

- La activación del eje hipotálamo-hipófisis-adrenal (HHA).
- La activación del sistema simpaticoadrenomedular (SAM).

En ambos casos, la activación de estas vías permite poner en marcha una serie de comportamientos que tienen como objetivo evitar la situación que está amenazando la homeostasis del animal.

Es importante mencionar que tienen como objetivo movilizar energía, nutrientes, oxígeno o potenciar el sistema inmunológico en algunos tejidos concretos, entre muchas otras funciones. De nuevo, todos estos cambios tienen como objetivo preparar al animal para hacer frente a una amenaza.

En resumen, los puntos clave de la respuesta de estrés se resumen como sigue:

- Es una respuesta fisiológica que prepara al organismo para evitar una amenaza a su homeostasis.
- La amenaza puede ser real o potencial. Significa que la respuesta de estrés puede ponerse en marcha en previsión de algo que no ha sucedido y puede que no llegue a suceder.
- Puede ser muy adaptativa, producir enfermedad o, incluso, la muerte del animal.
- El estrés es aditivo, en otras palabras, dos estímulos estresantes sumarán su efecto sobre la intensidad de la respuesta.
- La respuesta de estrés es muy inespecífica, es decir, la respuesta (cualitativamente hablando) que se pone en marcha ante estímulos potencialmente muy amenazantes y ante estímulos muy agradables (por ejemplo, la conducta sexual) es igual.
- Que la respuesta de estrés tenga consecuencias negativas o positivas sobre el animal no depende del estímulo estresante, sino de otros factores como la previsibilidad y el control (se analizarán en otro charla del congreso).

El miedo, sin embargo, es una respuesta emocional que, aunque es desagradable y negativa, es adaptativa y se produce en respuesta de una amenaza. La respuesta fisiológica que se activa ante una situación de miedo es el estrés. Como se ha comentado, el miedo es necesario y adaptativo, ya que permite al animal evitar situaciones que representan un peligro. Por el contrario, la fobia es una respuesta de miedo desproporcionada (en intensidad o duración) y, por lo tanto, limita el comportamiento normal y el bienestar del animal. Finalmente, la ansiedad es una respuesta anticipatoria, es decir, el animal pone en marcha una respuesta emocional (de miedo o fobia) ante un estímulo amenazante que todavía no ha aparecido.

El estrés modifica la frecuencia de aparición de ciertas conductas. Con independencia de si el estrés se produce como consecuencia del miedo, la fobia o la ansiedad, los cambios de conducta son los mismos y se resumen en la Tabla 1. Algunas de estas conductas que se modifican pueden representar un peligro para el animal o ser consideradas un problema de conducta por parte de los tutores.

Tabla 1. Principales cambios de conducta y salud asociados al estrés.	
Comportamiento	**Cambios**
Alimentación	Disminuye (+ frecuente) pero puede disminuir.
Acicalamiento	Disminuye (+ frecuente) pero puede disminuir.
Actividad general	Disminuye.
Juego	Disminuye.
Conducta de exploración	Disminuye.
Interacciones sociales positivas	Disminuyen.
Vocalizaciones	Aumentan.
Vigilancia	Aumenta.
Huida/esconderse	Aumenta.
Marcaje con orina	Aumenta.
Comportamiento agresivo	Aumenta.
Comportamientos compulsivos	Aumentan.

PROBLEMAS MÉDICOS ASOCIADOS AL ESTRÉS

Como se ha comentado anteriormente, el estrés se ha correlacionado en numerosas especies con problemas cardiovasvulares, dermatológicos, gastrointestinales, inmunológicos, neuroendocrinos, urinarios o reproductivos, entre otros. Los más frecuentes son:

- Problemas dermatológicos. El estrés puede inducir o exacerbar los problemas de piel. Las infecciones de piel, las recidivas de estas y la resistencia al tratamiento con antibióticos son más comunes en gatos que muestran un elevado grado de estrés. Además, existe una relación directa entre el estrés y el incremento de patologías pruriginosas (como, p. ej., la atopia). Además, esta relación entre el estrés y el prurito es bidireccional, ya que el estrés empeora cuadros atópicos, por ejemplo, pero a su vez el prurito induce más estrés en el animal. Finalmente, tal y como se ha mencionado en la Tabla 1., el acicalamiento puede verse alterado por el estrés lo que, secundariamente, afecta negativamente a la salud dermatológica del animal.
- Problemas gastrointestinales. Tanto las situaciones que generan estrés agudo como crónico pueden alterar la funcionalidad gastrointestinal por diferentes mecanismos como, por ejemplo, el aumento de la permeabilidad de la mucosa intestinal, cambios en el peristaltismo gastrointestinal o, incluso, por modificación de la *microbiota* intestinal. Además, el estrés también puede disminuir el apetito en gatos (anorexia por estrés) lo que, a su vez, puede poner en riesgo la vida del animal porque favorece la aparición de lipidosis hepática.
- Alteraciones urinarias. En los gatos, las enfermedades de las vías urinarias bajas (FLUDT – de sus siglas en inglés *Feline Low Urinary Tract Disease*) son muy comunes. Entre ellas, la cistitis intersticial felina (CIF) - en ocasiones también referida como cistitis idiopática felina, porque con frecuencia no se llega a un diagnóstico definitivo sino presuntivo) es una de las más importantes. Además, de nuevo, pone en riesgo la vida del animal por la elevada probabilidad de obstrucción uretral, especialmente en machos. Los factores que predisponen a la CIF se resumen en la Tabla 2. El estrés es uno de los más importantes y, por lo tanto, tanto en la prevención como en el tratamiento del CIF se deberá tener en cuenta el control del estrés.

Tabla 2. Principales factores que predisponen a la cististis intersticial felina.	
Factor predisponente	**¿Cómo afecta?**
Sexo	Más común en machos.
Estado reproductivo	Aunque existen algunas contradicciones, parece ser más común en machos castrados.
Nutrición	La obesidad favorece la CIF.
	La dieta húmeda disminuye la probabilidad de CIF.
	Administración *ad libitum* disminuye la probabilidad de CIF.
Juego	Disminuye.
Estrés	Numerosos estudios correlacionan el estrés como una de las causas principales de la CIF y como un factor importante para la mejora de los signos clínicos y la reducción de las recidivas.
Edad	Común en animales jóvenes y de mediana edad.

- Alteraciones inmunológicas: Si la respuesta de estrés está permanentemente activada durante un largo período de tiempo (estrés crónico) puede inducir inmunosupresión (por agotamiento de las células del sistema inmunológico, entre otros). Esta inmunosupresión se correlaciona con una mayor probabilidad a infección bacterianas (como se ha comentado anteriormente con las infecciones de piel), vírica o fúngica. Por ejemplo, el estrés parece tener un papel importante en la reactivación de algunos virus respiratorios felinos (p.ej., herpesvirus).

PROBLEMAS DE CONDUCTA ASOCIADOS AL ESTRÉS

Finalmente, desde un punto de vista clínico de la medicina del comportamiento (o etología), el estrés se ha relacionado con multitud de problemas de conducta. La agresividad, los problemas de eliminación, algunos tipos de trastornos compulsivos y el miedo, la fobia o la ansiedad serían algunos ejemplos de problemas de conducta que se asocian (bidireccionalmente) al estrés. Teniendo en cuenta que los problemas de conducta son una de las principales causas de abandono y de eutanasia de animales de compañía, poder evitarlos o tratarlos eficazmente es muy importante.

CONCLUSIONES

El estrés puede tener consecuencias muy negativas para la salud y la conducta del animal, incluso pueden producir la muerte. Conocer las consecuencias que puede tener el estrés será importante para evitar que suceda y para continuar la investigación sobre cómo ponemos evitarlas.

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HOW I INTEGRATE LASER THERAPY WITH ORTHOPEDIC SURGERY TO EXPEDITE HEALING IN BIRDS

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ABSTRACT BODY:

HOW I INTEGRATE LASER THERAPY WITH ORTHOPEDIC SURGERY TO EXPEDITE HEALEING IN BIRDS

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There has been an increase in the number of wild bird cases presented to veterinarians because of negative encounters with humans. From window strikes to vehicular traumas and gunshot wounds, there are a number of different human activities leading to an increase in orthopedic injuries in birds. The goal of the Wildlife Hospital of Louisiana (WHL), or any rehabilitation facility, is to treat avian patients and return them to the wild. This can be challenging for skeletal injuries when the damage affects the bird's function. Much like athletes, birds being released back to the wild must be able to perform at a high level. The duration a bird is in captivity is also an important consideration, as they can further injure themselves during the rehabilitation process or become imprinted (e.g., juveniles) during their care. Fortunately, many birds have an excellent capacity to heal, and if provided the best opportunity to heal will do so. Moreover, using new integrative techniques, such as photobiomodulation therapy ("laser therapy"), can potentially shorten the rehabilitation time.

Avian species presenting for orthopedic injuries need a thorough assessment. While fractures are often visible during the physical examination, other conditions, such as anemia or infection, will require additional testing. The first priority with these cases should be stabilization. Correcting any fluid or calorie deficits is key to ensure the patient can have the energy and resources to heal and process medications/anesthetics/analgesics being used to manage the case. Temporary stabilization of fractures can be accomplished with a figure-of-8 bandage for injuries of the wing that are distal to the elbow, or a figure-of-8 and body wrap for fractures in the humerus. The latter method, or a stockinet, can be used to immobilize a wing with a coracoid, clavicle, or scapular fracture. Modified Robert Jones splints or other splinting methods can be used to temporarily immobilize fractures on the legs. It can be challenging to immobilize femoral fractures because of the inguinal skin. As with domestic species, condition of the bones is key to the prognosis. Open fractures, especially those that are highly contaminated, pose special challenges. Because resources are often limited, it is important to triage these cases to increase the likelihood of having success with the case.

Differences in skeletal structure between birds and mammals does require a different approach when managing these cases. While plates and screws can be used in large birds, they are often of limited use for birds <500 grams. The cost associated with plates and screws can be prohibitive for wildlife cases. Fortunately, tie-in systems with intramedullary pins (IM) and cortical pins are an excellent strategy for bone repair in birds. Recommended fixation methods based on fracture location and type can be found in Table 1.

Fractures of the clavicle, coracoid, and scapula are typically best handled through cage rest and supportive care. In some cases, a figure-of-8 bandage and body wrap are used to immobilize the wing. In other cases, a stockinet fitted over the body is sufficient to decrease wing movement without the additional pressure on the patagium. For highly stressed birds, simply cage resting (without coaptation) the bird may be sufficient.

Fractures of the wing are common in raptors post vehicle collision or gunshot injury. Closed fractures carry a better prognosis than open fractures, but success can be had with open fractures. Stabilizing the wing and limiting soft tissue injury are key to the success and timely rehabilitation of these cases. Humeral fractures tend to be more common in larger raptors (> 1 kg) presented to the WHL, whereas radial and ulnar fractures tend to be more common in birds < 1kg. IM pin tie-in fixators are most commonly used to manage fractures of the humerus and ulna at the WHL. In both cases, a type I external fixator is used and tied-into the IM pin. IM pin diameters are typically held to approximately 50% of the bone marrow diameter to ensure cortical pins can be placed. External bars for the fixator have been made using a variety of products, including straws and Penrose drains.

Fractures of the legs typically carry a better prognosis than wing fractures because the soft tissue injuries of the legs are less likely to be devastating to the bird.

Femoral fractures are typically repaired using a type I external fixator tied-in with an IM pin. Care should be taken with pin placement in the distal femur, as there is a natural curve in the femur that can alter the direction of the IM pin. Tibiotarsal fractures and tarsometatarsal fractures are best stabilized with a type 2 external fixator; a tie in with an IM pin is typically used for tibiotarsal fractures but not with tarsometatarsal fractures.

Healing times with these fractures can vary, but significant healing is typically achieved within 4-5 weeks. However, at the WHL, integrative therapies are being combined with other medicinal therapies to speed healing and shorten this time to 2-4 weeks. Photobiomodulation, or 'laser therapy', has shown to be a successful adjunct option to traditional fracture treatment by increasing healing properties as well as decreasing inflammation. There are limited studies in birds to assess how photobiomodulation might be used to shorten fracture healing times in these animals. However, studies in other species (i.e. rats, rabbits, dogs) have demonstrated that photobiomodulation can be used to enhance healing properties and decrease the time it takes for an injury to heal. With bone, the periosteum is highly vascularized. By stimulating this area with the laser treatment, an increased blood flow will bring nutrients to aid and accelerate the healing of the fracture. There is a wide variety of conditions that photobiomodulation has been used to manage cases, including pre-surgical and post-surgical cases, as well as acute and chronic disease. Currently, we apply photobiomodulation and physical therapy three times a week to our cases, and based on this regimen, most orthopedic hardware is removed within 21 days.

Table 1. Recommended fixation methods based on fracture location and type.

Bone	Fracture type	Prognosis	Recommended fixation
Clavicle	Simple, closed	Good	Cage rest, coaptation
Coracoid	Simple, closed	Good	Cage rest, coaptation
	Severe displacement	Fair	IM-pin or plate
Scapula	Simple, closed	Good	Cage rest, coaptation
Humerus	Simple, closed	Good	IM-pin
			with type I external fixator
	Open, comminuted	Poor, guarded	IM-pin with type I external fixator
Ulna	Simple, closed	Good	Coaptation
Radius	Simple, closed	Good	Coaptation
Radius and ulna	Severe displacement	Fair	IM pin with type I external
			fixator (ulna), IM pin in radius
Metacarpus	Simple, closed	Fair	Coaptation
Phalanx (wing)	Simple, closed	Fair	Coaptation
Femur	Simple, closed	Good	IM-pin with type I External fixator
	Open, comminuted	Poor, guarded	IM-pin with type I external fixator
Tibiotarsus	Simple, closed	Good	Coaptation
	Severe displacement	Fair	IM-Pin and Tie in type II external fixator
Tarsometatarsus	Simple, closed	Good	Coaptation
	Severe displacement	Fair	Type II external fixator
Phalanx (toe)	Any	Fair	Coaptation

HOW I MANAGE WING AND LIMB INJURIES OF BIRDS INTEGRATING LIMB PERFUSION

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HOW I MANAGE WING AND LIMB INJURIES OF BIRDS INTEGRATING LIMB PERFUSION

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Wing and limb injuries are common findings in avian wildlife presented to veterinarians for traumatic injuries or infections. Because complete return to function is key to the success of these cases, it is important for us to not only repair these limbs but for the limb to function at a high level. Of course, it can be quite challenging to achieve this level of success in some cases because of the extent of the injury or infection (e.g., joint infection), so identifying novel methods to integrate into common therapeutic regimens is required to increase our success.

Regional limb perfusion is a therapeutic option commonly used in veterinary medicne, especially with large animals (e.g., cows, goats, horses). While suggested in birds, there is limited evidence for regional limb perfusion in this group. This method can be used to increase the likelihood of delivering therapeutics directly to a region of a limb based on the placement of a tourniquet. The theory behind this method is that the drug remains in a concentrated form within a specific region to increase the potential for the therapy to act directly on an infection/lesion. Of course, this method can also potentially lead to side effects for concentrated medications and thus selection of medication should be based on a case-by-case basis.

The author has primarily used antibiotics (e.g., amikacin) and analgesics (e.g., lidocaine) for regional limb perfusion in birds. The most common sites for regional limb perfusion in the author's practice are associated with the legs (e.g., joints infections, pododermatitis) of waterfowl and raptors. However, we have used regional limb perfusion for lesions in the wings too. When selecting an antibiotic for regional limb perfusion, it is highly recommended to choose an antibiotic based on a culture and antimicrobial sensitivity test.

Regional limb perfusion can be done with or without sedation/anesthesia, although the author prefers either sedation or anesthesia to minimize the stress and discomfort for the patient. There is likely some discomfort with the application of the tourniquet. For sedation, the author typically uses midazolam (0.5-1 mg/kg intramuscularly). For full anesthesia, isoflurane is used to effect and the animal is intubated to control the delivery of the inhalant. A soft pliable material or rubber band can be used for the tourniquet. The tourniquet is always placed just proximal to the region where the treatment is desired. For example, if the hock is the desired site for treatment, then the tourniquet is placed just distal to the stifle and the medial metatarsal vein is used as the site for injecting the therapeutic. The tourniquet is typically applied for 15 minutes. It is important to observe the distal limb for any changes in color or temperature (e.g., cold to the touch) during the procedure. Treatment frequency varies based on clinician preference; however, the author typically performs the procedure once daily for 3-7 days based on patient response. Pulse therapy, or alternating doses, is another option. The response rate for cases pursued in the Wildlife Hospital of Louisiana have been good, with a >60% release rate in animals treated with regional limb perfusion as an integrated component to their therapeutic plan.

Regional limb perfusion has shown good results in the author's practice; however, there remains much we still do not know about tissue concentrations and overall impact of treatment. It is important for us to document and share these results to improve our level of care for injured birds. As more evidence becomes available, it is likely that this form of treatment will become more commonplace in avian medicine.

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WSAVA ZOONOSES GUIDELINES IN DOGS AND CATS - RESPIRATORY, URINARY, AND WSAVA RECOMMENDATIONS

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ABSTRACT BODY:

Zoonotic diseases are defined as being common to, shared by, or naturally transmitted between humans and other vertebrate animals. Humans are infected with zoonotic agents from direct contact with the infected pet, contact via contaminated food or water, from shared vectors, and from the shared environment. Direct contact with animal feces (enteric zoonoses), respiratory secretions, urogenital secretions, or infected skin and exudates, as well as bites and scratches can result in human infections.

Pets that are healthy, being dewormed, and on flea and tick control are unlikely to be dangerous for their owners. Owners should bring their pet yearly to the veterinarian to assess for zoonotic risk and to appropriately provide preventative health care. Veterinarians should work closely with the family physician on pet ownership issues.

Please see other proceedings discussing other overview topics concerning dog and cat direct zoonoses and direct zoonoses from exotic pets. In this lecture, I will provide an overview of the zoonotic disease agents associated with respiratory and urinary tract diseases. In the lecture, we will emphasize how to recognize, treat, and prevent the most common agents.

Respiratory and ocular zoonoses. *Bordetella bronchiseptica* and *Chlamydla felis* cause mild respiratory disease and *C. felis* has rarely been associated with conjunctivitis in people. Most people with *Bordetella* infections are infected by *B. pertussus* but some immunocompromised people develop infection by *B. bronchiseptica*. Humans are the principal natural hosts for *Streptococcus* group A bacteria, *S. pyogenes* and *S. pneumoniae*, which cause "strep throat" in people. Dogs or cats in close contact with infected humans on rarely develop transient, subclinical colonization of pharyngeal tissues and so theoretically can transmit the infection to other humans. *Yersinia pestis* and *F. tularensis* can be transmitted from cats or dogs to people in respiratory secretions. Several influenza A viruses have been detected in dogs or cats. SARS-CoV-2 is a well documented reverse zoonosis (passed from infected owners to their pets) but only causes transient and mild infection in small companion animals. Recently, there was one case in Thailand where the owner infected their cat with SARS-CoV-2 and then the cat infected the veterinarian. Care should be taken when handling small companion animals that are permissive hosts, like cats and syrian hamsters, if the animals are from homes with owners recently exhibting signs of COVID-19.

Genital and urinary tract zoonoses. *Leptospira* spp. (dogs more than cats), *Brucella canis* (dogs), and *Coxiella burnetii* (cats more than dogs) are the most common zoonotic agents in this group. *Coxiella burnetii* is a rickettsial agent found throughout the world, including North America. Many ticks, including *Rhipicephalus sanguineus*, are naturally infected with *C. burnetii* and so this agent is also a shared vector zoonoses. It most commonly is associated with respiratory disease in infected humans that come in contact by inhaling the organism which is shed in high numbers in some cats during parturition. *Brucella canis* (dogs) is not known to infect cats. This agent is intracellular and so difficult to clear with antbiotic therapy. *Leptospira* spp. infected dogs rarely infect humans. However, the risk is real and appropriate barrier precautions should be used when handling dogs or cats with presumed leptospirosis. Vaccinated dogs exposed to Leptospira spp. are less likely than unvaccinated dogs to pass large amounts of the organism into the human environment.

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LEADING ACROSS GENERATIONS

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ABSTRACT BODY:

With four distinct generations in today's veterinary marketplace and workforce, it is little wonder veterinary managers are looking for guidance on how to create more compatible and efficient work environment.

Each of the generations in today's workforce sees the world through a unique lens, and they come with different expectations, assumptions, priorities, and approaches to work and communication.[1,2]

Baby Boomers (born between 1946 and 1964) favor collaboration, teamwork, and hierarchy, and they have a strong professional ethic that relies on commitment. They tend to arrive early, stay late, and embrace plenty of meetings. Baby Boomers tend to be motivated by leaders who get them involved and show them how they can make a difference. They prefer to be rewarded through personal appreciation, promotion, and recognition.

Generation X (born between 1965 and 1981), are independent and self-sufficient, technologically adept and well-educated. While Gen Xers tolerate regular work hours, they expect flexible work schedules for personal commitments and prefer email to conversation. They like to work autonomously. They appreciate free time, opportunities for development, and certifications to add to their resume.

Millennials (born between 1981- 1995) are multitaskers and comfortable with both group and individual interaction. They are generally adept at social media and sometimes prefer to text than call or email. Millennials embrace teamwork and like building relationships. They value work-life balance over career. They appreciate awards, certificates and tangible evidence of credibility.

Generation Z (born between 1996- 2010), is the first generation born into the digital world. They have the highest level of technological connectivity, with many spending virtually all of their waking hours connected to electronic devices. They have very short attention spans and prefer visuals to text. Having an impact on the world may matter to them more than their jobs. They like being acknowledged with personalized rewards.

Seventy-five percent of managers today say supervising multigenerational teams is difficult.[6] The generations have different perspectives on issues like work ethic, leadership, and authority. These differences, though subtle, can cause conflict, frustration, and misunderstanding if not managed well. However, those very differences can also lead to increased creativity and productivity—and ultimately to business success.

While leading such a diverse group will be challenging, there are many benefits to working in a multigenerational team, as members from different generations can offer unique perspectives and bring different strengths to the table.

The benefits of diversity in the workplace can be immense, through improvements in productivity, company culture, employee retention, and eradication of biases. Strengths of multigenerational work teams can include[2]:

- increased flexibility,
- better decision making from multi-generational perspectives,
- broader client appeal to a multi-generational market
- increased innovation and creativity.

Consider the following tips for managing a multigenerational veterinary successfully:

Initiate conversations about generations. This will increase understanding and help employees develop respect for other generations. While it's important to be aware of multigenerational differences, one should avoid automatically stereotyping a team member based on his or her age. Even though individuals fall into a certain group based on the year of their birth, they may not present the supposedly typical characteristics of their particular generational group.4

Provide management training on generational diversity. Leaders should learn about communication styles for each of the generations, study workplace motivators and de-motivators, and commit to specific leadership strategies.

Communicate preferences. Encourage your team to communicate their preferences openly. Just as there is no right or wrong work style, there is no right or wrong method of communication. Different generations may prefer one method over another.

Recognize and reward people appropriately. Do not take a one-size-fits-all approach to reward and recognition programs. Motivate team members with incentives that matter to them. Consider what inspires

certain generational groups or individuals in order to engage them.

Offer benefits suited to an employee's stage of life. All generations have different needs at different life stages. For employees with families this could mean need for generous family health coverage. Mature employees may need advice on long-term care insurance and maximizing retirement savings.

Foster cross-generational mentoring. Different generations are not only working together, but also learning from each other. Older veterinarians can help train younger veterinarians using the experience they have gained over the years. At the same time, a younger veterinarian may bring fresh knowledge and can teach older colleagues the newest and most innovative practices. Mentoring also helps individuals appreciate each other's differences and strengths. This mutual understanding can help bring these generations closer together and create a more positive and productive work environment.[5]

Working successfully with a mix of generations means offering choices to suit the needs and preferences of a diverse workforce. Every generation wants opportunities for promotion and the chance to work on challenging projects, which suggest that they all want to feel successful in their careers, regardless of life stage. Moreover, all generations share a universal desire to feel heard, respected and valued in their workplace.

Communication, humility, compassion and a deeper curiosity about the strengths and limitations of our team members and ourselves are required to help minimize any perceived generational gaps.

Instead of perpetuating an "us versus them" dynamic at work, we should honor each generation's unique contributions. Through awareness and flexibility, people of all ages can blend their diverse workstyles into more productive workplaces.

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USO RACIONAL DE ANTIBIÓTICOS EN GATOS –DONDE ESTAMOS EN 2022?

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ABSTRACT BODY:

Los antimicrobianos se encuentran entre los medicamentos más prescritos en la medicina veterinaria. Desafortunadamente, también se encuentran entre los más erróneamente utilizados.

El uso racional de los antibióticos comienza con la comprensión de que, a diferencia de otros medicamentos, en la clase de los antibióticos, cuanto a más largo plazo sea su uso, menor será su eficacia en el tiempo. El desarrollo de nuevas moléculas es más lento que la evolución de las bacterias, y la aparición de variantes resistentes es cada vez más frecuente, con cada vez menos antibióticos alternativos para usar. En resumen, el uso de antibióticos es la principal causa de resistencia bacteriana.

En medicina humana, el Centro para el Control de Enfermedades (CCE-EE.UU.) estima que el 30% de las recetas de antibióticos son innecesarias. En medicina veterinaria este número puede ser más alarmante.

Un informe de 1148 gatos que recibieron una cefalosporina de acción prolongada de tercera generación en Inglaterra mostró que solo el 0,5 % de estos animales habían sido sometidos a pruebas de cultivo y susceptibilidad bacteriana. Datos como este muestran la importancia de la estandarización, el uso de directrices y la racionalidad en la prescripción de antibióticos.

El enfoque de esta presentación estará en las situaciones de prescripción comunes en la clínica felina, trayendo las principales recomendaciones de las guías publicadas sobre el uso en enfermedades respiratorias y urinarias comunes (las condiciones de prescripción más comunes en la clínica felina).

Directrices para el uso de antibióticos en las principales enfermedades respiratorias felinas

Las principales directrices que rigen las prescripciones de antimicrobianos para enfermedades respiratorias fueron elaboradas por un grupo de expertos de la Sociedad Internacional de Enfermedades Infecciosas de Animales de Compañía (International Society for Companion Animals Infectious Diseases - ISCAID), publicadas en 2017 (Lappin et al., 2017).

Directrices para el manejo de las enfermedades del tracto respiratorio anterior

Se definen como enfermedades agudas del tracto respiratorio anterior aquellas con manifestaciones que ocurren hace menos de 10 días.

La mayoría de las enfermedades del tracto respiratorio anterior en gatos están inicialmente relacionadas con el herpesvirus felino tipo 1 y/o calicivirus. Algunos de estos gatos pueden desarrollar infecciones secundarias por bacterias oportunistas, muchas veces pertenecientes a la microbiota nasal. Sin embargo, algunas bacterias como *Mycoplasma spp.*, *Bordetella bronchispectica* y *Chlamydia felis* pueden formar parte de los agentes infecciosos causantes de los síntomas presentados.

Si se sospecha una infección bacteriana aguda en base a la observación de secreción mucopurulenta, las directrices recomiendan un período de observación sin el uso inmediato de antibióticos. Esta observación puede durar de 7 a 10 días, y los antibióticos se recomiendan solo si hay fiebre, postración o anorexia durante el período de observación.

En la selección empírica del uso de antibióticos, la doxiciclina o la amoxicilina son los antibióticos de elección, a utilizar durante 7-10 días, con reevaluación posterior.

En animales con manifestaciones crónicas del tracto respiratorio anterior, se recomienda una evaluación diagnóstica más extensa, en la diferenciación de problemas de base que predisponen a infecciones bacterianas secundarias, como neoplasias, infecciones fúngicas, cuerpos extraños y rinitis crónica secundaria a secuelas herpéticas, que son hallazgos comunes.

Directrices sobre el tratamiento antimicrobiano de las enfermedades de las vías respiratorias posteriores - bronquitis contaminada

En caso de sospecha de bronquitis con contaminación secundaria (basada en manifestaciones clínicas y hallazgos de examen radiográfico), las directrices primarias consisten en la recolección de material por traqueoscopia o lavado traqueobronquial (en casos difusos), con evaluación citológica, cultivo/antibiograma y PCR (*Mycoplasma spp.*). La presencia de inflamación neutrofílica y bacterias intracelulares en asociación con cultivo/PCR positivo sugiere bronquitis contaminada.

Mientras se espera el resultado del antibiograma (o si la enfermedad es grave y se hará uso empírico de antibióticos), se recomienda el uso de doxiciclina. Dependiendo del resultado del antibiograma, se debe continuar el uso o modificar según la sensibilidad

bacteriana. Si se obtiene una respuesta positiva dentro de los 7-10 días de tratamiento, se recomienda su uso por una semana más, después de la resolución de las manifestaciones clínicas de la enfermedad.

Directrices para el uso de antibióticos en enfermedades del tracto urinario

Las infecciones bacterianas del tracto urinario (ITU) son raras en los gatos y representan menos del 8% de las enfermedades del tracto urinario inferior en esta especie.

En primer lugar, debemos prestar atención a la importancia de la terminología y diferenciación entre cistitis esporádica (anteriormente denominada ITU simple o no complicada) y cistitis recurrente (anteriormente denominada cistitis complicada), dada su diferenciación diagnóstica y terapéutica.

La cistitis esporádica ocurre en animales sanos, con anatomía y función normal del tracto urinario, y sin comorbilidades que predispongan a ITU (menos de 3 episodios en los últimos 12 meses). La cistitis recurrente usualmente presentase en animales con comorbilidades que predisponen a la ITU, con una frecuencia superior a 3 episodios por año (o más de 2 episodios por semestre).

El diagnóstico se basa en las manifestaciones clínicas del tracto urinario inferior asociadas con evidencia de infección en el análisis de orina (piuria/leucocituria asociada con bacteriuria y hematuria). Preferiblemente, la orina debe recolectarse por cistocentesis, y antes del impulso para la prescripción empírica de antibióticos. Siempre que sea posible, se deben asociar exámenes de imagen en la evaluación de estos pacientes (especialmente en casos de cistitis recurrente).

El tratamiento debe guiarse preferentemente por antibiograma. Si no es posible la realización del antibiograma en la primera evaluación, se recomienda el uso de analgésicos, para generar comodidad a lo paciente y permitir la posterior recolección de orina. Ante la imposibilidad de recogida de la muestra o urgencia para iniciar el uso empírico de antibióticos, los fármacos de primera elección incluyen amoxicilina o sulfa/trimetoprima, mientras se espera el resultado del antibiograma.

El tiempo de uso de antibióticos en casos de cistitis esporádica es de 3 a 5 días, mientras que en casos de cistitis recurrente, de 7 a 14 días. En casos de cistitis esporádica no es necesaria la realización de cultivo y antibiograma tras finalizar el tratamiento, si el animal no presenta más síntomas. En casos de cistitis recurrente, se recomienda realizar cultivo bacteriano a los 5-7 días del inicio del tratamiento, especialmente si el animal está asintomático, para evidenciar la solución clínica y suspender el uso de antibióticos.

No es raro detectar bacteriuria asintomática (o subclínica) en gatos, sin embargo, su relevancia sigue incierta. Habitualmente, este hallazgo se produce en gatos con comorbilidades que predisponen a la ITU (ERC, diabetes mellitus, hipertiroidismo, inmunosupresión, etc.), pero estos pacientes no presentan sintomatología clínica. Para estos animales, no se recomienda el tratamiento, excepto en condiciones en las que:

- Hay sospecha de pielonefritis asociada
- Pacientes que van a ser intervenidos quirúrgicamente en las vías urinarias
- Pacientes que van a ser sometidos a procedimientos endoscópicos en vías urinarias con riesgo de hemorragia asociada
- Animales con cistitis incrustante secundaria a *Corynebacterium urealyticum*
- Pacientes en los que la vejiga urinaria es el foco sospechoso de infección extraurinaria
- Gatos diabéticos si la bacteriuria asintomática puede ser la causa del antagonismo de la insulina o cetosis

La presencia de un microorganismo multirresistente no debe afectar la decisión de tratar o no la bacteriuria asintomática.

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DISTOCIA EN LA PERRA

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Curriculum Vitae

"Licenciado en Veterinaria por la Universidad Complutense de Madrid en 1983.

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Es Acreditado por AVEPA en Reproducción y pediatría canina y felina

Realiza su actividad clínica en la Fundación Once del Perro Guía desde 1990, donde lleva la dirección del Centro de Cría de la Escuela de Perros Guía de la Once en los apartados de reproducción, neonatología y pediatría; además trabaja como Especialista en Reproducción en el Hospital Clínico Veterinario de La Universidad Alfonso X El Sabio y en la Clínica Veterinaria Plutos de Madrid.

Actualmente es Profesor de Obstetricia y Reproducción de la Universidad Alfonso X el Sabio.

Ha realizado Publicaciones en diversas revistas nacionales e internacionales destacando el Premio Científico Miguel Luera 2003 por el articulo Colposuspensión para prevenir la incontinencia urinaria en perras castradas.

Ha escrito el libro Reproducción y neonatología felina y canina publicado en el 2011

Ha coordinado y escrito el libro, Medicina Pediátrica en Pequeños Animales publicado en 2012 por Editorial Servet

Es miembro de la EVSSAR y miembro del GERPAC.

Ha sido miembro del Comité científico de AVEPA en el periodo 2002 al 2010 en la Especialidad de Reproducción, tesorero del Grupo de Estudio en Reproducción y Pediatría de Animales de Compañía (GERPAC), Vocal de AVEPA por Madrid y coordinador de vocalías de la Junta de AVEPA. Ha realizado los capítulos de reproducción en el Manual de medicina interna de pequeños animales publicado por Improve en 2015.

Ha sido ponente en el Congreso AVEPA-SEVC realizado el 20-22 de Octubre de 2017 en Barcelona

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ABSTRACT BODY:

Introducción

Podemos definir la distocia como una alteración durante el parto difícil, que altera el paso normal del feto por el canal de parto, el objetivo clínico consiste en realizar la ayuda obstétrica mas adecuada para conseguir la supervivencia del neonato y proteger la vida de la madre.

Signos clínicos indicativos de distocia en la perra.

Que hallan pasado mas de 72 días desde la primera monta o inseminación sin signos de parto o mas de 66 días desde el Pico de Lh o mas de 64 días desde la ovulación

Que no se produzca el parto dentro de las 24 horas siguientes al inicio de la fase I del parto o desde que la temperatura rectal haya disminuidos por debajo de 36,9º C

Presencia de flujo vaginal verdoso o negruzco antes de la expulsión del primer cachorros y sin signos expulsivos

Contracciones expulsivas durante mas de una hora sin expulsión del cachorro

Mas de 3 horas sin contracciones expulsivas entre fetos

Parto con expulsión de un feto muerto

Madre con signos evidentes de dolor o inquietud manifiesta

Hemorragía vaginal durante el parto

Presencia de membranas fetales que protuyen por vulva mas de 15 minutos sin expulsión

Frecuencia cardiaca fetal por debajo de 180 ppm

Muerte fetal confirmada por radiografía o ecografía

Confirmación por radiología de la presencia de fetos malformados, muy grandes o con posiciones incompatibles con su expulsión [1,2]

Diagnóstico de la Distocia:

Debemos realizar un examen clínico completo, valorando la temperatura rectal, el ritmo cardiaco, el número de respiraciones por minuto, y el grado de hidratación.

Hay que confirmar que la perra esta gestante, ya que a veces el propietario cree que lo está pero no es así. Tenemos que valorar el canal vaginal para comprobar si hay algún feto en vagina y comprobar también si existen bandas o estrechamientos en el canal vaginal.

El estudio radiológico nos dará datos del número de cachorros y sus posiciones y la ecografía nos servirá para evaluar el sufrimiento fetal o si alguno de los fetos estuviera muerto[3,4].

El ritmo cardiaco de los fetos en condiciones de

normalidad deberá estar por encima de los 200 ppm. Y además debe estar por encima del doble de las pulsaciones de la madre en el momento de la realización de la ecografía. Por debajo de 180 ppm. consideramos que hay estrés fetal claro, siendo 160 ppm. el mínimo aconsejable para realizar una cesárea de urgencia con éxito.

Causas de Distocia

Distocias de origen materno

- **Inercia uterina**

La inercia uterina es considerada como la causa más común de distocia y la podemos definir como la incapacidad del útero para contraerse.

Uno de los signos clínicos en las perras con inercia uterina primaria es la descarga vulvar de color verdoso sin expulsión posterior, este pigmento color verdoso es la uteroverdina, y es indicativo de separación placentaria dando lugar a hipoxia fetal y a la posterior muerte del feto.

- **Distocia obstructiva**

La distocia obstructiva puede producirse por varias causas: torsión uterina, ruptura uterina, hernia inguinal, fracturas de pelvis y alteraciones en los tejidos blandos de la vagina o la vulva.

- **Distocias por causas fetales**

Las principales causas de distocia de origen fetal son:

Mostruosidades fetales

Fetos demasiado grandes

Anasarca, edema congénito fetal

Muerte fetal

Malposiciones fetales

Las malposiciones fetales y los fetos de gran tamaño son causas frecuentes de distocia obstructiva de origen fetal. Dentro de las causas fetales de distocia

la mala postura fetal es una causa relativamente común de distocia obstructiva.

En nuestra experiencia clínica la madre tiene mayor dificultad en expulsar al feto en la presentación posterior, lo que puede dar lugar a un aumento en el riesgo de muerte neonatal.

TRATAMIENTO DE LA DISTOCIA

Tratamiento manual

Consiste en la manipulación y extracción cuidadosa de un cachorro que se encuentre atascado o retenido en el canal del parto y siempre que podamos resolver el problema con el mínimo riesgo para la madre. Nos podemos ayudar lubrificando el canal de parto con sustancias lubricantes como la vaselina estéril y evitando la utilización de instrumentos de tracción como los fórceps o cualquier tipo de pinza que pueda ejercer tracción y por tanto daño físico al neonato.

Tratamiento médico

No en todos los casos de distocia podemos aplicar el tratamiento médico, nos basamos en unos criterios generales para evitar bloquear el parto o tener que retrasar la cesarea[5].

Estos criterios son:

Que los fetos no presentan sufrimiento fetal y nos basamos en que los latidos cardiacos de los fetos están por encima de las 190 ppm. O sean el doble de los de la madre en el momento de realizar la ecografía

Que número de fetos que quedan en el útero no sea superior a 4, un número superior supone un riesgo para la madre y los fetos

Que el tamaño y la posición de los fetos sean compatibles con la expulsión de los fetos por el canal vaginal

Que la madre no esté en riesgo, que esté estable y en condiciones físicas de poder finalizar el parto vía vaginal.

Siempre antes de la administración de Gluconato cálcico y oxitocina debemos instaurar una fluidoterapía con fluidos glucosados del 10% al 20% a dosis de mantenimiento. Empezaremos con la fluidoterapia como mínimo 30 minutos antes de iniciar la administración de Gluconato de calcio y de oxitocina.

Cuando detectemos contracciones débiles, y si además estas se mantienen sin modificarse en el tiempo iniciamos la administración de fluidos glucosados y a continuación podemos administrar gluconato cálcico al 10% (0.465 mEq Ca++/ml) subcutáneo en una dosis de 0,22 ml/Kg. Prefiero la administración subcutánea ya que es igualmente efectiva que la intravenosa y no produce los problemas de arritmias, taquicardias y en ocasiones cuadros alérgicos que se caracterizan por inchazón de las orejas, y hocico y picores generalizados que pueden afectar al desarrollo del parto. La administración subcutánea de soluciones de calcio puede dar lugar a granulomas en el sitio de la inyección, por eso recomendamos repartir la dosis en diferentes puntos de aplicación. El calcio incrementa a los pocos minutos las contracciones uterinas y en la mayoría de las ocasiones no es necesario la aplicación de oxitocina. El Gluconato de calcio lo podemos repetir cada 4-6 horas para mantener la intensidad de las contracciones uterinas, administrarlo antes de la expulsión de cada cachorro si las contracciones son débiles o cada dos horas si no hay contracciones.

En los casos que este protocolo anterior no nos funcione, a continuación de la administración de gluconato de calcio, aproximadamente a los 30 minutos de aplicarlo podemos administrar oxitocina

(10 USP u/ml)[3] pero con unas condiciones estrictas, si las contracciones uterinas son poco frecuentes y de una baja intensidad o no se perciben y los ritmos cardíacos fetales son normales (190 - 220 latidos por minuto, o al menos dos veces el ritmo cardíaco de la madre). Dosis de oxitocina inferiores a las que aparecen en la bibliografía , pero administradas con mayor frecuencia (0.5 - 2.0 unidades por hembra vía intramuscular o subcutánea)[3], son eficaces también para mejorar la intensidad y aumentar la frecuencia de las contracciones miometriales, sin provocar efectos peligrosos para la madre, como pueden ser la torsión uterina, la rotura del cuerno uterino o a veces el bloqueo total de la contractibilidad uterina, en todos los casos se pone en riesgo la supervivencia fetal. Obtenemos mejores resultados aplicando la dosis de oxitocina a dosis bajas hasta un máximo de 3 veces por cachorro

Los posibles efectos secundarios derivados de elevadas dosis de oxitocina son la prolongada contracción del miometrio, el compromiso fetal o muerte como consecuencia de la interrupción del flujo sanguíneo umbilical, la ruptura uterina, el desprendimiento prematuro de la placenta o del feto y la vasodilatación e hipotensión materna[4,6].

La administración de glucocorticoides es de gran ayuda en el tratamiento de la distocia, en partos prolongados,grandes o si hemos tenido que manipular el canal del parto, utilizamos dexametasona a la dosis de 0,4mg/Kg vía intravenosa ya que disminuye la inflamación a nivel del cérvix y del canal del parto, aumenta también la viabilidad neonatal y contribuye a la maduración del hígado del feto. También hemos utilizado la metil prednisolona a dosis de 2 mg/Kg vía iv.

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FELINE ORAL PATHOLOGY

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ABSTRACT BODY:

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Introduction: Feline oral pathologies comprise a set of pathologies, which, like the dog, can occur in isolation or together. Feline oral pathologies deserve special attention from the veterinarian and the owner of the patient. The difficulty of the same pathologies, associated with a difficult management of the patient by the owner, means that the treatment is not as successful as expected.

Chronic gingivostomatitis: Chronic gingivostomatitis is an ulcerative process that can sometimes be proliferative, affecting all tissues that are in contact with the saliva and bacterial plaque: gingiva, alveolar mucosa, labial mucosa, sublingual mucosa, caudal mucosa, glossopalatine arch and soft palate. In mild cases, ulceration of the gingiva and alveolar mucosa will occur with or without involvement of the caudal oral mucosa, causing gingivitis, ulcerative stomatitis and caudal stomatitis (previously faucitis). In severe cases, even the labial mucosa, the soft palate and the glossopalatine arch. The clinical sign that differentiates gingivostomatitis from the disease periodontal, is the sign of inflammation around the entire piece (labial face and oral). Etiology: There is no single cause responsible for the development of gingivostomatitis. I know postulates that the origin lies in an alteration of the local immune system (IgE, IgA, IgM present in saliva) that in the presence of one or more chronic antigenic stimuli responds in an exaggerated way. Chronic antigenic stimuli present in the mouth and inducing exaggerated immune response are:

- Bacterial plaque
- Periodontal disease
- Food proteins
- Caliciviruses
- FORL

The diagnostic protocol should be directed towards the identification of Chronic antigenic stimuli:

1. Carrying out PCR of calicivirus in oral mucosa
2. Dental radiography to locate FORL or periodontal disease
3. Taking biopsies should be done in cases where there is Unilateral involvement or proliferation.
4. Bacterial culture of the glossopalatine arch in severe cases.

Treatment of mild to moderate cases:

1. Pain control through the use of Opiates (buprenorphine) and NSAIDs (long-term meloxicam)
2. Bacterial plaque control:
   a. Antibiotherapy: Clindamycin (5 mg/kg/12 hours, 3 weeks)
   b. Routine mouth cleaning and polishing.
   c. Control of bacterial plaque at home by methods manual (brushed).
   d. Use of rinses in drinking water (chlorhexidine) or directly (chlorhexidine bioadhesive gel)
3. Pieces with FORL or periodontal disease: they must be extracted.
4. New diet
5. Calicivirus: Feline Calicivirus acts as a chronic antigenic stimulus hence the positives have a worse prognosis. also explains why long-term corticosteroids do not control the condition, since increase the viral load and the local antigenic level, requiring each higher doses and for shorter periods. The administration of 100,000 units of interferon omega oral transmucosal, has been associated with a significant improvement in stomatitis lesions and a decrease in pain in cats with caudal gingivostomatitis and in severe cases refractory to dental extractions. Therefore, administration must be carried out in mild cases, moderate or severe, that are positive for Calicivirus by PCR

Treatment in severe cases:

If after this treatment protocol in mild to moderate cases, there is no positive response at 4 months, or in severe cases, the complete extraction of teeth. The complete extraction must respect the fangs if these are not affected. After the extraction, the analgesic, antibiotic and for calicivirus if it has tested positive.

Feline odontoclastic resorptive lesions (FORL): It is one of the most common conditions affecting cats. They are painful injuries that can affect each and every one of the teeth. This pathology has an incidence of 60% in cats older than 6 years. The most commonly affected teeth include the lower premolar and molar. During its process, the dental pieces suffer the erosion of its structure due to the activity of multinucleated cells

called odontoclasts. The aetiology is not known, so prevention is not possible. The process of destruction begins in the cementum and progresses to involve the dentin where it spreads along the dentinal tubules and finally involves the dentin of the crown as well as the root. The enamel eventually resorbs, or becomes so weak that it fractures, and a cavity in the crown becomes clinically evident. The lesions are progressive and diagnosis and treatment planning requires radiography.

Stage of Resorption Based on Radiographic Appearance in Cats, defined by the degree of resorption evident radiographically. One system (AVDC Nomenclature Committee 2019) is currently acknowledged and accepted.

The following transcription is part of the wsava Global Dental Guidelines

- Stage 1 (TR(Tooth resorption) 1): Mild dental hard tissue loss (cementum or cementum and enamel).
- Stage 2 (TR 2): Moderate dental hard tissue loss (cementum or cementum and enamel with loss of dentin that does not extend to the pulp cavity).
- Stage 3 (TR 3): Deep dental hard tissue loss (cementum or cementum and enamel with loss of dentin that extends to the pulp cavity); most of the tooth retains its integrity.
- Stage 4 (TR 4): Extensive dental hard tissue loss (cementum or cementum and enamel with loss of dentin that extends to the pulp cavity); most of the tooth has lost its integrity.

TR4a: Crown and root are equally affected

TR4b: Crown is more severely affected than the root

TR4c: Root is more severely affected than the crown.

- Stage 5 (TR 5): Remnants of dental hard tissue are visible only as irregular radiopacities, and gingival covering is complete.

Several types and stages of TR can coexist in the same patient. Any lesions with oral involvement require extraction, which is often complicated by significant ankyloses.

The purpose of the treatment is the relief of discomfort or pain. In most instances, extraction of the tooth, or coronal amputation, remain the preferable treatment options.

Periodontal disease: At two years of age, 70% of cats have some form of periodontal disease. Periodontal disease is infection and inflammation of the periodontium (the tissues that surround and support the teeth) due to plaque bacteria and the host's response to the bacterial insult. Periodontitis is a more severe disease that involves inflammation of the periodontal ligament and alveolar bone, eventually causing loss of attachment (periodontal pocketing, gingival recession, bone resorption).

The treatment of periodontal disease in the cat is similar to that of the dog, but we often see it associated with the two diseases described above. Making the differential diagnosis is extremely important. In felines, periodontal disease not only causes loss of support of the dental piece but can also be associated with Garre's osteomyelitis.

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SEDATION & ANESTHESIA FOR FRACTIOUS PATIENTS

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ABSTRACT BODY:

Fractious patients will need drugs for safe control. Not all of the drugs or drug dosages in this chart are licensed/approved for use in dogs and cats and not all are available in every country. However, all of the dosages in this chart are commonly used and can be referenced in the veterinary literature. A variety of drugs/protocols are available, choices should be made based on the veterinarian's experience and drug availability. Drugs are presented in alphabetical order in each category. PO= per os or oral, OTM = oral transmucosal membrane or buccal, FAS = fractious/anxious/stressed. **DOSAGES in mg/kg unless otherwise indicated.**

DRUG & DOSAGE (mg/kg – unless otherwise indicated)	ADVANTAGES	DISADVANTAGES/ CONCERNS	NOTES
Drugs to Administer at Home Before the Veterinary Appointment			
Acepromazine **Dog** & **Cat:** 0.025-0.05 OTM	Inexpensive, this is common injectable acepromazine	Effects are somewhat variable	Effects are variable, but much less variable than oral tables. See below for Chill Protocol
Benzodiazepines (all PO) **Cat:** Alprazolam 0.125-0.25 or lorazepam 0.25-0.5 mg/cat **Dog:** Alprazolam 0.02-0.1, Diazepam 0.5-0.2, Lorazepam 0.02-0.1	True anxiolytics	Can cause paradoxical excitement in some patients	Great choice for anxiety, generally not potent enough for aggressive or extreme FAS; may need extra dose night prior to hospital visit
Clonidine PO **Dog** & **Cat:** 0.01 to 0.05	Oral alpha-2 agonist	Bradycardia	Little to no information for using in this context
Dexmedetomidine gel (OTM) **Dog:** 0.01-0.04 **Cat:** 0.02 (**one dot** on the syringe barrel)	Effective for mild calming at low dosages, more sedation at higher dosages	Unlikely to be potent enough for aggressive patients unless the dose is increased	Licensed for noise phobia, not fractiousness and aggression. Not approved for, but used in, cats. Administer 30-60 minutes prior to leaving home
Diphenhydramine?			Effects are highly variable
Gabapentin PO **Dog:**10-20 (up to 40); **Cat:** 50-200 mg/cat	Wide safety margin	No major concerns. Cleared in part by the kidney so may have a more profound effect in patients with renal disease. This is more likely to occur with repeat dosing for chronic pain	Effective for calming, mild to moderate sedation, mild analgesia. Administer at least 2 hrs prior to leaving home, may need extra dose night prior to hospital visit
Trazodone PO **Dog:** 5-7 (up to 15 if aggressive); **Cat:** 50-100 mg/cat	Wide safety margin	No major concerns	Effective for calming, mild to moderate sedation. Administer at least 2 hrs prior to leaving home, may need extra dose night prior to hospital visit
Melatonin PO* Small **dogs and cats** 0.5-1 mg; med dogs 1-3 mg; large dogs 5mg	*Note dosing is mg per patient not mg/kg		Administer at least 2 hrs prior to leaving home, may need extra dose night prior to hospital visit

Chill Protocol Gabapentin PO (20-25 mg/kg **dogs** or 50-200/**cat**) and melatonin* PO (dosages listed above); Acepromazine injectable formulation (0.025-0.05 OTM **dogs & cats**)	Wide safety margin, very effective *Note melatonin dosing is mg per patient not mg/kg	No major concerns	Gabapentin & melatonin the night before & morning of (2 hours prior to leaving home) hospital visit; acepromazine 30 minutes before leaving home. *Costa RS, Karas AZ, Borns-Weil S. Clinicians Brief. May 2019.*
Sedative/Analgesic Drugs for In-Hospital Use (primarily IV or IM dosing)			
ACEPROMAZINE **Dog:** 0.01-0.03 IV or IM (up to 0.2 IM); **Cat:** 0.03- 0.05 (up to 0.2 IM); Can be used alone but best used in combination with opioids and/or other sedatives. Can use **OTM** as described in previous section	Mild to moderate sedation for several hours; can be given orally or OTM but higher doses will be required & onset of effects are slow	Not anxiolytic, analgesic or reversible; duration may be longer than desired	If anxiolysis rather than sedation is required, a benzodiazepine should be added to the protocol. No absolute contraindications but use with caution in patients with hepatic disease, clotting dysfunction, or hypotension; recent evidence proves that ace does NOT cause seizures.
ALFAXALONE **Cat and small dog**: 0.5-1.0 IM	Mild to moderate sedation for 20-40 minutes. More predictable effects if combined with an opioid or benzodiazepine	Mild dose-dependent cardiovascular & respiratory depression	Alfaxalone is an anesthetic induction drug that can be used IM for sedation. It is best used with opioids and in cats & small dogs since the injectate volume can be very large for medium-large patients.
ALPHA-2 AGONISTS *Dexmedetomidine* **For light to moderate sedation**: **Dog:**0.001-0.003 IV or 0.003-0.01 IM; **Cat:**0.001-0.005 IV or 0.005-0.015 IM; **For deeper sedation: Dog:**0.008-0.03 IV or 0.01-0.04 IM; **Cat:**0.02-0.04 IM; **OTM dosing:** **Dog:** 0.01-0.03 **Cat:** 0.015-0.04 Use low end of dosing range if used in conjunction with opioids or other sedatives, for older patients & patients with low level of fear/anxiety; Use high end of range if used alone, for younger patients and patients with higher level of fear/anxiety or aggression. *Medetomidine* Dosages are roughly double the mg/kg dexmedetomidine dosages.	Provide analgesia & sedation; effects are reversible rapid onset; titratable sedation from mild to profound; decreased stress as evidenced by decreased cortisol release. In dogs, 0.2 mg/kg OTM dexmedetomidine provided similar effects to 0.005 mg/kg IV (Dent et al. Am J Vet Res. 2019;80(10):969-975)	Cardiovascular effects including bradycardia, hypertension and increased cardiac work due to vasoconstriction; sudden, brief arousal can occur with painful stimulus – alleviated by concurrent opioid administration. Vomiting, especially if administered SQ. Excessive salivation, vomiting and bradycardia are fairly common with OTM administration.	Generally the most effective drugs for patients exhibiting moderate to profound FAS or aggression; most predictable effects when used in combination with opioids. Can reverse drug effects once procedure is complete and patient is in a calm, quiet area where restraint is possible if needed. Contraindication: do not use in patients with cardiovascular disease.

BENZODIAZEPINES *Midazolam* **Dog or Cat:** 0.1 -0.2 **IM** or IV *Diazepam* **Dog or Cat:** 0.1-0.2 IV only	Minimal to no adverse effects; enhance calming when used in combination with sedatives	Sedation is minimal; may not be effective if patient is already exhibiting FAS or aggression and paradoxical excitement can occur if used alone!	Never use alone in these patients. Use with an opioid and/or true sedative for those exhibiting FAS and/or aggression. Be cautious with reversal as it may cause sudden arousal. Generally no need to reverse effects.
OPIOIDS: Low Pain *Butorphanol* **Dog & Cat:** 0.2-0.4 IM or IV; *Buprenorphine* **Dog & Cat:** 0.02-0.03 IM or IV; 0.03-0.05 OTM (slow onset)	Opioids provide mild to potent analgesia depending on the drug & dose and have a wide safety margin; fast onset except buprenorphine (10-30 mins); reversible; many to choose from; variety of routes of administration; synergistic with sedatives	May cause vomiting, slow GI motility and some respiratory depression if used with other respiratory depressing drugs (eg, inhalants); more potent opioids may cause excitement and/or hyperthermia in cats	Combine with a sedative; with mild pain can use butorphanol or buprenorphine; with moderate to severe pain use hydromorphone, methadone, morphine or oxymorphone. No contraindications but use with caution in patients in which vomiting or slowed GI motility would be detrimental. Ref OTM methadone cat: *Ferreira et al. Am J Vet Res. 2011 Jun;72(6):764-71.*
OPIOIDS: High Pain *Hydromorphone:* **Dog:**0.1-0.2 IM, IV; **Cat:**0.1 IM, IV *Methadone:* **Dog:** 0.3-0.5 IM or IV; **Cat:** 0.3 IM or IV; **0.6 OTM** *Morphine:* **Dog:** 0.3-1.0 IM; **Cat**: 0.1-0.3 IM			
Anesthetic Drugs Any anesthetic drugs can be used if IV access is available. Listed here are drugs that can also be used IM and/or OTM.			
KETAMINE **Dog & Cat:** 1.0-2.0 IM when used in combination with a sedative may provide dissociation without anesthesia while the same dose IV will provide light anesthesia. 5.0-10.0 mg/kg IM for true anesthesia; IM is a good for route for cats but the volume at this dose may be too high for medium-large dogs **OTM Dog & Cat**: 5-10 **TILETAMINE-ZOLAZEPAM** **Dog & Cat:**1.0-2.0 IM or IV can be added to sedatives/opioids for light to moderate sedation For anesthesia WITH PREMEDS: **Dogs:**5-6 IM; 2-3 IV**Cats:** 6-8 IM; 2-3 IV **Dogs & Cats:** 5-7.5 OTM *(Cat study: Nejamkin P et al. J Feline Med Surg. 2020;22(2):108-113)*	Decrease CNS response to circulating neurotransmitters in those already exhibiting FAS and/or aggression; decrease incidence of sudden arousal to stimulus; ketamine (and maybe tiletamine) can contribute to pain relief.	Duration and/or depth may be longer and/or more profound than desired; ketamine & tiletamine are not reversible; ketamine is painful on injection; prolonged, rough recoveries are possible with tiletamine-zolazepam, especially in dogs.	This is anesthesia so patients should be monitored! There are no absolute contraindications but use with caution in patients with sympathetically driven cardiac arrhythmias and those with clinically significant hepatic or renal disease since these drugs are cleared by the liver & kidneys. For OTM, the most consistent effect is achieved if the ketamine is combined with the OTM alpha-2 agonist. Telazol can be used with or without the alpha-2 agonist with fairly consistent effects. Telazol has been used in meat-balls delivered **orally** to dogs at 20 mg/kg + 2 mg/kg acepromazine. In the same study, pentobarbital (63.2 +/- 5.1 mg/kg) was also effective in meatballs *(Ramsay & Wetzel, JAVMA 1998;213(2):240-2).*

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IMAGING THORACIC NEOPLASIA IN CATS

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ABSTRACT BODY:

Introduction

Radiography

Standard projections

Right and left lateral recumbent and ventrodorsal radiographs are adequate for most examinations of the thorax. Dorsoventral views can additionally be performed. Performing a 4-view examination would include both the ventrodorsal and dorsoventral views and this is referred to as "positional radiography". The reviewer should keep in their mind that they may need another view. Additional projections include a "humanoid" view, oblique views and horizontal beam projection, which will be discussed later. Regardless of the projection, the systematic analysis is important.

Normal Variations

Body condition

Geriatric, emaciated cats have a hyperlucent thorax, often have sternal malformations, a large lung size and hyperlucent lungs. This situation will mimic that of asthma, or hypovolemia due to the large lung size and hyperluceny. Obese cats have an increased opacity of the thorax, large deposits of fat around the heart. Subsequently, the cardiac silhouette has an unsharp border and the retraction of the lung lobes can be confused with pleural effusion. DV and VD radiographs are very helpful for differentiating pleural effusion from pericardial fat in obese cats. The lungs of obese cats will lead to an indistinct appearance of the air-filled lung and vasculature, making interpretation difficult. Fat can also appear as a mediastinal mass or pleural fluid. In these instances it is important to perform positional radiography to displace what may be fluid or to use ultrasonography to identify fat and differentiate it from fluid and masses.

Skeletal structures, shape of thorax, ribs, spine

Congenital defects such as pectus excavatum and carinatum create a compressed appearance of the thorax in the dorsoventral dimension as the sternum is generally dorsally displaced. This can affect the position of the heart which may be dorsally or laterally displaced. Geriatric cats may have mild to severe malformation of the vertebrae, ribs and sternum. Pathologic fractures of the ribs are not uncommon in cats with chronic lower airway disease and both acute and chronic rib fractures can be discovered incidentally on thoracic radiographs. An undulating shape of the sternum can also be commonly seen. Due to the flexibility of the cat's thoracic cage, large variation in inspiratory and expiratory radiographs is possible. This can also be used be diagnostically useful. For example, upper airway obstructions from the nares to the carina can result in dorsal deviation of the sternum and cranial deviation of the costal arch together with cranial displacement of the diaphragm.

Mediastinum

The central part of the thorax, the mediastinum divides the right and left halves of the pleural cavity. The heart occupies the middle part and the cranial and caudal parts are on either side. Normal variants are seen in kittens as the thymus is large and creates a soft tissue opacity on lateral images in the cranioventral thorax, often in contact with the cranial border of the heart, sometimes making its border indistinct. This can be mistaken for a mediastinal mass, sternal mass or pericardial mass. The mediastinum contains most of the intrathoracic vessels and nerves, the heart, as well as the trachea, lymph nodes and esophagus.

The esophagus is in the mediastinum and is typically not visible unless distended or if pneumomediastinum is present. Megaesophagus is diagnosed radiographically when it is dilated and either air or fluid-filled. On the VD image, the esophagus is midline and an increased air or soft tissue opacity may be recognized superimposed with the spine or adjacent to it. The air filled dilated esophagus has a thin opaque band to the left or both sides of the vertebral column in the VD image when severely dilated.

Sternal lymph nodes are located at approximately the second or third sternebrae and are generally not visible when normal in size. This is the same for the tracheobronchial nodes caudodorsal to the main stem bronchi and the cranial mediastinal nodes ventral to the trachea.

Mediastinal masses is recognized due to dorsal displacement of the trachea, caudal displacement of the heart, which can be masked by the presence of pleural effusion. Thymic lymphoma often results in a mass with effusion and any age cat can be affected. Fluid drainage, positional radiographs and ultimately ultrasonography are used to detect the masses and perform guided tissue sampling.

Lungs

The normal feline lung is very lucent, especially in the periphery and should appear fairly devoid of linear structures. Most difficulty in examining the feline lung is due to obesity or emaciation. Older emaciated cats tend to have a hyperlucent, large lung where the bronchial tree if very conspicuous and can be over interpreted as a bronchial pattern. Hazy interstitial patterns are commonly diagnosed in obese cats with normal lungs. Unfortunately, short of performing computed tomography, these cats remain difficult to assess. Important is to relate the clinical signs to the cats radiograph and create an action plan. If the animal has airway signs, then bronchoscopy is the next logical step. If the cat has no respiratory signs, the lung findings should be interpreted according to the body condition and other causes such as heart disease should be investigated if an increased respiratory rate or dyspnea is present.

Radiography of Interstitial lung disease

Primary Neoplasia

Primary pulmonary neoplasia is relatively uncommon in cats and generally has a poor prognosis. Radiographically it is typically a solitary or multiple masses, or, a disseminated lung pattern or lobar consolidation that looks like pneumonia. Adenocarcinoma may become cavitated. Bronchoalveolar cell carcinomas and squamous cell carcinoma are usually diffuse in the lung. Most pulmonary tumors are in the caudal lobes. Adenocarcinoma is reported as the predominant tumor type but shares many features with less common tumor types. Prevalence of suspected intrapulmonary metastasis was higher than in previous radiographic studies of cats with lung tumors. [1]

Metastatic neoplasia

Metastatic neoplasia generally present as multifocal small round soft tissue pulmonary nodules. In cats, lung-digit syndrome is an unusual pattern of metastasis that is seen with various types of primary lung tumors, particularly bronchial and bronchioalveolar adenocarcinoma. Tumor metastases are found at atypical sites, notably the distal phalanges of the limbs; the weight bearing digits are most frequently affected, and multiple-digit and multiple-limb involvement is common.[2]

Radiography of Mediastinal neoplasia

Tracheal neoplasia is uncommon in cats. Case reports of lymphoma, squamous cell carcinoma, and adenocarcinoma have been published. They typically efface the wall and lumen of the trachea due to a soft tissue mass and the tracheal lumen is typically narrowed.

Mediastinal masses are midline soft tissue opacities typically in the cranial thorax and cranial to the heart. These can be associated with pleural effusion. Ultrasound and CT are better suited to further assess and confirm a mediastinal vs. pulmonary origin of the mass and obtain tissue sampling. Thymoma and mediastinal lymphoma are the most common causes and ectopic thyroid carcinoma is less common. Thymic cysts also occur but require ultrasound and CT to differentiate from solid masses.

Thoracic wall

Neoplasms of the ribs lead to an extrapleural sign or mass on radiographs which is a broad-based lesion along the thoracic wall with a convex border towards the thorax. Primary bone tumors are uncommon in cats with osteosarcoma the most common. Lysis or proliferation of the rib is a common feature associated with a soft tissue mass.

Computed Tomography

CT is an important modality for assessing the thorax for numerous thoracic wall, mediastinal, and pulmonary disorders. It can be used to check for pulmonary metastasis and determine the location and extent of thoracic masses in order to develop treatment strategies. An important part of the assessment in CT that is superior to radiography is the involvement of adjacent structures such as the pleural, body wall, lung, bronchi, diaphragm, and lymph nodes. Pulmonary masses are typically solitary soft tissue or heterogenous lesions while metastatic disease is multifocal. CT allows alveolar consolidations seen radiographically to be differentiated from pulmonary masses. Mediastinal lymphadenopathy or masses are also more easily recognized on CT than radiography. Mediastinal disease such as lymphoma, ectopic thyroid carcinoma and esophageal or tracheal carcinoma can be differentiated from lung masses in the cranial thorax with CT.

Non-cardiac thoracic US

Ultrasound is a great way to complement radiography for further investigation of pleural and mediastinal disease, the thoracic wall and some pulmonary nodules or masses. Cranial mediastinal masses can be detected, and small volume effusions can be diagnosed and sampled. Cranial mediastinal masses are typically hypoechoic or heterogenous. Fluid filled thymic cysts can also be differentiated from solid masses and sampled. Lung masses are also hypoechoic with a hyperechoic rim of gas in most instances where they are growing out of the pulmonary parenchyma.

1. *Aarsvold S, Reetz JA, Reichle JK, Jones ID, Lamb CR, Evola MG, Keyerleber MA, Marolf AJ: Computed tomographic findings in 57 cats with primary pulmonary neoplasia. Veterinary radiology & ultrasound : the official journal of the American College of Veterinary Radiology and the International Veterinary Radiology Association 2015, 56:272-7.*
2. *Goldfinch N, Argyle DJ: Feline lung-digit syndrome: unusual metastatic patterns of primary lung tumours in cats. Journal of feline medicine and surgery 2012, 14:202-8.*

CONSEJOS PARA REDUCIR EL MIEDO EN NUESTROS HOSPITALES VETERINARIOS

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ABSTRACT BODY:

INTRODUCCIÓN

Un estudio del año 2011 (Volk et. al, 2011) asegura que el 37 % de los tutores de perros dicen que a sus perros no les gusta ir al veterinario y que el 26% considera que ir al veterinario es muy estresante. Un estudio anterior (Stanford, 1981) asegura que el 70% de los perros sanos no quieren entrar en el centro veterinario.

Que los perros y/o gatos tengan miedo de ir al veterinario es contraproducente por muchos motivos:

- Se pone en riesgo el bienestar del animal.
- Se dificulta el diagnóstico, tanto por el manejo como porque el estrés agudo puedo modificar algunos parámetros bioquímicos (p.e., la concentración de glucosa plasmática).
- Se pone en riesgo la salud de los trabajadores.

Asegurar un manejo *fear free* ayudará, por lo tanto, a mejorar los diagnósticos y a aumentar la probablidad de éxito del tratamiento. Además, maximizará el bienestar de los animales y de sus tutores, así como de los profesionales que deben manejarlo. Un manejo correcto del animal favorecerá el vínculo entre el animal y el médico veterinario que, a su vez, puede mejorar la confianza del cliente en el centro.

PUNTOS PREVIOS CLAVE SOBRE EL MANEJO FEAR FREE

- Es importante destacar que las medidas que se deberán adoptar no pueden ser generales para todas las especies que se visitarán en el centro veterinario. Deberá existir un protocolo de manejo individualizado por cada especie. Este tendrá en cuenta las características de cada una.
- La mayoría de recomendaciones no implican un gasto económico en infraestructura hopitalaria o clínica, sino que se limitan a mejorar el manejo del animal, su transporte u hospitalización con la finalidad de reducir el miedo y el estrés.
- Habrá tres grandes grupos de medidas:
  - Unas encaminadas a mejorar las infraestructuras del centro.
  - Otras a mejorar el manejo del animal por parte del personal.
  - Finalmente, las que tienen como objetivo enseñar al animal a que no tenga miedo o ansiedad a ir al veterinario (entrenamiento veterinario).
  - Es más efectivo que el entrenamiento veterinario se realice cuando el animal no necesita ayuda veterinaria, en otras palabras, que se haga prevención del problema.

DE CASA AL CENTRO VETERINARIO

El transporte de los animales al veterinario supone un estrés. Es importante señalar que el estrés es aditivo, en otras palabras, que si al llegar al centro el animal se ha estresado por el camino, su manejo será más dificil. En función de los diferentes estudios, entre el 23 y el 44% de los perros, aproximadamente, se estresan durante el transporte. Además, segun un estudio reciente (Herbel et. al., 2020) no parece producirse una habituación a corto o medio plazo al transportín, especialmente con los mareos (que es una experiencia muy negativa).

Se ha observado que si se habitúa a los gatos al transportin antes de acudir al veterinario se reducen los signos de estrés durante el transporte y se reduce el tiempo de consulta veterinaria (Pratsch et al., 2018). Finalmente, otro estudio (Argüelles et al., 2021) muestra que la habituación al transportín en gatos reduce a la mitad el tiempo de espera para que la sedación haga efecto y, además, se necesita menos cantidad de propofol para anestesiar el animal (en un procedimiento básico de esterilización).

Todo esto evidencia la necesidad de entrenar al animal al transporte antes de que el animal necesite asistencia veterinaria urgente.

LA LLEGADA AL CENTRO

Dos de las principales medidas a tener en cuenta son:

- En primer lugar separar a las especies que puedan ser fuente de estrés la una de la otra. Si no se disponen de diferentes espacios, se podrán crear con estructuras móviles. Además, planificar las visitas puede ser de gran utilidad para reducir la posibilidad de que coincidan diferentes especies al mismo tiempo. Preparar lo elementos de la recepción para minimizar el riesgo de estrés, por ejemplo, utilizando básculas enrasadas al suelo y cubiertas con plásticos antideslizantes.
- El manejo del personal auxiliar de la clínica es fundamental para que el animal esté relajada al entrar en la consulta. Esperar a que sea el animal el que se acerque o fomentar mediante el uso del

juego o la comida a que se acerque él serán dos medidas importantes en perros. En el caso de los gatos, lo más recomendable será mantenerlos alejados de los perros (punto 1) pero, además, que no pueda verse con los otros gatos que están en sala de espera (con la utilización de toallas que cubran las cajas transportadoras, por ejemplo).

LA CONSULTA

El manejo deberá ser diferente en función de la especie, y se presentarán durante la charla, sin embargo, será importante seguir unos puntos generales:

- Tomarse el tiempo necesario para no tener que forzar un manejo que genere estrés. Todo el tiempo que se dedique a que el animal esté a gusto en la consulta se ganará en futuras visitas.
- La utilización de comida y/o juego durante la manipulación es una estrategia recomendable, siempre que sea posible por el tipo de procedimiento que vamos a llevar a cabo.
- Se deben evitar sobresaltos durnate las consultas.
- Se minimizarán los manejos dolorosos (lubricación del termómetro antes de utilizarlo, se atemperarán los fármacos refrigerados, se realizarán manejos suaves, etc.).
- Si es necesario utilizar contenciones, deberán asegurar que no se produzcan lesiones en los animales. Durante la charla se discutirán los diferentes tipos de contención según la especie.
- La presencia de los tutores durante la consulta suele ser beneficiosa para que el animal pueda manejar mejor la situación, sin embargo, en algunos casos podría incluso ser contraproducente. Si el tutor/a está muy nervisoso, podría faciliar, especialmente en perros, el estado de nerviosismo del perro.

LA HOSPITALIZACIÓN

Durante la hospitalización, las estancias deberían cubrir las necesidades mínimas de los animales. A parte del espacio suficiente, deberían poder garantizar el descanso, entre otras necesidades básicas.

A parte de las instalaciones, el manejo también debería garantizar el descanso. Coordinar las medicaciones de todos los animales, siempre que sea posible, apagar la luz durante las horas de descanso y mantener un entorno silencioso será crucial para garantizar el descanso. Poder dormir es esencial para la recuperación de los animales y será uno de los puntos clave a tener en cuenta.

CONTENCIÓN DEL ANIMAL

En algunos casos en los que el manejo sea difícil y no pueda esperar la intervención clínica, será necesaria la contención física o química del animal. Sin embargo, cabe destacar que siempre que el animal no se deje manipular pero no sea una intervención urgente, se debería aplazar para poder empezar un proceso de habitaución al manejo previo a la nueva visita.

- Contención física. Debería utilizarse solo en casos extremos ya que no garantiza la ausencia de estrés severo durante el manejo.
- Contención química. Exiten muchos fármacos que pueden ayudar a que la visita sea más tranquila y eficiente. De hecho, muchos de estos fármacos pueden administrase ya en casa previo al transporte. Los fármacos más usados son los ansiolíticos a corto plazo como las benzodiacepinas, la trazodona, la gabapentina y/o alpha2-agonistas, entre otros.

EL REGRESO A CASA

En el caso de los perros no suele ser problemática, todo lo contrario. Sin embargo, en el caso en que convivan dos gatos y uno haya tenido que ser hospitalizado, es probable que la vuelta pueda conllevar problemas de convivencia.

Se deberá tener especial cuidado en realizar una reintroducción progresiva del gato que estuvo hospitalizado. Aunque nunca hubieran tenido problemas de agresividad en el pasado, la recomendación es que la reintroducción sea paulatina.

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TUMORES TESTICULARESCUÁNTO SABEMOS?

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ABSTRACT BODY:

Tumores testiculares en el perro

¿Cuánto sabemos?

RESUMEN:

El presente estudio retrospectivo se realizó en la ciudad de Trujillo durante los años 2014 al 2021, con el objetivo fue reportar la incidencia de tumores testiculares, donde se evaluaron andrológicamente y ecográficamente a 1033 perros machos, mayores de 6 años, clínicamente sanos, de diversas razas, condiciones corporales, no castrados incluyendo criptorquidios.

El examen andrológico detecto únicamente el 1.74 % (18 perros) quienes mostraron alteraciones a la palpación testicular, posteriormente con la finalidad de corroborar la presencia de alteraciones del parénquima testicular se usó la ecografía en modo B asociada a la inspección Doppler color, pudiéndose determinar que el 14.8 % (153 perros) presentaron neoplasias testiculares.

De los 153 perros con tumores testiculares un 81.70 % (125 perros) no fue criptorquidío y 18.30 % (28 perros) presentaron criptorquidismo. De éstos últimos, 13.73 % (21 perros) presentaron retención testicular derecha y 4.57 % (7 perros) retención testicular izquierda, 12.42 % (19 perros) retención en canal inguinal y 5.88 % (9 perros) retención intra abdominal. Respecto a la contextura corporal predominante fue la obesidad representada con un 49.01 % (75 perros), y las razas grandes fueron las más afectadas 35.29 % (54 perros), tanto en no criptorquidios y criptorquidios, las neoplasias testiculares se ubicaron predominantemente en el lado derecho, con un 64 % (80 perros) y 50 % (14 perros).

La ecografía en modo B, mostró que las lesiones testiculares son principalmente de tamaño mediano e hipoecoicas tanto para perros no criptorquidios y criptorquidios.

La señal Doppler color: principalmente fue negativa en un 60 % (75 perros) y 60.71 % (17 perros) de los perros no criptorquidios y criptorquidios. Respecto a la ecogenicidad y la señal Doppler, ésta estuvo presente principalmente en lesiones hipoecoicas y de mediano tamaño, finalmente los testículos del lado derecho con tumores presentaron un predominio de señal Doppler color en ambos grupos.

La principal raza afectada fueron los mestizos con 47.71 % (73 perros), bóxer 9.15 % (14 perros), schnauzer 7.19 % (11 perros), según tamaño los perros fueron: pequeños un 14.38 % (22 perros), medianos un 50.33 % (77 perros) y grandes un 35.29 % (54 perros).

INTRODUCCIÓN

En la práctica clínica veterinaria de perros, los médicos veterinarios no incluimos como rutina el examen clínico del aparato reproductor del macho, por ende las diversas patologías que afectan principalmente a los testículos no están adecuadamente reportadas, sabiendo que las patologías tumorales testiculares se incrementa con la edad en perros con descenso testicular normal; en perros con patologías del descenso testicular, éstas suelen presentarse más tempranamente y la incidencia es mayor (1,2).

De las diversas técnicas para evaluar los testículos de los perros, el estudio ultrasonográfico es la técnica de elección; y las más ampliamente usada, debido a su alta sensibilidad para determinar la presencia o ausencia de lesiones, así mismo, caracterizar la arquitectura del tejido testicular (1,3), donde el modo B, ha reportado más datos, actualmente asociado a las técnicas Doppler color se ha incrementado la información en el examen testicular (3).

Características ecográficas del testículo fisiológico

Se describen a los testículos como estructuras ovoides, localizadas en el escroto, de granularidad fina, ecogénicamente intermedios y homogéneos, rodeados por una estructura muy fina y ecogénica, la túnica albugínea; al ser explorados sagitalmente y transversalmente se aprecia en el centro de su parénquima una línea hiperecoica o un punto ecogénica que corresponde al mediastinum testis, el volumen y tamaño tienen variaciones proporcionales al tamaño y peso del perro(4).

Alteraciones en el descenso testicular

La ausencia de un testículo en posición escrotal se denomina criptorquidia, trastorno relativamente frecuente en perros, cuya localización principal es el

canal inguinal y menos intraabdominal, la presentación unilateral es más común, afectando principalmente al testículo derecho. El criptorquidismo incrementa altamente las probabilidades de que el testículo retenido tenga probabilidad de ser neoplásico, donde los Sertolinomas son frecuentes en testículos abdominales, sin embargo, en las localizaciones inguinales la predisposición es al desarrollo de seminomas (5).

Caracterización ecográfica de las alteraciones neoplásicas testiculares

A pesar de la identificar lesiones macroscópicas, la ecografía tiene como limitante de no poder diferenciar y clasificar: neoplasias de procesos, inflamatorios, infecciones entre otros; debido a que los cambios ecográficos de las patologías mencionadas no son lo suficientemente robustas como para poder emitir un diagnóstico definitivo siendo la histopatología la encargada de clasificar y nombrar las lesiones, convirtiéndose (3,6).

Los principales tumores testiculares reportados son los Sertolinomas, tumores de células de Leydig y seminomas, éstos dos últimos tienen potencial productor de hormonas y por ende pueden generar mayor probabilidad de complicaciones como metástasis. Los tumores testiculares alteran la ecoarquitectura fisiológica, presentando formas variables, pudiendo apreciarse desde pequeñas lesiones nodulares bien circunscritos hasta grandes masas homogéneas o no (4).

Diversos autores reportan que la mayor incidencia y prevalencia de patologías del aparato reproductor son las neoplasias testiculares, su presentación se ve incrementada en perros viejos (2,7), ocupando un segundo lugar en los machos no castrados (8).

Se reportó un dato importante, mencionando que lesiones con un diámetro mayor a 0.3 cm tenían mayor robustes en sospechas de tumoración y las menores a esta medida serían lesiones del parénquima, por lo que recomendaban tener en cuenta esta medida al momento de evaluar ecográficamente testículos. Finalmente concluyen que des las enfermedades neoplásicas diagnosticadas en perros machos los tumores testiculares representan un 17. 6% (8).

RESULTADOS

De los 153 perros con neoplasias testiculares, el 81.70 % (125 perros) fueron no criptorquidios y un 18.30 % (28 perros) presentaron criptorquidismo.

Ubicación de las neoplasias testiculares

De los 125 perros no criptoruidios afectados, el 64.0 % (80 perros) tuvo la neoplasia en el testículo derecho, el 30.4 % (38 perros) en el testículo izquierdo y un 5.6 % (7 perros) tuvieron neoplasias en ambos testículos.

De los 28 perros criptorquidios afectados, el 50 % (14 perros) presentaron la neoplasia en el testículo derecho, el 28.57 % (8 perros) presentaron la neoplasia en el testículo izquierdo, ninguno en el canal inguinal y el 21.43 % (6 perros) presentaron neoplasia intra abdominal.

Tabla 1. Relaciones entre señal Doppler color, diámetro, ecogenicidad y ubicación de neoplasias testiculares en perros con neoplasias testiculares

		Diametro			Ecogenicidad				Ubicación de la neo			
	Doppler	Pequeña	Mediana	Grande	Hipoecoica	Ecogénica	Isoecoica	Heterogénea	Derecho	Izquierdo	Bilat	Intra abd
No criptoquidios	Positiva	4	31	15	35	3	8	4	31	17	2	0
	Negativa	41	33	1	7	5	61	2	49	21	5	0
Criptorquidios	Positiva	1	8	0	10	1	0	0	6	4	0	1
	Negativa	8	7	4	3	2	11	1	8	4	0	5

Recomendaciones

Es necesario implementar como parte del examen clínico en perros macho la inspección del aparato reproductor.

Se recomienda el uso de la ecografia en modo B y asociada al Doppler color como herramienta importante, facil y económica para la detección o descarte de tumores testiculares en perros.

En estuido más amplios se recomienda relacionar los hallazgos ecográficos de neoplasias testiculares con la histología o inmunohistoquímica con la finalidad de mejorar el diagnóstico.

Crear un banco de reportes de neoplasias en perros con la finalidad de obtener datas de incidencias y prevalencias de las mismas.

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HOW I TREAT CANCER WITH PALLADIA® (TOCERANIB PHOSPHATE)

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ABSTRACT BODY:

What is Palladia®

Toceranib is an oral anticancer agent. Also known as a small molecule inhibitor, tyrosine kinase inhibitor or a signal transduction inhibitor, toceranib phosphate was the first anticancer drug approved for dogs.

How does Palladia® work?

Toceranib has multiple mechanisms of action. It blocks a tyrosine kinase called KIT, important in the progression of mast cell tumors and possibly gastrointestinal stromal tumor or GIST. It is also has anti-angiogenic properties through inhibition of VEGFR-2 [vascular endothelial growth factor receptor 2] and PDGFR-◘ [platelet derived growth factor receptor ◘]

What tumors does Palladia® treat?

Toceranib is approved for use in dogs with mast cell tumors, but oncologists are using it in cats and for tumors other than mast cell tumors, such as anal gland adenocarcinoma, thyroid carcinoma, oral squamous cell carcinoma, pancreatic adenocarcinoma, insulinoma, mammary gland tumors, pulmonary carcinoma and gastrointestinal stromal tumors or GIST. It has rapidly become an important drug in veterinary oncology.

How do I dose Palladia®?

Most veterinary oncologists are successfully using toceranib at approximately 2.5 - 2.7 mg/kg PO every other day or Monday, Wednesday, Friday. This dose is less than the label dose of 3.25 mg/kg EOD, but is still an efficacious dose with lower toxicity.

What precautions should owners take when administering Palladia®?

When dispensed to owners for home administration, owners should not crush or dissolve tablets in water, should wear gloves and wash their hands after administration. Small children and pregnant women should avoid any contact with the drug.

What are the adverse events associated with Palladia® administration?

Toceranib can cause a mild neutropenia, but treatment is not typically delayed because of neutropenia. The neutrophil count will rebound spontaneously even without discontinuation of toceranib. Because toceranib is administered on a continuous basis, it should be discontinued at the first sign of an adverse event such as loss of appetite, vomiting or diarrhea. If the drug is not discontinued, gastrointestinal side effects can be serious. Toceranib can cause a protein losing nephropathy and hypertension. Pancreatitis is an uncommon side effect of toceranib. The medication comes with an owner information sheet outlining side effects which should be sent home when toceranib is dispensed.

How do I monitor patients receiving Palladia®?

My monitoring plan is to schedule the initial recheck 2 weeks after starting toceranib if the pet is very ill. For pets with minimal clinical signs, I may not recheck the pet until 1 month after starting toceranib. At the two week visit, I check weight, blood pressure and confirm the owner is able to medicate their pet. For monthly visits, I check a CBC, biochemical profile, urinalysis, urine protein to creatinine ratio and blood pressure. If adverse events occur, dosage can be decreased as low 2.4 mg/kg MWF which has been shown to retain efficacy. For pets with stable clinical status, I may recheck every 6 weeks.

How do I get Palladia®?

Toceranib is manufactured and marketed by Zoetis. Some internet pharmacies stock toceranib, but beware of counterfeit medications. Tablet sizes are 10, 15 and 50 mg. Toceranib loses potency when made into a liquid. It can be compounded into smaller capsules from larger tablets in a biological safety cabinet. Owners and veterinarians should not split or crush tablets, but have a specialized compounding pharmacy make smaller dose units if necessary.

WSAVA ZOONOSES GUIDELINES IN EXOTIC ANIMALS

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ABSTRACT BODY:

Exotic pet is regularly defined as unusual pets other than dogs, cats, and livestock. There is an increasing trend towards keeping exotic pet such as small mammals, reptiles, and birds. Approximately one third of the pets in the US and Europe are exotic animals[1]. Although most of them are unique and endearing, some of them can carry and transmit zoonotic pathogens[2]. Zoonotic pathogens should be considered when dealing with these exotic pets. The number of emerging diseases is increasing and 75% of known emerging infectious diseases are zoonoses[3]. Numerous factors involve in the increasing number of emerging zoonotic diseases, mainly affecting human-animal interface such as urbanization, climate change, trends towards owning pets and activities related to animals[4,5,6,7]. Therefore, it is vital to apply a 'One Health' approach to zoonotic diseases to improve the health of animals, humans, and environment[8,9]. Veterinarians treating exotic pets should concern the potential risks of zoonoses. In addition, veterinarians as one of the front-liners in one health discipline should also dedicate themselves to public health by preventing zoonotic diseases, educating staffs and pet owners, as well as focusing on treating individuals. With recognition the importance of zoonotic diseases from exotic pets, WSAVA one health committee is going to publish the zoonoses guidelines in exotic pets in order to minimize the risks from these diseases.

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A DIVERSITY, EQUITY, AND INCLUSION (DEI) APPROACH TO ENHANCING VETERINARY WELLBEING

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Since then, Debbie has held various positions within industry, wherein she developed a fully accredited, evidencebased veterinary continuing education program called The Social Side of Practice. This program specializes in the areas of veterinary wellness, communication, teamwork, organizational culture, and leadership. She has given over 400 presentations internationally and published close to 30 articles in veterinary and social work journals as well as two book chapters. She volunteers with a variety of organizations, from local charities and non-profits to international professional associations. Debbie is a caregiver, healer, and teacher by nature, with a passion for supporting the health and welfare of people and animals.

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ABSTRACT BODY:

A DIVERSITY, EQUITY, AND INCLUSION (DEI) APPROACH TO ENHANCING VETERINARY WELLBEING

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Introduction

Diversity refers to the presence of difference – differences in race, gender, socioeconomic status, sexual orientations, religion, ethnicity, national origin, and abilities, among infinite other attributes. These differences can be visible or invisible, and since they are most often measurable, and thus quantifiable, diversity is considered *a fact*.

While diversity is about differences, inclusion is about embracing those differences. Inclusion refers to the deliberate actions we take to create spaces and places where differences are welcomed, accepted, and appreciated. Inclusion, thus, is *an action*.

Equity refers to the policies, practices, and processes that ensure that each person has access to the same opportunities. Since we choose whether we create equitable systems, equity is *a choice*.

The *outcome* of diversity, equity, and inclusion is belonging. Belonging refers to the fundamental human need to be an accepted member of a community.

The strong psychological need to belong is deeply rooted in our evolutionary history. Since living in groups is what enabled our survival, belonging is hardwired in our DNA. As social animals, just as we need food and water to survive, we also need to feel that we belong. It is part of our inherent desire to be a part of something greater than ourselves and is connected to purpose and meaning.

When the need to belong is not met, it can set off a "neural alarm system." Think of how you felt the last time you felt slighted, overlooked, or outright rejected. Research has shown that the pain of social exclusion is very similar to the pain of physical injury; it actually follows the same neural and physiological patterns as physical pain. When the need to belong *is* met, however, people feel safe and secure as well as good about themselves, others, and the world in general. In fact, with belonging, people are healthier and happier.

The Importance of Belonging

Studies show that belonging is key to health and wellbeing. Belonging reduces stress, anxiety, and depression; and is associated with higher levels of physical activity, lower rates of sickness and disease, and lower levels of drug, alcohol, and tobacco use, all of which enhance health and wellbeing. Belonging enables the ability to reach one's highest potential (i.e., to self-actualize) and inspires participation and engagement. In the workplace, these improve job performance and satisfaction, which further enhance health and wellbeing.

The benefits of belonging are as important in the veterinary workplace as any other environment. This article will focus on the imperative of creating a culture of belonging within the veterinary environment, whether a private or corporate veterinary practice, a shelter or humane society, or a veterinary or vet technician/technologist/nursing college.

How to Create a Culture of Belonging

- Seek Knowledge – Become Informed

Although none of us know everything about diversity,

equity, inclusion, and belonging, we can stay committed to learning new things every day. Learn about these terms. Each term carries powerful content for us to understand.

Familiarize yourself with the challenges that people of difference face (e.g., in relation to color, minority religious traditions, abilities, gender/sexual identity (LGBTQ), and immigrant status) in the workplace. Don't ignore or dismiss the challenges. Become sensitive to them. With knowledge comes the power to make a difference.

Commit to learning about oppression. What's in the news, social media, movies, and books? Oppression is ubiquitous, woven within our systems, making it seemingly invisible. Set your intention to see it for what it is and how it limits and marginalizes people.

Be deeply curious about others. Show interest in who they are, what they have to say, and what they bring to the table. Embrace "generous listening." This is listening to understand, not to judge. It's listening only to find out what is true for the other person, knowing that what the person is saying *tells you about them* – that it's a reflection of their worldview.

- Be Aware of Unconscious/Implicit Bias

We all have biases. They're normal. They're sourced from the shortcuts our brains naturally use to navigate the vast amount of information in the world, and they lead us to make snap decisions about who we prefer to be with and who we would like to avoid. They can, however, lead to discriminatory beliefs and practices. They can be misleading.

Some biases are conscious, but many are unconscious. Unconscious or implicit bias refers to the attitudes and stereotypes that affect our understanding, decisions, and actions without our awareness. We have unconscious biases that have been with us since childhood, absorbed through our social environments. They can be very ingrained. We may be tempted to deny that we have them, but we need to acknowledge them. Once we do, we can have some ownership over them.

Become more aware of your biases. This requires being willing to look at yourself, reflect on yourself, and ask questions of yourself – to see the truth of who you are and what you believe. Then set the intention to adopt more inclusive attitudes and behaviours. Practice putting your assumptions and stereotypes aside. If you find yourself, either by a comment or action, subtly expressing a prejudiced attitude, catch yourself and apologize. We're all learning.

- Commit to Cultural Humility

Culture plays an integral role in everyone's life. Cultural humility is being willing and able to look at the culture of another nearly through *their* eyes, opening yourself to their culture to recognize aspects of their cultural identity that's important to them, from *their* way of seeing it. Cultural humility requires a willingness to learn from others *from their perspective*.

Practice open-mindedness and self-reflection. We typically think our culture is the norm, so to really learn about another's culture requires taking their culture in and balancing it in turn with our own through self-reflection so our own culture is not so dominant, *not* the norm. Honour the beliefs, customs, and values of others. Acknowledge the differences and accept people for who they are.

- Share Experiences and Stories

The sharing of experiences and stories creates social bonds, and these are vital to fostering a sense of belonging. Team building activities are not a waste of time. Encourage them. They are a unique way of helping people see each other with new eyes. Encourage storytelling too. Stories reflect the diversity of our lives. It may require vulnerability, but when people show vulnerability, they allow *stronger* bonds to be formed. With stories, we inspire others, humanize the workplace, and make space for others to tell theirs. Recognize, appreciate, and value the differences in them, avoid making assumptions because of those differences, and be patient as you work across them. Try to put yourself in the other person's shoes, especially if a differing viewpoint rubs you the wrong way. Consider what the experience might be like for the other person. Remember that our differences add value to the workplace and our lives in innumerable ways. Your diversity matters, and the diversity of others matters as well.

- Treat People with Respect

Most of us are familiar with *The Golden Rule*: "Do unto others as you would have them do unto you." This rule implies the assumption that other people would like to be treated the way you would like to be treated. The alternative is *The Platinum Rule.* Both rules attempt to teach lessons of mutual respect and kindness. However, the latter acknowledges that we are all different, and therefore prefer to be treated differently. Practice *The Platinum Rule:* "Do unto others in the way they wish us to do unto them." Treat others how they would like to be treated. "Everyone is you, living a different life. Treat them as such."

- Be an Ally

Be an ally. Be authentic, brave, and responsible. Allies work to end discrimination, oppression, and prejudice against those who are marginalized. They lift

others up by advocating, leveraging their power and privilege to amplify the voices and work of those who are underrepresented. They state their commitment authentically, bravely challenge the status quo, and take personal responsibility for change. They create a welcoming environment for people with less-privileged identities and backgrounds. And remember that learning about discrimination, oppression, and prejudice is *your* responsibility – not that of the other person. Expecting otherwise is adding to their oppression.

Summary

Now is the time to spread the word about diversity, equity, inclusion and belonging. These are more than trendy words; they're here to stay, to push us to look at people with fresh eyes and embrace the diversity each of us brings to the workplace. Creating a culture of belonging in our workplaces is everyone's responsibility. I hope this will help you to create more diverse, positive and welcoming spaces, wherever you are, whoever you are, and whatever you do.

References available on request.

ACTUALIZACIÓN SOBRE EL TRATAMIENTO DE LAS COLELITIASIS EN GATOS

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ABSTRACT BODY:

Las enfermedades inflamatorias de las vías biliares se encuentran entre las enfermedades hepáticas más comunes en los gatos. Hay tres formas principales de colangitis reconocidas por el comité de estandarización de la Asociación Mundial de Veterinarios de Pequeños Animales (WSAVA) en gatos: colangitis crónica neutrofílica, linfocítica y asociada a parásitos.

Aunque se encuentran entre las causas más comunes de enfermedad hepatobiliar en felinos, existen pocos informes clínicos e investigaciones sobre colangitis en la espécie felina, con menos de 150 casos con una descripción más detallada.

Recientemente, una investigación presentada y publicada por el Dr. Sharon Center (Center et al., 2022) trajo una nueva perspectiva al estudio y evaluación de gatos con colangitis supurativa/colangiohepatitis, en un estudio prospectivo de tres décadas con 168 casos.

Este estudio trae datos muy interesantes sobre la fisiopatología y comorbilidades asociadas a la colangitis supurativa, donde el 43% de los animales (n=71) presentaban colelitiasis.

De los 71 gatos con colelitiasis, 20 (28%) tenían diagnóstico histopatológico de pancreatitis; 4 de ellos con diabetes mellitus asociada. Dieciséis gatos con colelitiasis presentaron debilidad aguda e ictericia secundaria a obstrucción de las vías biliares extrahepáticas.

Los estudios de imagen (ultrasonidos o radiografías) detectaron colelito(s) en solo el 58% de los gatos con colelitiasis, generalmente afectando el conducto biliar común, los conductos hepáticos o la vesícula biliar. Doce gatos solo tenían colelitos descubiertos en la intervención quirúrgica, 7 detectados por lavado retrógrado del conducto biliar común a través de la duodenostomía.

Algunos animales tenían cálculos alojados en la región de la ampolla (unión de los conductos biliares común y pancreático), causando impactación y obstrucción de ambos sistemas ductales. En 20 de los 55 gatos derivados al hospital que recogió los datos para el trabajo, hubo sospecha de "pancreatitis idiopática". En estos se observó obstrucción transitoria de la papila duodenal por microcolelitos, con diagnóstico histológico de pancreatitis asociada.

Alrededor del 71% de las muestras tenían cultivo bacteriano positivo, con más del 80% de estos compuestos por grampositivos, más del 50% de gramnegativos y un 35% con infección polimicrobiana. Utilizando marcadores inmunohistoquímicos como LTA (detecta material de la pared celular de bacterias grampositivas) y TLR4 (detecta la exposición a endotoxinas secretadas por gramnegativos), la positividad de detectar implicación bacteriana se eleva a casi el 90%.

Se concluyó que la colelitiasis está íntimamente asociada a procesos infecciosos de las vías biliares, con especial atención a los cambios inflamatorios de la vesícula biliar (colecistitis, con edema y hiperplasia mucosa), que son algunos de los factores incitadores para la formación de colelitos.

Manejo de la colelitiasis asintomática

El hallazgo incidental de un colelito en un animal sin síntomas o variaciones en las enzimas hepáticas no es infrecuente. Aunque asintomático, la presencia del colelito indica que en algún momento hubo/hay una alteración en las vías biliares.

En animales completamente asintomáticos, la intervención quirúrgica aún no está indicada. Es recomendado un control periódico con examen de ultrasonido y medición de enzimas hepáticas. En presencia de manifestaciones características, o ante la sospecha de pancreatitis "idiopática", el animal debe considerarse sintomático.

Manejo de la colelitiasis sintomática

Las principales manifestaciones clínicas de los gatos con colelitiasis incluyen vómitos, hiporexia, pérdida de peso, letargo, aumento de las enzimas hepáticas, ictericia y malestar abdominales (que pueden ser posprandiales).

En pacientes sintomáticos está indicada la intervención quirúrgica, siendo la colecistectomía la maniobra de elección (siempre que exista viabilidad de la papila duodenal y del colédoco). La extirpación de la vesícula biliar es prudente siempre que sea posible, ya que los colelitos generan hiperplasia e inflamación de la mucosa, lo que hace que el ambiente sea susceptible al desarrollo de nuevos colelitos. Los animales con colelitiasis sintomática que se someten a colecistectomía muestran tiempos de supervivencia más prolongados en comparación con los animales que no se someten a colecistectomía.

En animales que no presenten viabilidad del colédoco y

papila duodenal está indicada la colecistoenterostomía, no implicando la realización de este procedimiento un peor pronóstico o menor tiempo de supervivencia.

El envío asociado de muestras de bilis, colelito macerado, fragmento de pared de vesícula biliar y fragmento de hígado, en el mismo medio de cultivo, aumenta las posibilidades de detección de agentes infecciosos, orientando la terapia antimicrobiana postoperatoria.

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LA CESAREA

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Curriculum Vitae

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Ha realizado Publicaciones en diversas revistas nacionales e internacionales destacando el Premio Científico Miguel Luera 2003 por el articulo Colposuspensión para prevenir la incontinencia urinaria en perras castradas.

Ha escrito el libro Reproducción y neonatología felina y canina publicado en el 2011

Ha coordinado y escrito el libro, Medicina Pediátrica en Pequeños Animales publicado en 2012 por Editorial Servet

Es miembro de la EVSSAR y miembro del GERPAC.

Ha sido miembro del Comité científico de AVEPA en el periodo 2002 al 2010 en la Especialidad de Reproducción, tesorero del Grupo de Estudio en Reproducción y Pediatría de Animales de Compañía (GERPAC), Vocal de AVEPA por Madrid y coordinador de vocalías de la Junta de AVEPA. Ha realizado los capítulos de reproducción en el Manual de medicina interna de pequeños animales publicado por Improve en 2015.

Ha sido ponente en el Congreso AVEPA-SEVC realizado el 20-22 de Octubre de 2017 en Barcelona.

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ABSTRACT BODY:

Introducción.

Actualmente se establece que entre el 60- 80% de los casos de distocia en la perra y en la gata terminan en cesárea, teniendo dentro de estos porcentajes más casos de cesárea en la gata que en la perra.

Cuanto menos tiempo transcurra entre el comienzo de la distocia y la cesárea, mayor será la supervivencia neonatal y materna.

Cuando está indicado realizar una cesárea

- Ritmo cardiaco fetal (Valoración del estrés fetal). Un ritmo cardíaco fetal inferior a 150 latidos por minuto lo debemos valorar como una emergencia debiendo realizar la cesárea lo antes posible. Si la frecuencia fetal está entre 150 y 170 latidos por minuto, esto nos indica la existencia de estrés fetal de severo a moderado, mientras que valores por encima de 180 latidos por minuto los podemos considerar normales[1]
- Inercia uterina primaria completa que no responde al tratamiento médico
- Inercia uterina primaria parcial que no responde al tratamiento
- Inercia uterina secundaria que no puede resolverse por manipulación.
- Anormalidades de la pelvis materna o de los tejidos blandos del canal del parto(Complejo hiperplasia-edema vaginal, presencia de tumores vaginales)
- Tamaño excesivo del feto, sobre todo si sucede en la mayoría de ellos.
- Monstruosidades fetales, síndrome del cachorro solitario, o tamaño realmente excesivo.
- Exceso o déficit de fluidos fetales
- Mal posición fetal no corregible de forma manual
- Muerte fetal
- Toxemia o enfermedad de la madre
- Distocia por negligencia del propietario o criador.
- Cesáreas programadas.
- Descargas vaginales de color rojo, negro o verde oscuras
- Conveniencia para el veterinario y el criador

Como planificar una cesárea

Los cachorros en el útero van a doblar el peso en las últimas dos semanas de gestación y si realizamos una cesárea incluso un día o dos del momento adecuado podemos poner en serio peligro la supervivencia de los neonatos[1]. Podemos tener problemas hepáticos, disminución de los tiempos de coagulación, ausencia de la producción del calostro, mal comportamiento

maternal y muerte neonatal[1], por lo que es fundamental esperar hasta que tengamos los datos de los valores de progesterona en sangre.

1. Datos en los que nos podemos basar para realizar una cesárea programada[123].
   - 63 días después de la ovulación, que coincide con los 63 días después de que la progesterona se ha elevado hasta los 5 ng/ml
   - 65 días después del pico de LH, que coincide con la elevación de la progesterona desde valores basales a 2,5-3 ng/ml
   - Valores de progesterona por debajo de 2,5 ng/ml que son indicativos de que el parto a comenzado y que se há liberado el surfactante pulmonar de los fetos
   - Comienzo de la fase 1 del parto
   - Temperatura rectal por debajo de 37, 22 º C [1]
2. Como podemos potenciar la maduración fetal
   - Aplicando Aglepristone a dosis de 15 mg/Kg 18 a 24 h antes de la realización de La cesárea.
   - Aplicando metil-prednisolona a dosis de 10mg/Kg desde las 24 h antes de realizar la cesárea hasta el momento previo a su realización dependiendo del tiempo el que dispongamos. También podemos utilizar la dexametasona a dosis de 2,2 mg/Kg vía intravenosa.

Anestesia, principios generales.

Colocaremos un catéter intravenoso para mantener el aporte de fluidos.

Preoxigenar comenzando antes de la inducción anestésica siempre que sea posible. Colocaremos a la hembra en una posición sobre la mesa del quirófano con el tórax elevado para impedir que el peso del útero caiga sobre el diafragma e inhiba la respiración. En aquellas hembras de más de 30 kilogramos sería conveniente el lateralizar su cuerpo hacia un lado unos 10º a 15º para evitar que todo el peso del útero grávido caiga encima de la vena cava y de esta manera evitar el síndrome de hipotensión supina ya comentado[123].

Si sospechamos de infección o sospechemos o de la existencia de muerte fetal, podemos utilizar la cefazolina a dosis de 20mg/kg vía intravenosa.

1.- Premedicación[2,3]

Utilizaremos los anticolinérgicos, están indicados en el caso de bradicardia materna y la atropina al cruzar la placenta va a mantener el latido cardiaco fetal, el Glycopirrolato no es capaz de atravesar la placenta. Así mismo nos van a servir para evitar los reflejos vagales que se van a producir al extraer el útero del abdomen.

La gata es todavía más sensible a los reflejos vagales, por lo que el uso de anticolinérgicos es más recomendable. La atropina la utilizaremos a dosis de 0,02/0,1mg/kg en la perra, y de 0,045-0,1mg/kg en la gata vía subcutánea. El glicopirrolato lo utilizaremos a dosis de 0,01mg/kg vía intramuscular.

En el caso de perras muy nerviosas podemos utilizar el butorfanol, que es el narcótico que produce menos depresión respiratoria.

Está descrita la utilización de la hidromorfona[1,4] a dosis de 0,05 mg/Kg previa a la inducción con propofol. Tambien hemos utilizado para la inducción la combinacion de dexmetomidina a dosis de 2 microgramos /Kg iv. com propofol com excelentes resultados[5]

2.-Inducción anestésica.

Los tranquilizantes tienen efectos hipotensores[1], por lo que no los recomendamos en la inducción anestésica, de forma que preferimos aplicar propofol a dosis de 6mg/Kg, esta dosis es en la mayoría de los casos es suficiente para inducir la anestesia y permitirnos la intubación de la madre. Es imprescindible una intubación extremadamente rápida y una oxigenación inmediata, ya que la apnea transitoria que produce el propofol produce una hipoxia y acidosis metabólica en el feto. No está indicado el mantenimiento con propofol porque vamos a disminuir la viabilidad de los neonatos.

Con esta inducción obtenemos aproximadamente 18 minutos de anestesia que nos permitirán en la mayoría de los casos realizar la extracción de los fetos del útero para después mantener la anestesia con agentes inhalatorios. La utilización del propofol nos ha dado muy buenos resultados; por un lado hemos obtenido una alta viabilidad de los cachorros, y por el otro, hemos obtenido una rápida recuperación anestésica de la madre que hace que podamos poner a mamar a los cachorros lo antes posible.

3.-Anestesia de mantenimiento.

Utilizamos como agente inhalatorio solamente cuando hayamos sacado todos los cachorros del útero, el isofluorano, a dosis del 2 al 3% ya que se elimina en su mayor parte en la respiración y sólo se metaboliza en un 1%.

Técnica quirúrgica.

La incisión abdominal debe ser amplia ya que nos va a permitir el poder extraer el útero del abdomen con facilidad, evitando la rotura de los vasos que irrigan el útero y debe ir desde el ombligo al pubis, debemos tener mucho cuidado en no cortar las glándulas mamarias. La incisión en el útero, con preferencia en el cuerpo debe permitir la extracción rápida de los fetos y evitar los desgarros del tejido uterino, esto es muy importante para que la perra pueda volver a gestar con normalidad. Debemos tener cuidado de no exponer

una gran superficie del útero fuera del abdomen ya que esto puede causar hipotermia especialmente en perras pequeñas, una vez que hemos extraído todos los cachorros debemos comprobar que no quedan más cachorros dentro del útero, comprobando ambos cuernos hasta el ovario y en cuerpo uterino.. Administraremos oxitocina, sobre todo si el útero está muy distendido y vemos que tarda en contraerse o presenta una hemorragia abundante a dosis de una a cinco unidades vía intramuscular o intravenosa en perras y a dosis de 0,5 unidades vía intramuscular o intravenosa en gatas donde podemos repetir la dosis pero sin superar las tres unidades como dosis total. También podemos utilizar maleato de ergonovina a dosis de 0,02-0,1 mg/kg, im. La oxitocina se debe administrar también si hay hemorragia del endometrio comprimiendo además las paredes uterinas. A continuación lavaremos el útero para eliminar los coágulos que estén adheridos con solución salina, a continuación cerraremos el cuerpo uterino con una sutura reabsorbible de patrón continuo, tipo Cushing de inversión[2].

Control del dolor después de la cesárea.

Es muy importante el control del dolor después de la cesárea ya que este puede afectar al comportamiento maternal, de forma que la madre se puede despreocupar de los cachorros. los AINES están contraindicados durante la lactación ya que las encimas COX-2 son necesarias para el desarrollo del riñón del neonato, teniendo en cuenta que el cachorro es inmaduro al nacimiento, nosotros recomendamos controlar el dolor post cesárea con butorfanol ya que los efectos sobre el neonato son mínimos porque la dosis ingerida por la leche es muy baja.

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IMPROVE YOUR INTERPRETATION OF RADIOGRAPHS IN THORACIC EMERGENCIES

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ABSTRACT BODY:

Introduction

At least two orthogonal radiographs are important to have as a minimum database in the dog or cat following trauma but should only be completed once the animal is stabilized. Recognizing the presence of free pleural air or fluid and its degree can be critical to the timely performance of thoracocentesis. Ultrasound is not commonly used to assess animals for thoracic trauma but can play a role in specific situations. Computed tomography is very important in animals with spontaneous or traumatic pneumothorax as the source of air leakage is often not detected radiographically. This presentation will cover a large variety of traumatic pathologies of the thoracic and explain how to diagnose them with numerous case examples.

The thoracic wall

Focal soft tissue swelling is commonly observed post-trauma or bite wounds in dogs and cats and that region should be more heavily scrutinized. Rib and sternal fractures may be missed if the fracture fragments remain aligned. Sternal fractures are best identified on lateral images while rib fractures are best diagnosed on the VD or DV image which helps to recognize asymmetry. Multiple rib fractures in segments of at least 2 adjacent ribs is a flail chest which leads to paradoxical movement of the segment during breathing. Healed rib fractures appear as focally rounded or expansile regions of the rib with smooth periosteal borders. These can be difficult to differentiate from aggressive lesions in older animals, especially if there is no history of past trauma. Repeat radiographs in 2 weeks usually show the lesion to be progressive if infectious or neoplastic disease is present.

The diaphragm

Trauma to the diaphragm can result in an abnormal location or shape. Ruptures can lead to a lack of complete visualization of the contour of the diaphragm, cranial displacement without herniation of organs, and herniation of organs with caudal displacement of the diaphragm. Ultrasonography can also be used to help determine if the diaphragm is intact, but can be difficult due to the dome shape and inability to trace all of its aspects. Oral barium sulfate can be administered to confirm the location of the gastrointestinal tract. 20-40 ml of barium suspension can be given orally for this purpose and radiographs performed 15-20 minutes later. Positive contrast peritoneography can also be used to show a communication between the peritoneal and pleural cavities. This procedure can be performed by injecting 2ml/kg of iodinated, non-ionic contrast medium into the peritoneal cavity and then positioning the animal head down (if not in respiratory distress), then performing radiographs afterward.

Radiographic signs of diaphragmatic rupture and hernia:

- Abdominal viscera in the thorax
- Displacement of abdominal structures in the cranial abdomen
- Displacement of the thoracic structures cranially or to one side
- Divergence of the diaphragmatic crura or cranial angulation of the diaphragm.

The mediastinum

Mediastinal hemorrhage can occur secondary to trauma or coagulopathies and appears as a focal, cranial, midline, or left-sided soft tissue opacity that can simulate a consolidated left cranial lung lobe. CT or US may be necessary to locate the left cranial lung lobe and confirm the source to be mediastinal. Tissue aspiration is necessary to confirm.

Pneumomediastinum can often appear following trauma or coughing but can also be associated with open thoracic wounds. Sources of air are the skin, a ruptured bronchus or alveolus, ruptured trachea, and the esophagus. It is recognized radiographically due to thoracic hyperlucency or increased visibility of mediastinal structures such as the great vessels, esophageal wall, and pericardium. Pneumomediastinum can lead to pneumothorax if it becomes severe and can also enter the retroperitoneal space, which is easily recognized radiographically.

The pleural space

Pleural effusion and free air are the most common pleural space abnormalities following trauma. Ultrasound is an excellent screening method for small volumes of pleural effusion and helps to differentiate fat in the pleural space from fluid which can appear similarly on radiographs.

The following are radiographic signs of pleural fluid:

Visualization of widened pleural fissures

Retraction of the lung from the pleural surface of the thoracic wall

Increased soft tissue opacity dorsal to the sternum on lateral radiographs

Blunting of the costophrenic sulci

Obscuring of the normally visible intra thoracic structures and diaphragm

The following are signs of pneumothorax:

Retraction of the pleural surface of the lung or lungs from the thoracic wall

Hyperluceny within the pleural space between the body wall and lung margins

Lung markings do not extend to the thoracic wall

Dorsal displacement of the heart on the lateral image

Can be unilateral or bilateral

Horizontal beam radiography during expiration can be performed in lateral or dorsal recumbency when small volume pneumothorax is present. It can also help to assess the lobe borders in cases of spontaneous pneumothorax when looking for a lung lesion as the source of air leakage.

Tension pneumothorax occurs if pleural space pressure exceeds atmospheric and leads to severe retraction and collapse of the lung lobes. The thoracic size is often very large and it appears hyperlucent. The lungs appear small, triangular, and diffusely soft tissue opaque and retracted. It occurs due to a one-way valve in the lung that lets air escape into the pleural space but not back out. If noticed on a lateral radiograph, the radiographic procedure should be aborted and thoracocentesis should begin immediately.

Pitfalls of pneumothorax diagnosis are often due to the presence of skin folds. For pleural fluid, it can be confused with excessive fat in the pleural space. In both instances, the VD or DV view should be scrutinized along the thoracic wall to determine if fissure lines are present and if the pulmonary vessels extend to the thoracic wall.

The lung

Following trauma, bulla and bleps can occur and can lead to pneumothorax. They appear as small to medium-sized lucent cavities within the lung and can be solitary or multiple. They do not always lead to complications and they can be monitored radiographically. Pulmonary contusions are often focal or multifocal ill-defined zones of hazy increased soft tissue opacity in the lung that can become worse with time.

HOW TO STABILIZE THE DECOMPENSATED DIABETIC PATIENT

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ABSTRACT BODY:

Diabetic emergencies may result from hyperglycemia, secondary acid-base and hydroelectrolytic disorders. Ketoacidosis, hyperosmolality at presentation and hypoglycemia during or after treatment are also common complications. The main objective of the emergency treatment is to prevent the severe sequels of tissue hypoxia, seizures and osmotic shifts over general and central nervous systems as a result of fluid and electrolytes imbalances. Expedite shock resuscitation and adequate correction of seizures, hydroelectrolytic and glucose balance are the corner stone of the therapeutic approach. Due to the possibility of SNC damage, in patients with >48hs of symptom presentation, it is very important to prevent plasmatic variations of osmolality larger than 3-4mOsm/L/h

Diabetic Ketoacidosis (DKA) and Hyperosmolar Hyperglycemic Syndrome (HHS)

DKA and HSS share potentially fatal complications. Morbidity and mortality from these syndromes are related to the significance of metabolic disturbances, concomitant diseases and possible complications derived from improper therapy related to electrolytes and osmolality.

Diabetes mellitus results from the absence of insulin (type I) or insulin resistance (type II). The contributors to diabetes type I development are amyloid deposit, chronic pancreatitis and pancreatic β cells vacuolization. Diabetes type II is the result of some comorbidity factors like obesity, endogen or corticoid administration or the over production of hyperglycemic hormones. The four classic signs of decompensated diabetes mellitus are polyuria, polydipsia, polyphagia and weight lost.

HHS is characterized by severe hyperglycemia (> 600mg/dl), absence or low presence of ketones and serum osmolality over 350mOsm/L. Comatose presentation is not uncommon.

Between 69-92% of patients presented with decompensated DKA or HHS present with an infection, pyometra, gastroenteritis, renal or hepatic failure, pancreatitis, hyperadrenocorticism, neoplasia and/or cardiac failure as precipitant causes. Corticosteroids, thiazides, sympathomimetic agents and progesterone (luteal phase, contraceptives) alter the carbohydrate metabolism and may cause a crisis.

The absolute or relative deficiency of insulin in combination with an increment in hyperglycemic hormones (glucagon, catecholamine, cortisol, growth hormone) increases glycogenolysis, gluconeogenesis rate, decreases glucose tissue extraction, activates lipid metabolism, fatty acid production and their hepatic metabolism into ketones. The absence of insulin activates the hormone-sensitive lipase increasing the production of free fatty acids (FFA). FFA could be utilized as a source of energy by many tissues, they are converted into triglycerides by the liver or totally metabolized into CO_2 and water. FFA could be also transformed into ketone bodies, i.e., acetone, acetoacetic acid, and beta-hydroxybutyric acid but this is limited in non-complicated diabetic patients. Development of ketoacidosis requires increased production of hyperglycemic hormones ("diabetogenic") secondary to an additional stressful insult such as concomitant diseases. In addition of stimulating gluconeogenesis and glycogenolysis, these hormones augment protein catabolism releasing amino acids for the hepatic production of glucose while increasing lipolysis. The resulting FAA will not be used as a source of energy leading to the production of ketone bodies (KB). The accumulation of KB produces the classic DKA triad of hyperglycemia, ketonemia and acidosis, which is generally accompanied by ketonuria, glycosuria, and osmotic diuresis.

The reduction of Glomerular filtration rate (GFR) secondary to dehydration and hypovolemia (due to glycosuria and osmotic diuresis) and concomitant diseases (shock, renal and/or cardiac failure) will favor a marked increase of glycemia, over 600mg/dl and osmolality, greater than 350mOsm/kg (see fig. 1 for osmolality formula) main features of the HHS. The limited insulin concentration present in HHS is enough to inhibit lipolysis which therefore limits ketosis but not to enough to prevent hyperglycemia. Some patients, however, may display a combination of both syndromes (DKA + increased osmolality, or HHS + moderate ketosis).

Osm. osm/kg = (Na+ K) 2 + glu/18 + BUN/2.8

Fig.1 OSMOLALITY. Na sodium, K potassium, glu glycemia, BUN blood Urea Nitrogen

Glycemia (mg/dl)	Fluid therapy DOGS 2.2U/kg/250ml CATS 1.1U/Kg/250ml	Infusion Rate ml/h	Seric Potassium mEq/L	mEq/L K+ added to fluids
> 250	0,9% NaCl	10	> 3.5	20
200-250	0,9% NaCl + 2,5% Dex.	7	3.0-3.5	30
150-200	0,9% NaCl + 2,5% Dex.	5	2.5-3.0	40
100-150	0,9% NaCl + 2,5% Dex.	5	2.0-2.5	60
<100	0.9% NaCl + 5% Dex	Discontinue	< 2.0	80

Adjustments for intravenously administered regular crystalline insulin, dextrose, and potassium. Adapted from Soren Boysen Fluids and Electrolyte Therapy in Endocrine Disorders: Diabetes Mellitus and Hypoadrenocorticism. *Vet. Clin North Am Small Pract*.2008; 38(3):705-707

Table 1.

Treatment

Rapidly stabilize the cardiovascular system and restore water and electrolyte loses before initiating insulin therapy. In order to treat shock, end goal directed therapy with warm ringer lactate RL or plasmalyte should be given at a rate of 10 – 15 cc/kg in 30 minutes. This will increase tissue perfusion and GFR reducing acidosis by promoting aerobic metabolism, liver metabolism of lactic acid and KB as well as the renal excretion of H+.

Fluid replacement therapy should be continued after reversion of shock. Fluid therapy will reduce glycemia by 30-50% and hyperglycemic hormones facilitating the cellular response to insulin therapy. Insulin therapy should be withheld for the first 4 to 6 hs to prevent rapid reduction of osmolality and fluid shifts that may cause cerebral damage. Fluid therapy will gradually reduce the osmolality by maintaining certain degree of hyperglycemia while normalizing natremia thus preventing cerebral edema. Serum glucose and electrolytes should be monitored every hour. Glycemia should be kept over 250mg/dl during the first 4-6 hours, avoiding significant changes of glycemia, less than 100mgGLU/dl/h and osmolality, less 3-4mOsm/l/h. Dextrose 2.5% administration along with insulin titration may be needed to accomplish this therapeutic goal.

Hyperkalemia and Hyperphosphatemia are common at presentation; however, body depletion of these electrolytes is generally severe. Fluid and insulin therapies tend to rapidly reduce their serum concentrations. GFR should be restored, and renal failure oligo anuria should be ruled out as a concomitant cause of hyperkalemia. Serum potassium tends to severely drop once GFR is normalized. As long as K+ administration is ≤ 0.5mEq/kg/h and renal function is normal, fluid therapy can be supplemented according to Table 1.

If signs of hypokalemia persist despite an aggressive K+ supplementation, hypomagnesaemia should be addressed by adding magnesium sulfate as constant rate infusion CRI of 0.75- 1.0mEq/kg/day in 5% dextrose. Dosage should be gradually reduced by 50% in 3-5 days. Hypophosphatemia (<1.5mg/dL) may cause hemolysis and seizures; it is advisable to supplement 0.01-0.06 mmol/kg/h of potassium phosphate in RL during the first 12-48 hs, (consider the additional source of potassium). A quarter of the calculated base deficit of NaHCO3 should be administered only if acidosis persists (pH < 7.0) after one hour fluid therapy.

Regular insulin administration should be started after 4-6hs of fluids and only when K^+ concentration is within the high-normal range. 2,2U/kg (dogs) and 1,1U/kg (cats) or regular crystalline insulin in 250ml of NaCl 0,9% or Lactated Ringer should be administered adjusting the infusion rate according to patient glycemia (Table 1).

Once the patient is eating appropriately, long lasting acting insulin (NPH o zinc insulin) should be administered SC every 12 to 24hs, depending on the insulin of choice. Oral administration of lipoic acid (200mg/kg/8hs), and chromium picolinate (200 µg/day) might help stabilizing serum glucose concentration by enhancement of the biological action of insulin. Ovariohysterectomy (OVH) could definitively resolve the occurrence of diabetes mellitus and should be recommended to all affected intact bitches once they are clinically stable.

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CURRENT CONCEPTS OF PERIODONTAL DISEASE

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ABSTRACT BODY:

Current Concepts of Periodontal Disease

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Periodontal disease is the **number one** medical condition in small animal veterinary medicine. We will begin this presentation with an overview of the current knowledge as to the pathogenesis of periodontal disease. This will allow us to properly treat the condition. Following this is a discussion of the local and systemic effects of periodontal disease. This will give attendees the ability to improve client compliance with dental recommendations. In addition, a firm grasp of the disease process will improve practitioner understanding of proper treatment modalities.

Due to the plethora of new and concerning information about this condition, treatment and prevention is the subject of significant research. This focus has resulted in numerous new products and procedures to prevent and treat periodontal disease and this presentation is designed as an introduction to these new and future therapies.

Periodontal disease overview

Periodontal disease is the number one health problem in small animal patients. By two years of age, 70% of cats and 80% of dogs have some form of periodontal disease. However, there are generally few to no visible clinical signs, and therefore therapy typically comes very late in the disease. Consequently, periodontal disease may also the most undertreated disease in our patients.

Pathogenesis:

Periodontal disease is generally described in two stages, gingivitis and periodontitis. Gingivitis is the initial, **reversible** stage in which the inflammation is confined to the gingiva. The gingival inflammation is created by plaque bacteria and may be reversed with a thorough dental prophylaxis and consistent homecare. Periodontitis is the later stage of the disease process and is defined as an inflammatory disease of the deeper supporting structures of the tooth (periodontal ligament and alveolar bone) caused by microorganisms. The inflammation results in the progressive destruction of the periodontal tissues, leading to attachment loss. This can be seen as gingival recession, periodontal pocket formation, or both. Mild to moderate periodontal pockets may be reduced or eliminated by proper plaque and calculus removal, however, periodontal bone loss is **irreversible** (without regenerative surgery). Although bone loss is irreversible, it is possible to arrest its progression. However, it is more difficult to maintain periodontally diseased teeth in comparison to healthy teeth. Additionally, periodontal attachment loss may be present with or without active inflammation.

Periodontal disease is initiated by oral bacteria which adhere to the teeth in a substance called plaque. Plaque is a biofilm, which is made up almost entirely of oral bacteria, contained in a matrix composed of salivary glycoproteins and extracellular polysaccharides. Calculus (or tartar) is basically plaque which has secondarily become calcified by the minerals in saliva.

It is important to note that rough tooth surfaces will greatly increase the speed of plaque and calculus formation. Therefore any condition which detracts from the smooth tooth surface should be addressed. This can be as simple as a bonded sealant or in some cases can be better treated by a composite restoration. Another situation which commonly hastens the onset of periodontal disease is crowding. This will impair the not only homecare efforts, but also the natural cleaning ability of the patient.

Plaque and calculus may contain up to 100,000,000,000 bacteria per gram. More importantly, bacteria become must more resistant to antiseptics and antibiotics than their free living or "planktonic" counterparts. In fact, they are 1,000 to 1,500 times more resistant to antibiotics and antiseptic concentrations need to be up to 500,000 times that which would kill singular bacteria.

Plaque on the tooth surface is known as supragingival plaque. Once it extends under the free gingival margin and into the area known as the gingival sulcus (between the gingiva and the teeth or alveolar bone), it is called subgingival plaque. Supragingival plaque likely affects the pathogenicity of the subgingival plaque in the early stages of periodontal disease. However, once the periodontal pocket forms, the effect of the supragingival plaque and calculus is

minimal. **Therefore, control of supragingival plaque alone is ineffective in controlling the progression of periodontal disease.**

Initial plaque bacteria consists of predominately non-motile, gram-positive, aerobic facultative rods and cocci. Gingivitis is initiated by an increase in the overall number of bacteria, which are primarily motile gram negative rods and **anaerobes.** The specific plaque hypothesis is based on the fact that these few species are seen in virtually all cases of chronic periodontal disease.

In addition to their metabolic products, the bacteria in the subgingival plaque secrete toxins such as cytotoxins and bacterial endotoxins which invade tissues on their own, and in turn cause inflammation to the gingival and periodontal tissues. This inflammation causes damage to the gingival tissues and initially results in gingivitis. Eventually, the inflammation can lead to periodontitis, i.e. the destruction of the attachment between the periodontal tissues and the teeth.

In addition to directly stimulating inflammation, the bacterial metabolic byproducts also elicit an inflammatory response from the animal. White blood cells and other inflammatory mediators migrate out of the periodontal soft tissues and into the periodontal space due to increased vascular permeability and increased space between the crevecular epithelial cells. White blood cells fight the infection by phagocytizing bacteria, but may also release enzymes to destroy the bacterial invaders either by design or after their death. When released into the sulcus, these enzymes will cause further inflammation of the delicate gingival and periodontal tissues. **In fact, the progression of periodontal disease is determined by the virulence of the bacteria combined with the host response.** It is the host response that often damages the periodontal tissues. However, patients with deficient immune systems typically have more severe periodontal disease than those individuals in good health.

The inflammation produced by the combination of the subgingival bacteria and the host response damages the soft tissue attachment of the tooth, and decreases the bony support via osteoclastic activity. This causes the periodontal attachment of the tooth to move apically. The end stage of periodontal disease is tooth loss; however the disease has created significant problems prior to tooth exfoliation.

Clinical Features:

It is important to be familiar with normal features in order to identify abnormal findings. Normal gingival tissues are coral pink in color (allowing for normal pigmentation), and have a thin, knife-like edge, with a smooth and regular texture. There should be no demonstrable plaque or calculus on the dentition.

The first **obvious** clinical sign of gingivitis is erythema followed by edema of the gingiva. However, it is now known that the **first** evidence of gingivitis is bleeding during brushing, probing, or after chewing hard/rough toys. Therefore it is important to realize that normal appearing teeth/gums can actually be infected. If the first stages of gingivitis are not treated, it will progress into edema, spontaneous bleeding, and halitosis.

Gingivitis is typically associated with calculus on the involved dentition, but is primarily elicited by **plaque** and thus can be seen in the absence of calculus. Alternatively, widespread supragingival calculus may be present with little to no gingivitis. It is critical to remember that calculus itself is essentially non-pathogenic. Therefore, the degree of gingival inflammation should be used to judge the need for professional therapy.

As gingivitis progresses to periodontitis, the oral inflammatory changes intensify. The hallmark clinical feature of established periodontitis is attachment loss. In other words, the periodontal attachment to the tooth migrates apically. As periodontitis progresses, alveolar bone is also lost. On oral exam, there are two different presentations of attachment loss. In some cases, the apical migration results in gingival recession while the sulcal depth remains the same. Consequently, tooth roots become exposed and the disease process may be identified on conscious exam. In other cases, the gingiva remains at the same height while the area of attachment moves apically, thus creating a periodontal pocket. This form is typically diagnosed only under general anesthesia with a periodontal probe. It is important to note that both presentations of attachment loss can occur in the same patient, as well as the same tooth. As attachment loss progresses, alveolar bone loss continues, until tooth exfoliation in most cases. After tooth exfoliation occurs, the area generally returns to an uninfected state, but the bone loss is permanent.

SOLUCIONES PARA MASCOTAS RECIÉN ADOPTADAS

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ABSTRACT BODY:

Las primeras etapas de la vida de un cachorro son fundamentales para el desarrollo de su comportamiento adulto. En las especies altriciales, como los perros, el desarrollo del sistema nervioso central (SNC) no se ha completado al nacimiento y, por lo tanto, la maduración final se da en el exterior de la madre. El entorno del cachorro y su manejo durante los primeros meses de vida debería garantizar el correcto desarrollo de las estructuras que gestionan la respuesta de estrés. De esta manera se evitarán problemas de conducta y/o de gestión emocional en la edad adulta.

PRINCIPALES ETAPAS DEL DESARROLLO EN EL CACHORRO Y SUS IMPLICACIONES PRÁCTICAS

La ontogenia de la conducta estudia los cambios que se producen en el comportamiento de un individuo y los mecanismos responsables de estos. Las etapas que se definen del desarrollo del cachorro son:

- Prenatal. El desarrollo del SNC empieza durante la gestación en el útero materno. El estrés durante la gestación tiene efectos duraderos e intensos en las estructuras que controlan la respuesta de estrés. Los perros de madres estresadas durante la gestación tiene una respuesta de estrés menos ajustada, es decir, es desproporcionada a la amenaza real en intensidad o duración. Esto puede afectar al bienestar del perro cuando es adulto y, además, se correlaciona con una mayor presencia de conductas relacionadas con el miedo y la ansiedad.
- Neonatal. En el perro comprende desde el nacimiento hasta los 15 días de vida. Este período, de nuevo, es crucial para el desarrollo equilibrado de las estructuras que controlan la respuesta de estrés. Estresar demasiado el cachorro durante esta etapa (con manejos dolorosos, separaciones temprandas de la madre, etc.) tienen consecuencias muy intensas y duraderas durante toda la vida del animal. Como ocurría en la etapa anterior, la respuesta de estrés será desproporcionada en la edad adulta, correlacionándose con conductas asociadas al miedo, como la agresividad. Sin embargo, un punto importante es que cierta cantidad de estrés durante la fase neonatal será beneficioso para el desarrollo del sistema límbico. Para conseguir este mínimo estrés, se aconseja que varias veces al día el cachorro sea separado de la madre durante un breve período de tiempo (1-2 min). Esta separación hace que, al ser reintroducido el animal en la camada, la madre centre sus lamidos en el cachorro que ha sido separado. Esta estimulación tactil favorece el correcto desarrollo de las estructuras implicadas en la respuesta de estrés. Por lo tanto, en el período neontal, debe garantizarse un grado de manipulación tactil pero, sin embargo, se debe evitar el manejo estresante intenso. En este perro es sordo y ciego.
- Período de transición. Comprende de las 2 a las 3 semanas. El animal continúa siendo sordo y ciego, pero ya ha abierto el canal auditivo y los ojos. Su capacidad motora empieza a mejorar y ya tiene cierta autonomía.
- Período de socialización. En el perro, va de las 3 semanas a los 3 meses de vida (aproximadamente). En algunas razas podría ser que este período finalizara más tarde (sobre las 13-15 semanas). Se caracteriza porque el perro ya es maduro sensioralmente, pero no tiene la capacidad de sentir miedo. Las primeras reacciones de miedo aparecen sobre la quinta semana y madura completamente sobre el tercer mes, cuando termina el período de socialización. Es un período crucial ya que el animal aprende las pautas sociales, se lleva a cabo el reconocimiento de las diferentes espcies, el animal empieza a moldear el lenguaje y su conducta (con sus relaciones con la madre y con los hermanos) y, además, se produce la habituación a los estímulos más habituales en la vida del animal. Asegurar una correcta socialización evitará problemas de miedo durante la edad adulta (y sus conducta asociadas) y asegurará que el animal utilice correctamente las estrategias de comunicación. Finalmente, una correcta socialización permitirá, entre otras muchas cosas, que el animal aprenda a gestionar correctamente las situaciones de frustración cuando sea adulto.
- Período juvenil/adolescencia. Recientemente se ha demostrado que durante la adolescencia se observa, al igual que sucede en los seres humanos, un empeoramiento de la conducta. Estas conductas indeseadas, con frecuencia, hacen que los tutores utilicen manejos incorrectos basados en el castigo

positivo. La utilización de castigo (positivo) favorece la creación de un vínculo inseguro del perro hacia su tutor, lo que dificulta sus habilidades sociales cuando es adulto, entre otras cosas. Utilizar métodos amables de educación, garantizar un correcto enriquecimiento ambiental, así como un manejo consistente en las rutinas diarias, favorecerá que el comportamiento del cachorro sea bueno y estable cuando sea adulto.

MANEJO CLÁSICO (E INCORRECTO) DEL CACHORRO EN CASA

Clásicamente se ha realizado un manejo que no atendía a las necesidades reales del cachorro. Son frecuentes los siguientes manejos:

- Separación abrupta de la madre. Durante las primeras semanas de vida, cuando el cachorro está formando el apego con la madre, que será su figura de seguridad, se le separa de forma abrupta de la madre. Esta separación hace que el cachorro se encuentre en un entorno nuevo sin tener acceso a su figura de seguridad. Será, por lo tanto, normal que durante los primeros días el animal muestre el estrés creado por la ausencia de la madre (de su figura de seguridad) vocalizando e, incluso, destruyendo parte del mobiliario. Utiliza el castigo en estas situaciones incrementa el estrés del cachorro y, quizás peor, impide la posibilidad de que genere un apego seguro con sus nuevo cuidadores.
- Ignorar las vocalizaciones de las primeras noches. Una recomendación frecuente (e incorrecta) era que el animal debía ser ignorado si por la noche ladraba o aullaba, bajo el pretexto que si se atendían sus demandas podía empeorar la situación ya que se reforzaba esa conducta. Sin embago, hoy en día se sabe que este manejo es incorrecto, ya que el hecho de no atender a las demandas emocionales del cachorro empeora la calidad del vínculo con los cuidadores (los tutores). Nunca deberían ignorarse las vocalizaciones que se deben a ansiedad, miedo o cualquier otra demanda emocional del cachorro. Con el tiempo se le educará, progresivamente (y una vez que se haya creado un vínculo de seguridad), a quedarse solo.
- Adquisición de las pautas de higiene. Durante tiempo se utilizó la técnica de castigar al animal si orinaba y defecaba en lugar incorrecto. Sin embargo, no será hasta los 5-6 meses en los que el animal será capaz de controlar la eliminación y de hacerlo en un lugar o sustrato específico. Ademas, el castigo no solo no es efectivo, sino que es contraproducente. La consecuencia de castigar cuando se le pilla al cachorro orinando o defencando en que no quiera orinar o defecar delante de la persona que le ha castigado (normalmente alguno de los tutores). Además, de nuevo, el vínculo puede verse gravemente afectado.
- Lo mismo ocurre con las conductas de juego o las conductas de exploración que están muy motivadas en cachorros y que, a menudo, acarrean que rompa cosas en casa. En este caso tampoco debería castigarse o intentar inhibirse la conducta, sino que deberá redirigirse hacia objetos o juguetes que sí sean aceptables por los tutores.

Como se ha descrito, y otras situaciones que se describirán durante la charla, el manejo del cachorro clásicamente ha sido incorrecto y ha sido, sin duda, la fuente de muchos problemas de conducta.

CONCLUSIONES Garantizar un correcto manejo, tanto antes de la adopción (de la madre y de la camada estando con la madre) como una vez que el cachorro llega al nuevo hogar, es fundamental para que el comportamiento adulto del cahorro sea estable. En términos generales, el manejo debería cubrir las necesidades emocionales y fisicas el perro, debería garantizar que el cachorro pudiera expresar las conductas de necesarias y, finalmente, debería ser consistente a lo largo del desarrollo y crecimiento del perro.

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PACIENTE ONCOLÓGICO. CALIDAD DE VIDA AL FINAL DE SUS DÍAS

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ABSTRACT BODY:

Paciente oncológico. Calidad de vida al final de sus días

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El cáncer es una enfermedad crónica y es la causa de muerte más frecuente en los animales geriátricos.

A menudo enfermedades propias de la edad avanzada como problemas articulares, cardiorrespiratorios, de obesidad, hormonales, renales y/o hepáticos pueden hacer complicado un tratamiento oncológico y se debe de tener presente al momento de la discusión con el propietario sobre las posibles alternativas a seguir. Nuestro objetivo siempre será combinar el tratamiento de estos cambios ligados al envejecimiento con los propios de la enfermedad oncológica. (7)

Los veterinarios a menudo debemos de realizar un buen trabajo psicológico, pues nos corresponde informar sobre el diagnóstico y pronóstico de nuestros pacientes a sus propietarios. (7)

El Cáncer en las mascotas

Para hablar de neoplasias en las mascotas debemos de tener en cuenta los siguientes factores.

La edad del paciente oncológico

En un paciente geriátrico los problemas articulares, cardiorespiratorios, problemas de obesidad, trastornos hormonales, enfermedades renales y/o enfermedades hepáticas pueden hacer en exceso muy complicado el tratamiento oncológico. (7).

Además de las indicaciones terapéuticas que requiera el paciente es muy importante considerar su estado y características físicas del paciente como por ejemplo facilidad para coger vías venosas, carácter del animal. En conclusión los factores más importantes a considerar en el paciente son su actividad física y su salud general. (3).

El estado nutricional del paciente oncológico

Utilización del sustrato

La mayoría de células neoplásicas se reproduce mejor en un medio con alto contenido en glucosa. Esto posiblemente se deba a un metabolismo limitado de los ácidos grasos junto con un aumento de las vías metabólicas que utilizan la glucosa, esto se ha denominado tradicionalmente el efecto “Warburg”, en el que se sugiere que la glicolisis es la vía principal para la producción de energía en las células neoplásicas. (9).

La pérdida de peso progresiva y severa se observa con mucha frecuencia en pacientes felinos y caninos con cáncer. La mascota puede perder su apetito durante el tratamiento del cáncer, y el apoyo nutricional es especialmente importante durante este tiempo. (8)

Una alteración sistémica muy frecuente y manifiesta en pacientes con cáncer es la profunda malnutrición que se produce así como el desgaste y la pérdida de peso que esto conlleva. La pérdida de peso y las alteraciones metabólicas observadas en pacientes con cáncer, a pesar de su adecuada nutrición, se conocen como caquexia, mientras que las alteraciones observadas debidas a una nutrición pobre se conocen como anorexia. Estos síntomas son frecuentes en el síndrome paraneoplásico en perros y gatos y es preciso atender estos aspectos. (7).

El dolor en el paciente oncológico

Aliviar el dolor es importante por motivos físicos y éticos ya que el dolor puede producir una respuesta de estrés en las pacientes que se asocia al aumento de corticotropina, cortisol, hormona antidiurética, catecolaminas, aldosterona, renina angiotensina 2 y glucosa y una disminución de insulina y testosterona, estos cambios pueden producir un estado catabólico general con catabolismo de proteínas musculares y lipólisis, además de retención de agua y sodio y excreción de potasio (2).

Es de suma importancia, para hacer la vida de su mascota lo más cómoda posible y sin dolor, que este sea tenido en cuenta y tratado. Estos tratamientos deben ser realizados únicamente por su veterinario u oncólogo veterinario de confianza. Los veterinarios están éticamente obligados a reconocer, evaluar, prevenir y aliviar el dolor a todos los animales de compañía a su cuidado. (8).

El vínculo humano - animal

Los recientes cambios sociales han provocado un cambio de paradigma en la relación veterinario-propietario-paciente. Las crecientes expectativas de los propietarios, el fuerte apego entre las personas y sus animales de compañía y el creciente conocimiento de los propietarios exigen un cambio en el estilo de comunicación. Pasando del tradicional enfoque paternalista a una relación de colaboración. (9).

La relación afectiva propietario-paciente muchas veces es determinante a la hora de elegir un tratamiento. Hay que tener en cuenta que la familia está preocupada y los conceptos cáncer y quimioterapia tienen connotaciones muy negativas. Para evitar un rechazo absoluto a las alternativas disponibles es conveniente transmitir las malas noticias a los propietarios gradualmente y darles tiempo para asimilar y tomar decisiones. (3).

La calidad de vida

En medicina veterinaria cuando realizamos un diagnóstico de cáncer las expectativas iniciales del propietario se centran en la curación de la enfermedad y en que la mascota viva más tiempo. Luego los propietarios buscan pasar más calidad de tiempo al lado de sus mascotas, es aquí donde es crucial hablar sobre la calidad de vida de las mascotas. (9).

Quality of life scale		
Criterio		
Hurt	-	Dolor
Hunger	-	Hambre
Hydratation	-	Hidratación
Hygiene	-	Higiene
Happiness	-	Felicidad
Mobility	-	Movilidad
More good days than bad days	-	Más días buenos que malos
Adapted from Canine and Feline Geriatric Oncology: Honoring the Human–Animal Bond, Villalobos A, Kaplan L—Hoboken, NJ: Wiley-Blackwell, 2007		

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- Como manejamos el momento de la eutanasia
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HOW I TREAT ACUTE PAIN FROM ABDOMINAL SURGERY

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ABSTRACT BODY:

Abdominal pain from surgery or medical conditions can be difficult to treat and pain from any source can cause gastrointestinal complications. Thus, pain relief is critical.

Options for abdominal pain relief:

Systemic drugs

Opioids are an option and should be part of the protocol for moderate/severe abdominal pain – as would occur in patients undergoing abdominal surgery – both intra- and post-operatively. Severe pain will require a full-opioid agonist (morphine, methadone, hydromorphone, fentanyl) but opioids alone are not often sufficient for control of visceral pain and high dosages of opioids can contribute to slowed GI motility – of course so can pain – so it is a balance of treatments.

NSAIDs may be an option depending on the source of pain, type/location of surgery or disease (ie, surgery for resection/anastomosis vs surgery for removal of bladder stone – both cause abdominal pain but tissue at intra-abdominal surgical site is very different).

Infusions of lidocaine +/- opioids +/- ketamine are an excellent choice for both surgical and medical pain.

In addition to pain relief, lidocaine CRIs are anti-inflammatory, antiarrhythmic and improve postoperative GI function (proven in humans and horses). The mechanism of action of systemic lidocaine is not entirely clear. Proposed mechanisms include blockade of sodium channels or potassium currents in the dorsal horn of the spinal cord and direct inhibition of abnormal electrical charges from injured or inflamed peripheral nerves. Lidocaine CRIs are extremely useful in dogs but are somewhat controversial in cats because decreased cardiac function occurred when lidocaine infusions were administered to halothane-anesthetized cats. However, the halothane was likely part of the problem. Because of the uncertainty of lidocaine effects in cats, some veterinarians feel that lidocaine CRI is not warranted in the cat at all while others feel that it is an appropriate means to treat pain, especially in patients where other options may be limited. If lidocaine CRI is chosen, using low dosages in conscious cats (ie, not under anesthesia) is recommended. Lidocaine CRIs are commonly used in dogs, especially in dogs with gastro-intestinal pain (eg, pain from exploratory laparotomy, gastric dilatation-volvulus [GDV], pancreatitis, parvovirus, etc…). There is a very good open-access CRI calculator at IVAPM.org (see reference list at the end of the document). Using lidocaine as an infusion:

Loading Dose	CRI dose	Quick Calculation	Comments
0.25 mg/kg IV (dog); none or 0.1 mg/kg IV (if used in cats)	1.5 mg/kg/hr (25 mic/kg/min) Some sources recommend no more than 10 mic/kg/min in cats	Add 750 mg to 500 ml fluid & run at 1 ml/kg/hr 10 mic/kg/min would be 300 mg lidocaine in 500 ml fluid with a rate of 1 ml/kg/hr	750 mg=37.5 ml, remove 37.5 ml LRS before adding L; can be combined with opioid &/or ketamine **Lidocaine may be contraindicated in the cat due to cardiovascular effects. If used in cats, use ½ the dog dose.**

Local/regional analgesics

Peritoneal lavage

- After opening the linea (preferred) OR after completing the surgery (more practical), draw up 2-4 (cat) – 4-6 (dog) mg/kg lidocaine OR 1-2 (cat) – 2-4 (dog) mg/kg bupivacaine and, if necessary dilute the drug with saline – the total volume needs to be 0.4-0.6 ml/kg to 'lavage' or 'bathe' the entire abdominal cavity. 'Bathe' the peritoneal cavity with the mixture by instilling it into the abdomen through the incision. Complete the OHE and close the incision as usual.
- Publications are available in both dogs and cats for the efficacy of this block for OHE. In humans, the block is also used for abdominal exploratory surgeries and cesarean sections. These uses are also common in veterinary medicine, but there are no research studies. For a good review and more information see Steagall et al. 2020 (see the reference list at the end of the document).

Lumbosacral epidural analgesia

- **Opioids** are most commonly used but local anesthetic drugs can be used in conjunction with opioids.

- 0.1 mg/kg morphine (preservative-free is gold standard but morphine with preservative is commonly used in veterinary medicine)
- Dilute to 1 ml/4.5 kg with bupivacaine, sterile saline or sterile water

- Provides up to 24 hours of analgesia with little to no systemic effects. The opioids will cause sensory blockade but will not cause motor blockade. The local anesthetics can cause motor blockade, however, the motor effects are generally minimal or absent by the time the patient recovers from anesthesia to the point that it is ambulatory.
- Consider for any pain in caudal half of patient. Examples include, rear limb soft tissue or orthopedic surgery, abdominal exploratory and bladder surgeries, surgeries on the tail or perineal region, etc…
- Technique:
  - Place the anesthetized patient in sternal or lateral recumbency
    - Legs can be placed forward or to the back. I prefer forward in cats and small dogs.
  - Locate the wings of the ilium and palpate the lumbo-sacral (LS) space (almost directly in line with the wings of the ilium on the mid-line).
  - Clip and scrub this region. Wear gloves and use a small drape or glove wrapper.
  - Insert an epidural needle into the caudal portion of the LS site with the needle angled at approximately 45° from vertical.
  - Slowly advance the needle until the epidural space is entered.
    - 'Hanging' drop often works (aspiration of fluid in the hub of the needle as the epidural space is entered).
    - Several 'pops' will be felt.
    - 'Walking off' the bone is the most definitive determination of proper placement of the needle in between vertebrae.
  - STOP as soon as the space is entered and slowly inject the drug.
    - The drug should inject easily if the needle is in a space.
    - Stop injecting and take your thumb off the plunger. the fluid should momentarily continue to flow if the tip of the needle is in the epidural space. This is the most definitive determination that the needle is in the lumbosacral space.
    - If the drug does not inject easily, back up a VERY tiny amount and try again.
  - Once drug has been injected, remove needle and proceed with surgery.
    - If local anesthetic drugs have been used, may want to lay patient with surgical side down for about 5 minutes.
  - Opioid epidurals do NOT affect motor function of the rear limb or diaphragm. Local anesthetic drugs can affect motor function but rarely do (volumes that are described here do not migrate far enough cranially to affect the diaphragm so ventilation is not impaired).
- Complications include ineffective or incomplete block (by far the most common complication), epidural hematoma or abscess, hyperalgesia (VERY rare). Contraindications include bleeding disorders (to prevent hematomas) and skin disease over the LS space (to prevent abscesses). Abnormal pelvic anatomy (either from congenital lesions or trauma) may make epidurals difficult.
- Epidural catheters are fairly easily placed in larger dogs and can be maintained for several days to allow continuous or intermittent delivery of analgesic drugs.

Sacrococcygeal or intercoccygeal epidural

- This block is often used to provide analgesia for tail amputations, perineal urethrostomies, and placement of urinary bladder catheters for urethral obstructions. Won't desensitize the entire abdomen.
- Move the tail up and down in a 'pumping' motion while palpating the sacrococcygeal region of the patient. The first movable space at the caudal end of the sacrum is either the sacrococcygeal or intercoccygeal space. Either site is appropriate for injection.
- Insert a 22-G needle through the skin ON MIDLINE at a 45-degree angle to the skin surface.
- Proceed slowly until needle enters the space (generally hit bone and 'walk off' the bone).
- Hanging drop technique often works. Should have no resistance on injection.
- Use lidocaine for rapid onset (0.1-0.2 ml/kg 2% lidocaine), can add an opioid (same as for lumbosacral epidural) for long-term analgesia. Don't inject air, air bubble may cause incomplete block since this is a very small space.

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HOW I TREAT LARYNGEAL PARALYSIS

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ABSTRACT BODY:

Emergency Treatment: Acute exacerbation of laryngeal paralysis is a life-threatening condition that may require aggressive intervention. Dogs (and cats) can present in respiratory distress with marked inspiratory and likely expiratory stridor. Animals can be cyanotic and hyperthermic.

Initial treatment is directed at improving ventilation, reducing laryngeal edema, and minimizing the animal's stress. A typical treatment regimen in dogs involves oxygen supplementation and administration of short-acting steroids and sedatives. Administration of acepromazine, buprenorphine, butorphanol, or midazolam can be considered in both dogs and cats that are in distress. Supplemental oxygen is administered by nasal insufflation, flow-by, face mask, oxygen cage, or endotracheal intubation. These animals are often also hyperthermic and appropriate cooling procedures should also be instituted.

If respiratory distress cannot be abated, intubation or a temporary tracheostomy should be considered. However, temporary tracheostomies are associated with significant complications, particularly obstruction from blood clots or mucus, tube dislodgement, aspiration pneumonia, and stoma swelling. Therefore, intensive monitoring of a patient with a temporary tracheostomy tube is required to avoid life-threatening complications. This procedure has also been shown to make dogs with laryngeal paralysis more at risk for aspiration pneumonia postoperatively.

In cadaveric and experimental models, translaryngeal percutaneous arytenoid lateralization has been assessed, which involves placing a mattress suture through an arytenoid cartilage from an oral approach.[1]Clinical application has yet to be reported. Two small studies have evaluated the use of a silicone stent in dogs with laryngeal paralysis and reported promising outcomes.[2,3] Acute improvement was documented in all dogs, thus this option could be a used for temporary stabilization. Emergency unilateral arytenoid lateralization can also be considered.

Medical Treatment: Often dogs are not severely affected clinically until they have bilateral laryngeal paresis or paralysis. Therefore, dogs with unilateral laryngeal dysfunction are typically not surgical candidates. For dogs with bilateral laryngeal paralysis, the decision to recommend surgery is based on the quality of life of the dog, severity of clinical signs, and time of year. The goal of conservative management of dogs with laryngeal paralysis is to improve the quality of life through environmental changes, reduction of daily exercise, owner education, weight loss, and consideration of anti-inflammatory drugs to minimize laryngeal swelling. Doxepin and other tricyclic antidepressants have been anecdotally recommended to help dogs with laryngeal paralysis. A recent double-blinded, randomized, placebo-controlled trial did not find any benefit of use of doxepin in canine laryngeal paralysis.[4] Regardless, medical treatment is insufficient for long-term management. For dogs that are diagnosed with concurrent hypothyroidism, thyroid supplementation should be instituted, but as noted earlier, this rarely improves clinical signs of laryngeal paralysis but can assist with general overall wellness.

Surgical Treatment: Laryngeal paralysis is a surgical condition. Numerous surgical techniques are available to treat laryngeal paralysis. Unilateral arytenoid lateralization is the current technique of choice for most surgeons, but various methods of partial laryngectomy (bilateral ventriculocordectomy, partial arytenoidectomy) are also performed. Bilateral arytenoid lateralization is not recommended as it has been shown to result in unacceptable morbidity. Other techniques include castellated laryngofissure, reinnervation of the laryngeal musculature, and permanent tracheostomy. Permanent tracheostomy is considered a salvage procedure for dogs most at risk for aspiration pneumonia, but it is associated with a high rate of major and minor complications and requires diligent postoperative and long-term care. As stated prior, laryngeal silicone stenting in dogs with laryngeal paralysis has recently been reported with good outcomes up to 13 months.[2]

Several variations of unilateral arytenoid lateralization have been described. The most common technique involves suturing the cricoid cartilage to the muscular process of the arytenoid cartilage. This mimics the directional pull of the cricoarytenoid dorsalis muscle and rotates the arytenoid cartilage laterally. Increasing the surface area of the rima glottis beyond the edges of the epiglottis may put the animal more at risk for aspiration. Limited lateral displacement of the

arytenoid cartilage will significantly reduce airway resistance within the larynx and may decrease the risk for postoperative aspiration pneumonia. This is accomplished by minimizing the degree of dissection: separation of the cricothyroid articulation, transection of the sesamoid band connecting the paired arytenoid, and complete disarticulation of the cricoarytenoid joint are not necessary. A partial opening of the cricoarytenoid articulation allows accurate visualization of needle placement through the muscular process of the arytenoid but limits the degree of arytenoid cartilage abduction.

Postoperatively, dogs are recovered with minimal to no systemic opioid administration to avoid respiratory depression. Local analgesia can be used in subcutaneous tissues; nonsteroidal anti-inflammatories can be administered if no contraindications exist. Dogs are often discharged from the hospital the same day as surgery with two to four weeks of cisapride to minimize risk of aspiration pneumonia.[5]

Prognosis: Laryngeal Paralysis

Aspiration pneumonia is the most common complication in dogs surgically treated for laryngeal paralysis (15-20%). Although aspiration pneumonia occurs most likely in the weeks following surgery, it has been recognized that these dogs are at risk for aspiration pneumonia for the rest of their lives. Factors that have been significantly associated with a higher risk of developing complications and influencing long-term outcome include preoperative aspiration pneumonia, development of oesophageal dysfunction, progression of generalized neurologic signs, temporary tracheostomy placement, and concurrent neoplastic disease.[6] Without surgical complications, unilateral arytenoid lateralization results in less respiratory distress and stridor and improved exercise tolerance. Owner satisfaction with this procedure has been reported as excellent, with most owners believing that the quality of their dog's life was dramatically improved.[7]

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CHALLENGES AND OPPORTUNITIES FOR TACKLING COMPANION VECTOR-BORNE DISEASES IN LATIN AMERICA

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ABSTRACT BODY:

Companion vector-borne diseases in Latin America (LATAM)

Companion vector-borne diseases (CVBD) are a group of illnesses of dogs and cats, which are caused by a magnitude of pathogens transmitted by arthropod vectors (e.g., ticks, fleas, lice, mosquitoes, phlebotomine sand flies, and triatomine bugs). The CVBD-causing pathogens include bacteria, protozoa, helminths, and viruses. Most of these pathogens can infect both dogs and cats, potentially causing a wide spectrum of clinical signs, from localized skin lesions to life-threatening disease.

The prevalence of CVBDs is disproportionally high in tropical regions, including in LATAM [1,2,3], where these diseases are strongly tailored to poverty, poor education and lack of prevention [4,5,6]. Herein, I discuss some challenges and opportunities for tackling CVBDs in LATAM.

Challenges for tackling CVBDs in LATAM

The diversity of pathogens causing CVBDs in dogs and cats in LATAM is impressive. These pathogens are transmitted by one or more species of arthropod vectors, which may further complicate the epidemiological picture and affect the effectiveness of control measures. Co-infections by multiple vector-borne pathogens are very common in areas where CVBDs are endemic.

The burden of CVBDs is heavier on people and animals living in suburbs and rural areas, with limited access to veterinary services. Indeed, many dog owners in LATAM cannot afford the costs of preventatives, diagnostics and treatments. Local legislation and lack of registered products for particular CVBDs may also impede veterinary practitioners from recommending first-line treatments for their clients. In some particular cases, the off-label use of alternative protocols (e.g., slow kill protocol for adulticide heartworm treatment) may be the only available option.

In LATAM there is a huge population of stray dogs and cats. Population control programs are inefficient or virtually inexistent in many regions. Furthermore, many owned dogs and cats have free access to the streets and actually live as community animals [7]. Hence, they are also exposed to many other diseases (e.g., common viruses, especially in young, unvaccinated animals) and health risks, such as fights, poisoning, and running over.

Opportunities for tackling CVBDs in LATAM

Besides their clinical significance for dogs and cats, some vector-borne pathogens are zoonotic. This may be an opportunity to increase public awareness about these diseases and how they can be prevented. Educational campaigns promoted by local veterinary councils, the government, ONGs or the private sector should be encouraged. These initiatives should also focus on the importance of responsible ownership and populational control of dogs and cats.

Some countries in LATAM have well-established control programs for diseases like leishmaniasis and Chagas disease, but other CVBDs of zoonotic concern (e.g., heartworm) [8] are often neglected. Considering the high incidence of mosquito-borne viruses (e.g., dengue, zika and Chikungunya) in LATAM, control programs focused on mosquito-borne diseases could also include heartworm in their agenda. This could be a great opportunity to approximate veterinary practitioners from public health workers, ultimately to unify and optimize the efforts against this group of diseases.

Public veterinary hospitals and clinics can provide accessible veterinary care to pet owners that cannot afford veterinary services otherwise. This is already a reality in some large cities (e.g., Recife, Brazil) in LATAM, but should be expanded to smaller cities and remote rural areas.

Concluding remarks

Tackling CVBDs in LATAM has inherent challenges, which have cultural, political and economic roots. Public-private partnerships is key to reduce inequalities and make veterinary care more accessible to everyone. Education is key to controlling CVBDs in LATAM. Dedicated guidelines elaborated by expert groups (e.g., Tropical Council for Companion Animal Parasites - TroCCAP) [9] are pivotal for the continuous education of veterinary practitioners in LATAM. Local, hands-on workshops for veterinary practitioners are instrumental to increase the detection of CVBDs and to improve their management.

More accurate and affordable point-of-care diagnostic

tests against neglected CVBDs (e.g., Chagas disease) [10] are also pivotal to increase the recognition of these diseases, especially in remote rural areas, where they are usually more prevalent and where pet owners are usually less assisted.

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MOVING FROM COMPASSION FATIGUE TO COMPASSION RESILIENCE

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Upon graduating from veterinary college, Debbie spent the first years of her career in general and emergency practice, upon which she founded the Pioneer Pet Clinic, a family-centered companion animal hospital in Kitchener, Ontario. With room in her heart for helping children as well as animals, she began fostering with Family and Children's Services. Introduced to, and inspired by, an alternate helping profession, she returned to school to study social work. She then merged her expertise to complete a PhD in Epidemiology, specializing in veterinary-client-patient communication.

Since then, Debbie has held various positions within industry, wherein she developed a fully accredited, evidence-based veterinary continuing education program called The Social Side of Practice. This program specializes in the areas of veterinary wellness, communication, teamwork, organizational culture, and leadership. She has given over 400 presentations internationally and published close to 30 articles in veterinary and social work journals as well as two book chapters. She volunteers with a variety of organizations, from local charities and non-profits to international professional associations. Debbie is a caregiver, healer, and teacher by nature, with a passion for supporting the health and welfare of people and animals.

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ABSTRACT BODY:

MOVING FROM COMPASSION FATIGUE TO COMPASSION RESILIENCE

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Understanding Compassion Fatigue

Compassion fatigue is understood as the deep physical, emotional, and spiritual exhaustion that can be felt from working day to day in an intense caregiving environment. There are two ways of understanding it. The first is conceptualizing it within the *Professional Quality of Life* scale, a survey which measures the positive and negative aspects of helping others. The positive aspects lead to compassion satisfaction, while the negative aspects lead to compassion fatigue, conceptualized as the culmination of burnout and secondary trauma. This is the traditional way of understanding compassion fatigue.

A newer perspective is seeing it not as *compassion* fatigue, but as *empathic distress* fatigue. Research using fMRI neuroimaging has shown that the functional areas of the brain for compassion (empathic concern) and empathic distress are different. Based on this, it suggests that it is empathic distress, *not* compassion, that causes fatigue. The key message is that "compassion does not fatigue." Rather, it rejuvenates, and motivates even greater caring, and in this enables us to feel joy in our work (i.e., compassion satisfaction).

There are many causes of compassion fatigue, including (1) working in a hospital setting, exposed to illness, disease, accidents, death and dying (i.e., the "sick" aspects of care), (2) work-related stress caused by long work hours, excessive workload, emergency on-call, work-home interference, under-staffing, lack of resources, low remuneration, low profit margins, financial pressures, cyberbullying, difficult relationships, conflict, and administrative duties, (3) the provision of care to animals *and* their owners, (4) medical care decision-making, ethical conflict and moral di/stress, (5) euthanasia and exposure to death, (6) the need to keep up with new skills and technology, (7) high levels of job complexity and concern about maintaining expertise, (8) relative social and professional isolation, and lastly, (9) who we are and how we carry ourselves in practice.

The classic symptoms of compassion fatigue are a decline in the ability to feel pity, sympathy and empathy, and thus, act from a place of compassion; and profound physical, emotional, and spiritual exhaustion. While emotional emptiness interferes with the ability to care and provide care, the across-the-board fatigue can significantly affect thinking, feeling, and behavior — the keystones of day-to-day functioning. Over time, the declines in the ability to feel, think clearly, use good judgment, and make decisions can lead to a negative self-image and feelings of inadequacy, helplessness, and hopelessness. The consequences of these changes are significant.

The *physical* consequences include various psychosomatic conditions (e.g., headaches, gastrointestinal upsets, and chronic pain and fatigue); increased susceptibility to illness (with high circulating cortisol); and over the long term, cardiovascular disease, obesity, diabetes, various gastrointestinal conditions and immune dysfunction. The *psychiatric* consequences include mood disorders (e.g., anxiety and clinical depression), dissociative

disorders, addictions, eating disorders, and personality disorders.

Compassion fatigue can have professional as well as personal consequences. It is typically accompanied by declines in work performance, reduced quality of patient and client care with less-than-optimal outcomes, a sense of dread working with certain patients/clients, and medical oversights or errors. Many will find professional life no longer rewarding, which can lead to substance use, addictions, premature job changes, and leaving the profession altogether.

Compassion fatigue can also have organizational consequences. It can negatively impact practice culture, reduce client satisfaction, and make it impossible for practices to sustain profitability or realize their growth potential.

Moving to Compassion Resilience

Although resilience can be seen as a capacity or outcome, it's most commonly understood as a process by which we draw on our resources and use specific strategies to navigate challenges and work toward adaptive outcomes. Although some may think of resilience as a personality trait or natural tendency, it's actually a learned competency, one that – with awareness, intentional pursuits, and lived experience – can be strengthened over time. There are many ways to build resilience. These can be thought of as opportunities to enable you to thrive, feel alive, provide the very best of yourself – and hold onto the heart of what drew you to the profession to start with.

- Personal Approaches

Practice Self-care. Selfcare is not just the cornerstone for alleviating compassion fatigue, but also a professional, ethical responsibility. It follows the principle: *To care for others, one has to first care for the self.* Selfcare necessitates self-stewardship, taking personal responsibility for, and investing in, one's body, mind, and spirit, to wholistically sustain health and wellbeing. It involves learning – and regularly practicing – what works to sustain your energy, buoyancy, and hope. There needs to be balance between the activities of life that fatigue and those that refresh.

Practice mindfulness and self-compassion. Research has shown that mindfulness and self-compassion, both drawn from Buddhist philosophy, enhance resilience. They're associated with less stress, anxiety, depression, and burnout, thereby reducing the risk of compassion fatigue. Mindfulness can be enhanced through mindfulness training, a variety of methods that guide the mind to stay focused in the moment. Self-compassion can be enhanced through various exercises and guided practices. Both mindfulness and self-compassion can bring you an inner calmness and groundedness to better manage the stressors of practice.

Cultivate compassion for others. Although compassion is an intrinsic aspect of our nature, described as an evolutionary instinct of sorts, it can also be taught, learned, and nurtured, or in other words, cultivated through training. Compassion training makes use of Buddhist-derived meditative techniques. The most popular techniques are compassion meditation, which focuses on cultivating affective empathy for the suffering of others, and loving kindness meditation, which focuses on cultivating a feeling of love for all beings. The many benefits, which include reducing empathic erosion, improving empathic accuracy, and deepening compassion, all serve to enhance compassion resilience.

Grow compassion satisfaction. The positive feelings of reward, fulfillment, and accomplishment with compassion satisfaction can offset the negative feelings that characterize, and contribute to, compassion fatigue. You can grow your compassion satisfaction by increasing your awareness of (i.e., noticing) the hope, joys, and rewards, and sense of meaning and purpose within your work; the relationships that you take pleasure in, with coworkers, clients, and patients; and the many differences, great and small, that you make every day. From there, you need to intentionally lean into them, savor them, taking the time to genuinely appreciate them.

Sustain your social support. Social support is crucial to managing work stress. It is an essential element of resilience. Research has shown that high social support reduces stress, anxiety, and depression; and has protective and buffering effects on both mental and physical illness. We tend to assume that others will naturally step in to provide the support needed, and when they don't, we find ourselves wanting, let down, and lonely. With intention, you can carefully craft a social safety net, one that will offer you the sustenance you know will help you stay well.

- Organizational Approaches

Reduce workplace stress. Open a conversation with your team about the opportunities to reduce the stress in your workplace, asking about workloads, working hours, and work processes. We often get into patterns of working that are so ingrained that we can't imagine doing anything differently! Another way to reduce stress is to encourage "power with" vs. "power over" approaches to leadership, moving away from the top-down, directive, controlling style to bringing people together within a more democratic, facilitative,

and participative style, enabling voices to be heard. Promoting continuing education can also reduce stress, especially in job-related skills where the lack of skills is generating stress. Professional development is integral to job endurance.

Minimize euthanasia stress. Euthanasia-related stress can be minimized by creating criteria under which euthanasia is recommended "in advance," identifying the situations within which euthanasia would be refused. It can also be minimized by dividing the euthanasia workload, in this way dividing the stress, distress, and grief. The stress can further be divided through debriefing with trusted others who "understand," who "get it." Informal sharing can be helpful to reduce the emotional load. Anything that can be done, such as providing incremental care and referring to community programs that support accessible care, to avoid unnecessary euthanasia, is also helpful. Access to care is something we all need to attend to.

Enhance social support. Interventions that promote connectedness – such as team building, peer support, debriefing, and regular check-ins – can enable you and your coworkers to better work together and safely discuss the impact of your work on your lives. Being able to share the difficult experiences permits these experiences to be put behind you, and in this, ameliorate the stressful effects. It also makes them "normal," and reduces the sense of isolation that we so often feel, thinking we're the only ones who feel that way.

Summary

Compassion fatigue can impact everyone and everything in practice. Please learn all that you can and move you and your team towards compassion resilience. Start small, think big, and take it forward. It's well worth the effort.

References available on request.

MANEJO DE GATOS CON DOLOR CRÓNICA OSTEOARTICULAR

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ABSTRACT BODY:

Introducción y presentación general

En los últimos 15 años hubo un gran avance en la comprensión, reconocimiento, prevención y tratamiento del dolor en gatos. Al evaluar el dolor crónico, sin duda los procesos degenerativos osteoarticulares fueron los de mayor volumen de investigación y actualización sobre gatos. A los procesos degenerativos osteoarticulares se consideran la causa más frecuente de dolor crónico (mala adaptación) en gatos, con sus primeras menciones en la literatura hay unos veinte años. Hoy se sabe que este grupo de artropatías degenerativas son descritas en todos los grupos de edad, pero con un aumento sustancial en su prevalencia radiográfica en gatos mayores de 10 años. Las presentaciones clínicas del dolor osteoarticular en gatos son diferenciadas y insidiosas, con frecuencia (erróneamente) asignadas al proceso de envejecimiento. Cambio en hábitos de eliminación, dificultad para subir/bajar escaleras, cambio en los saltos hacia arriba/abajo, la reducción de las carreras y del hábito de cazar objetos están entre las principales modificaciones de la movilidad en gatos con enfermedad articular degenerativa. Se dispone de cuestionarios validados científicamente para evaluar la presencia de dolor osteoarticular, que deben incorporarse a la rutina clínica, especialmente para pacientes de edad avanzada. El tratamiento analgésico debe, siempre que sea posible, incluir estrategias farmacológicas y no farmacológicas, además de un manejo amigable (Cat Friendly). Tengas en mente que un abordaje analgésico multimodal es siempre más interesante y indicado para el mejor manejo del dolor.

Estrategias farmacológicas Fármacos antiinflamatorios no esteroideos (AINE)

Los AINE fueron la primera categoría farmacológica ampliamente estudiada y validada para la analgesia en gatos portadores de la enfermedad articular degenerativa (EAD). Estos fármacos inhiben la expresión de la enzima COX, reduciendo la liberación de mediadores inflamatorios. Meloxicam y robenacoxib son los AINE más adecuados para este manejo analgésico en gatos, ya que su metabolismo se realiza por oxidación (y no por glucuronidación, como ocurre con otros AINE). Los AINE se pueden utilizar en gatos con dolor osteoarticular crónico, incluidos los gatos con enfermedad renal crónica asociada, siempre que estén estables y se controlen cuidadosamente. Estos animales deben someterse a un examen físico completo, además de una evaluación hematológica y bioquímica completa, medición de la presión arterial, T4 total y análisis de orina. Los animales correctamente evaluados y con enfermedad renal estable, siempre que estén hidratados y con apetito adecuado, también pueden utilizar a los AINE. Las dosis utilizadas para tratar la EAD son diferentes de las que figuran en los prospectos para traumatismos agudos. Fármacos como el meloxicam tienen una indicación de dosis variable entre 0,01-0,03mg/kg SID o en días alternos (EDA) (siempre con cálculo en base a masa magra). Robenacoxib puede ser utilizado en dosis variables entre 1-2mg/kg cada 24-72 horas. Se deben buscar las dosis más bajas que son efectivas para el paciente.

Gabapentina

La gabapentina actúa bloqueando los canales de calcio, reduciendo la excitabilidad neuronal. Es un fármaco muy seguro y presenta varias indicaciones en casos de dolor crónico en humanos, factor que trae la recomendación de su uso en gatos con dolor osteoarticular crónico, convirtiendo a la gabapentina en el fármaco más prescrito para este fin por los veterinarios norteamericanos. Un solo estudio probó su eficacia para gatos con dolor osteoarticular crónico, aumentando la actividad de los pacientes en la percepción del propietario, pero causando disminución de la actividad motora (posiblemente debido a la sedación). Las dosis de gabapentina recomendadas oscilan entre 5 y 10 mg/kg BID-TID. Este es un fármaco muy utilizado en la rutina del autor.

Amantadina

La amantadina es un antagonista de NMDA, indicado en el tratamiento de la sensibilización central. Este fármaco está indicado a auxiliar en protocolos analgésicos, con sinergismo y potenciación de otros fármacos analgésicos. Así como con la gabapentina, en un solo estudio se probó la amantadina, con un aumento en la actividad de los animales en la percepción de los propietarios, pero una disminución en la actividad motora. Las dosis recomendadas de amantadina son variables entre 3-5 mg/kg SID a BID.

Tramadol

Tramadol actúa uniéndose a los receptores μ, además de inhibir la recaptación de serotonina y norepinefrina. Su uso mejora el nivel de actividad y la calidad de

vida, siendo utilizado principalmente en casos de exacerbación del proceso crónico. Las formulaciones veterinarias palatables son más adecuadas, dado el sabor amargo de la fórmula destinada a humanos. Las dosis recomendadas son de 1-2 mg/kg BID. Las dosis superiores a estas tienden a causar excitación y euforia.

Anticuerpos monoclonales anti-NGF

El factor de crecimiento neural (NGF) es una neurotrofina con un papel central en la sensibilización de los nocioceptores presentes en una gran variedad de procesos dolorosos agudos y crónicos, incluida la osteoartritis. Recientemente se aprobó el uso de anticuerpos monoclonales anti-NGF para gatos en algunos países europeos, Canadá y EE. UU. Se considera una terapia innovadora, con una excelente respuesta en la mejora de la movilidad y actividad de los pacientes, en la percepción del propietario.

Otras clases farmacológicas

Se menciona el uso de condroprotectores y omega-3, en base a sus propiedades farmacológicas. Sin embargo, faltan las evidencias científicas que apoyen su uso como terapia analgésica en casos de enfermedad osteoarticular crónica.

Estrategias no farmacológicas

El uso de acupuntura, láser y fisioterapia están indicados después de una cuidadosa evaluación individual, recordando la importancia de que la analgesia se realice ANTES de los procedimientos de manipulación física.

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NEONATOLOGÍA CANINA Y FELINA

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Curriculum Vitae

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Realiza su actividad clínica en la Fundación Once del Perro Guía desde 1990, donde lleva la dirección del Centro de Cría de la Escuela de Perros Guía de la Once en los apartados de reproducción, neonatología y pediatría; además trabaja como Especialista en Reproducción en el Hospital Clínico Veterinario de La Universidad Alfonso X El Sabio y en la Clínica Veterinaria Plutos de Madrid.

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Ha realizado Publicaciones en diversas revistas nacionales e internacionales destacando el Premio Científico Miguel Luera 2003 por el articulo Colposuspensión para prevenir la incontinencia urinaria en perras castradas.

Ha escrito el libro Reproducción y neonatología felina y canina publicado en el 2011

Ha coordinado y escrito el libro, Medicina Pediátrica en Pequeños Animales publicado en 2012 por Editorial Servet

Es miembro de la EVSSAR y miembro del GERPAC.

Ha sido miembro del Comité científico de AVEPA en el periodo 2002 al 2010 en la Especialidad de Reproducción, tesorero del Grupo de Estudio en Reproducción y Pediatría de Animales de Compañía (GERPAC), Vocal de AVEPA por Madrid y coordinador de vocalías de la Junta de AVEPA. Ha realizado los capítulos de reproducción en el Manual de medicina interna de pequeños animales publicado por Improve en 2015.

Ha sido ponente en el Congreso AVEPA-SEVC realizado el 20-22 de Octubre de 2017 en Barcelona.

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ABSTRACT BODY:

Introducción

El periodo neonatal dura en el perro desde el nacimiento hasta las tres semanas de edad, mientras que en el gato es mas corto y dura aproximadamente 2 semanas. El estado de salud del cachorro cuando al nacimiento, va a depender de numerosos factores, entre lo que figuran el potencial genético de los padres, la salud de la madre, la alimentación que ha tenido la madre durante la gestación y si ha sido sometida o no a determinados tratamientos farmacológicos y finalmente y muy importante a como ha sido el desarrollo y la asistencia al parto, siendo el parto distócico uno de los problemas principales que van a afectar al neonato en el futuro

Fisiología neonatal.

Tenemos que tener en cuenta que el neonato tiene una inmadurez fisiológica elevada.

En los primeros días de vida, el cachorro tiene una anemia que podemos considerar fisiológica, en la primera semana los valores de glóbulos rojos, están entre 4,6 x 10^6

Durante la primera semana y un poco mas bajos durante de la 2ª semana, (3,9x 10^6), aunque el hematocrito se mantiene en unos valores similares a los del adulto, 30-40%, En el caso del gatito, el hematocrito está entorno al 35% y el número de glóbulos rojos está alrededor de 5,2 x 10^6

El 82% del cuerpo del neonato es agua y la función renal es inmadura por lo que es importante un grado de al menos un 60% de humedad ambiental para evitar la deshidratación. Los neonatos durante los primeros días tienen los reflejos neuromusculares poco desarrollados[123], por lo que no son capaces de alejarse del exceso de calor ni de evitar las zonas demasiado frías, a esto hay que sumarle que no tienen reflejo de temblor el cual no aparece hasta el sexto o séptimo día y que no tienen grasa subcutánea, por lo que el riesgo de hipotermia es muy elevado.

Al carecer de mecanismos termorreguladores hasta aproximadamente las cuatro semanas de edad, la temperatura ambiente debe estar suficientemente alta, entre

27-32 ºC para facilitar el mantenimiento de la temperatura corporal al menos en 36 ºC. La hipotermia afecta negativamente principalmente a la inmunidad y a la digestión. Los neonatos con temperaturas corporales menores a 35 ºC deben ser calentados lentamente antes de alimentarlos[4].

Con respecto a la orina ,está mas diluida y la glucosuria es un hallazgo frecuente en el análisis de orina[4]. La recogida de la muestra se puede realizar en el neonato

suavemente limpiando el prepucio o la vulva con una torunda de algodón esteril para estimular la micción.

Los neonatos son muy susceptibles al estrés ambiental, a las infecciones, y a la desnutrición.

Principales patologías del neonato.

1.- Patologías del neonato propiamente dichas

2.- Patologías del neonato derivadas de la madre

1.- Patologías del neonato propiamente dichas

Hipoxia neonatal

Es en la primera semana de vida, donde el cachorro presenta mayor riesgo de hipoxía[12]

Etiología.

Está directamente relacionada con la intensidad de la primera inspiración al nacimiento. Cuanto mas intensa sea la primera inspiración, menos serán los alvéolos pulmonares que permanezcan colapsados o no funcionales. Existe una relación directa entren la hipoxia y la inmadurez pulmonar

Sintomatología.

Hay un aumento de frecuencia respiratoria, superior a 40 respiraciones por minuto y disminución de la frecuencia cardiaca a 80-100 ppm, finalmente existe dificultad respiratoria, apnea y cianosis, los neonatos con estos síntomas son rechazados por la madre[1].

Tratamiento.

Debemos realizar una limpieza de vías aéreas mediante la succión con una jeringuilla de la zona faringea y laringea, tenemos también que estimular a los cachorros para activar la respiración mediante masaje torácico y también frotar el tórax del cachorro con una toalla suave y caliente, aplicando presión positiva de oxigeno con una mascarilla adecuada a la vez que presionamos el balón,

Hipotermia (Enfriamiento)

Etiología

El neonato no alcanza la tª basal de 38,5º C hasta las 4 semanas, siendo su temperatura rectal al nacer de 35,5 ºC. Camadas poco numerosas, de dos o 3 cachorros son mucho mas sensibles al enfriamiento que las camadas muy numerosas.

Síntomas

Pérdida del reflejo de succión, aumento de la frecuencia respiratoria que se acompaña de una disminución de la frecuencia cardiaca, hipoxia tisular y acidosis metabólica producida por la disminución de la ingesta y finalmente el cachorro disminuye su actividad para entrar en shock, coma y muerte

Tratamiento

Calentamiento progresivo lento en 1 a 3 horas, con botellas de agua caliente, con manta eléctrica a baja potencia o incubadora.

Profilaxis

La zona de la paridera deberá tener un control exhaustivo de la temperatura y no bajar de los 30-31ºC durante por lo menos los 8 primeros días, a partir de la segunda semana estabilizaremos la temperatura a 23º y la mantendremos así hasta la 4º semana.

Deshidratación

Se produce por una disminución de la ingesta de leche, o por una humedad ambiental por debajo del 35%, los síntomas principales son pérdida del reflejo de succión, lloriqueos que van en aumento, disminución de la vitalidad y finalmente enfriamiento[1]. Hay persistencia del pliegue cutáneo. Hay aumento de la frecuencia cardiaca y disminución en la producción de orina

El diagnóstico lo podemos realizar controlando la curva de peso, de forma que deberemos pesar al cachorro diariamente. El cachorro disminuye su peso al nacimiento en las primeras 24 h, debiéndolo recuperar a las 48 h de vida, a los 10- 14 días debe pesar el doble del peso con el que nació, y la ganancia diaria media es de 2-3 gr por Kg de peso del adulto[1,2].

El tratamiento se basa en rehidratar al cachorro con biberones de agua azucarada y si esto no es suficiente recurriremos a la lactancia artificial o incluso a la fluidoterapia, 45 ml/Kg en bolo.

Hipoglucemia.

Se produce en casos de camadas muy numerosas, en madres con mastitis, agalactia, con poca leche o en caso de estrés y en cachorros huérfanos. Los valores normales de glucosa varían de 80 a 140 mg/dl, la hipoglucemia se establece con valores de glucosa por debajo de 50 mg/dl

El cachorro presenta además hipotermia y deshidratación, vocalización continua, pérdida del reflejo de succión, bradicardia, disnea, y alteraciones de tipo nervioso que comienzan con pequeños temblores e irritabilidad y que continúan con convulsiones, coma y muerte[1]. El tratamiento se basa en la fluidoterapia, (dextrosa al 5 o al 10%)[3] hasta que estabilicemos al neonato, en la lactancia artificial y en caso de criaderos podemos valorar el poner a los neonatos con otra madre que tenga menos cachorros o más producción láctea. Hay que evitar la alimentación oral si la temperatura esta por debajo de los 36ºC

Malformaciones congénitas

Las malformaciones congénitas, pueden tener varios orígenes, y en ningún caso significa que sean heredables, la etiología es variada pudiendo ser de origen alimentario por exceso de vitamina A durante los días 17 a 22 de la gestación dando lugar a paladar hendido, por exceso de vitamina D, que da lugar va un

cierre precoz de las fontanelas o por dar alimentación hipoproteica que puede favorecer la aparición del Síndrome del cachorro nadador.

Las principales patologías congénitas del neonato son, el anasarca, el paladar hendido, la diabetes insípida nefrogénica y el síndrome del cachorro nadador

2.- Patologías derivadas de la madre

Las principales patologías del neonato derivadas de la madre son:

- La carencia de vitamina K y síndrome hemorrágico
- Infecciones bacterianas localizadas
- Onfalitis neonatal
- Piodermitis neonatal
- Oftalmia neonatal
- Síndrome de la leche tóxica
- Septicemia neonatal
- Lesiones producidas por la madre

Onfalitis neonatal

De forma preventiva el ombligo de los recién nacidos se debe tratar con solución de clorhexidina y prevenir la entrada de bacterias ambientales en la cavidad peritoneal (onfalitis-peritonitis)[4,5].

Septicemia bacteriana neonatal

La septicemia bacteriana neonatal puede causar un deterioro rápido, dando lugar a la muerte del neonato si no nos damos cuenta y no la tratamos con rapidez.

Los organismos más frecuentemente asociados con septicemia son: E.coli, estreptococos, estafilococos, y Klebsiella spp[4,5].

Los signos clínicos principales son: disminución de la curva de peso, no maman, hematuria, diarrea persistente, vocalización excesiva, distensión y dolor abdominal, y el temblor de las extremidades.

Tratamiento:

Las cefalosporinas de 3ª generación (ceftiofur sodico) se deben administrar en una dosis de 2.5 mg/kg SC cada 12 h, no más de 5 días[4], y debe ser continuado hasta 48 horas después de desaparecer los síntomas.

Como los neonatos con menos de 48 horas de vida tienen reducidos los niveles de trombina, la administración de vitamina K puede ser útil (0.01 - 1.0 mg SC por neonato[4,5]).

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RADIOGRAPHIC INTERPRETATION OF URINARY TRACT DISEASE

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ABSTRACT BODY:

RADIOGRAPHIC INTERPRETATION OF URINARY TRACT DISEASE

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With the widespread availability of diagnostic ultrasound and computed tomography in veterinary practice, one might question the role of radiographs in the diagnosis and staging of urinary tract disease. However, radiographs are often obtained in the diagnostic workup of patients with abdominal disease and signs such as vomiting and nausea, or if clinical signs are vague. Thus, interpretation of the radiographic appearance of the urinary tract remains an important skill. Importantly, radiographs provide an excellent way of detecting radiopaque urinary calculi and an overall overview of the abdomen including the retroperitoneal and lumbar region, skeletal structures and at least portions of the thorax where important and helpful clues to the patient's problems may be found. Associated disease such as metastatic pulmonary disease, or discospondylitis in patients with urinary tract infection may be identified radiographically. A diagnostic radiographic study of the abdomen should always include both left and right lateral as well as ventrodorsal views of the abdomen. In some animals, compression radiography with a wooden paddle allows displacement of the intestinal tract away from the urinary tract to improve visibility.

Evaluation of the kidneys includes assessment of their size, shape, opacity, margination, and surrounding structures such as the retroperitoneal fat. The normal canine kidney is oval, soft tissue opaque and smoothly marginated. The right kidney is more cranially located than the left and typically located at the level of the last rib. The cranial pole of the right kidney abuts the caudate lobe of the liver and may be poorly visible due to silhouetting with the liver as well as superimposition of other cranial abdominal organs. The left kidney is usually better visible as it is more caudally positioned, usually approximately at the level of L1-3. The left kidney is more loosely attached and can be displaced ventrally in animals with a large amount of retroperitoneal fat. The feline kidneys are more rounded than canine kidneys and tend to be almost at the same level with the right kidney being more caudally positioned than in dogs. They should also have a smooth outline and be predominantly soft tissue opaque, although in some cats, particularly if overweight, a more radiolucent region in the renal hilus may be visible, representing fat deposits. Normal renal size has been described as a comparison with the length of the L2 vertebra. Renal length in dogs ranges between 2.5 and 3.5 times the length of L2. The normal length of the feline kidney lies between 2 and 3 times the length of L2; however, renal length can be increased in intact cats due to hormonal influence. The absolute renal size is not an accurate measurement, and always has to be interpreted in light of other findings comparing left and right.

Changes in renal size are commonly present in renal disease, the most common being reduced renal size in chronic kidney disease. A smaller renal size has been shown in older cats without signs of renal disease, however subclinical disease is so common in the aged feline population that it is difficult to determine if these cats were completely normal or not. Chronic renal disease is also associated with other radiographic findings including irregular shape due to cortical infarcts, and renal mineralizations. Other differentials for bilaterally reduced renal size include congenital anomalies such as dysplasia or hypoplasia. Renal atrophy is seen with chronic, longstanding ureteral obstruction and is usually associated with hydronephrosis, the affected kidney (typically only one, or asymmetric) can thus be reduced or relatively normal in size. Bilateral mild renomegaly may be seen with acute kidney injury, pyelonephritis, amyloidosis, and with congenital portosystemic shunts as well as acromegaly in cats. Acromegalic cats have been shown to have measurably larger kidneys, adrenal glands and pancreas as well as subjectively larger livers than non-acromegalic cats, caused by the anabolic effects of IGF-1. Unilateral mild renomegaly with a normal shape and smooth outline is most commonly seen with compensatory hypertrophy caused by contralateral chronic degenerative or obstructive disease, occasionally also due to subcapsular fluid accumulation (urine, hemorrhage or pus) or renal neoplasia.

Bilateral markedly enlarged kidneys with a smooth outline are seen with renal lymphoma, feline infectious peritonitis, bilateral hydronephrosis or bilateral

perinephric pseudocysts. Hydronephrosis and perinephric pseudocysts are more common unilateral, or at least asymmetrical in appearance. Unilateral severe renomegaly with a smooth outline is most commonly seen with hydronephrosis, renal tumors and subcapsular fluid as mentioned above, or unilateral perinephric pseudocyst. Enlarged kidneys with an abnormal shape and irregular outline increases the concern for a more severe disease process such as neoplasia, abscessation, granuloma or hemorrhage. Renal cysts, however can result in markedly irregular and enlarged kidneys. Bilateral enlargement is typically present with polycystic kidney disease whereas a idiopathic renal cyst may be unilateral. Bilateral renal neoplasia is most commonly due to metastatic or multicentric disease (hemangiosarcoma, histiocytic sarcoma). Primary renal tumors such as adenocarcinoma are typically unilateral in occurrence, although they can metastasize to the contralateral side. Irregular renomegaly in a juvenile patient may be seen with nephroblastoma which can manifest in the kidney or the spine.

Changes in renal opacity should also be noted when examining abdominal radiographs. Decreased opacity in the feline renal pelvis is a common finding as mentioned above, due to deposits of fat. Focal increases in renal opacity is commonly seen with nephrolithiasis. Nephrolithiasis or nephrocalcinosis can manifest in different ways. Occasionally, calculi are visible outlining the renal pelvis whereas more commonly mineralizations tend to extend along the renal pelvic diverticula, leading to a striated appearance radiating to the periphery from the renal pelvis. Focal mineralizations in the region of the renal pelvis should be differentiated from proximal ureteroliths. Slightly oblique ventrodorsal projections or compression views can be helpful to determine the location of a mineralized focus within or outside the renal silhouette.

Evaluation of the perinephric tissues, as well as the entirety of the abdominal radiographs should always be performed and taken into account when forming differential diagnoses. The retroperitoneal fat should normally have a mostly uniform fat opacity, the only discernible structures including the large abdominal vessels, the aorta and caudal vena cava, and some of their branches particularly the circumflex iliac arteries which manifest as round or ovoid soft tissue nodules superimposed with or just ventral to the aorta, usually at the level of L6. These should not be confused with ureteral calculi. Perinephric retroperitoneal effusion causes increased opacity and a striated appearance of the retroperitoneal space. Distended ureters can occasionally be seen radiographically, and mineralizations in the region of the bladder trigone and/or prostate may indicate presence of a bladder or prostatic mass that consequently could lead to ureteral obstruction and hydronephrosis.

The normal urinary bladder is of uniform soft tissue opacity and visible cranial to the pubis, whereas the cranial extent of the bladder depends on the volume of urine. Overdistension can be seen with urethral obstruction, lack of recent voiding, polyuria/polydipsia and neurologic disorders. Radiopaque urinary calculi are located in the lowest, gravity dependent portion of the bladder which is the center in both lateral and VD radiographs. Occasionally, adhered calculi are seen more peripherally. Radiopaque calculi located at the caudodorsal aspect of the urinary bladder may be located within the ureteral papilla. Increased radiolucency that is centrally located typically originates from procedures such as cystocentesis or catheterization. Gas located peripherally along the bladder wall is indicative of intramural gas as with emphysematous cystitis.

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THE DIAGNOSTIC AND TREATMENT CHALLENGE OF AN ADDISONIAN CRISIS

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ABSTRACT BODY:

Hypoadrenocorticism is an uncommon disorder in dogs and rare in cats. Based on its origin, it can be characterized by a dual deficiency of mineralocorticoids and glucocorticoids, or just of glucocorticoids.

Aldosterone, the main body mineralocorticoid, has the specific vital action of keeping sodium (Na), potassium (K), and body water homeostasis. It plays the important role of regulating renal handling of sodium (1). Aldosterone is also the most important hormone affecting renal potassium excretion (2) with lesser actions in the intestinal mucosa, salivary glands, and sweat glands (3). Aldosterone primary site of action is the cortical collecting ducts. It stimulates the insertion of epithelial sodium channels in the luminal surface of the cortical collecting duct cells and the activity of the sodium-potassium ATPase pump on the basolateral side of the same cells. Therefore, aldosterone's main effects involve reabsorption of Na and chloride (Cl), and concomitant excretion of K+ and hydrogen (H+).

Water absorption and expansion of the extracellular fluid volume (ECFV) occur secondary to retention of Na (4). Aldosterone deficiency consequently reduces ECFV; reduced ECFV stimulates antidiuretic hormone activity in the distal collector and hypo osmolar water retention further worsening hyponatremia.

With a dose dependent effect, cortisol influences almost every tissue in the body (5). Cortisol increases vascular sensitivity to catecholamines and helps to maintain vascular tone, vascular permeability, and endothelial integrity. It also aids in maintaining blood pressure, water balance, and vascular volume, particularly in the dog (3). By increasing lipolysis and gluconeogenesis while decreasing peripheral glucose utilization glucocorticoids help to preserve normoglycemia in the fasting animal (5). Cortisol suppresses inflammatory responses and has catabolic effects on connective tissue, muscle, and bone (3). Finally, cortisol stimulates erythrocytosis and counteracts stress (1,3).

Clinical Signs

Addison's disease causes vague and nonspecific clinical signs that can be attributed to multiple body systems and diseases, including gastrointestinal disease, renal failure or neurological disease. Clinical history will point out to waxing and waning episodes, PU/PD and GI episodes (emesis, diarrhea and melena), lethargy, weakness, collapse and sometimes seizures, which could be all resolved by proper fluid therapy (5). However, the hydroelectrolytic disturbances, as well as the consequences of an inadequate therapy could put at risk the patient's life. Severe hypovolemic shock with bradycardia should prompt the suspicion of a potential hyperkalemia.

Blood work will typically show hyponatremia and hyperkalemia, with hypochloremia. The normal sodium to potassium ratio lies between 27:1 and 40:1; ratios Na/K < 27:1 is suggestive of primary hypoadrenocorticism, while patients with ratios < 20:1 are highly likely to have the disease (1,5). Ratios < 15:1 strongly suggest hypoadrenocorticism rather than other disorders that can cause the ratio to decrease, although the diagnosis still must be confirmed with an ACTH stimulation test (5).

Osmotic shifts secondary to hyponatremia could result in neurological signs (coma, seizures, stupor) before and/or during the treatment.

Hypoglycemia, mild to severe hypoalbuminemia and mild to moderate increases in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) may be also present and prompt the differential from liver disease and even leptospirosis in the azotemic patient.

The total white blood cell count generally remains in the normal range, but it may vary from low normal to mildly increased (5). A mildly increased white blood cell count may result from concurrent infection. Although it is not a consistent feature of Addisonian patients, lack of a stress leukogram provides a valuable clue in identifying these suspects. Normal to increased eosinophils and/or lymphocytes in a sick dog should prompt the clinician to consider Addison's disease particularly in glucocorticoid deficient dogs (5).

Pre-renal azotemia and mild to severe acidosis, results from decreased renal perfusion and decreased glomerular filtration rate (GFR) and is present in most (66% to 95%) dogs with primary hypoadrenocorticism at the time of initial diagnosis (1,5). An increase in BUN in proportion to creatinine may result from gastrointestinal bleeding. Hyperphosphatemia secondary to decreased GFR and renal excretion of phosphorus is reported in 66–85% of dogs with primary hypoadrenocorticism (5). Although incompletely understood, hypercalcemia is also seen

in 30 % of patient (1,5). Urine specific gravity < 1.030 secondary to chronic urinary sodium loss and renal medullary washout is a common finding in the Addisonian patient (1,5). Azotemia and dilute urine specific gravity, hyperphosphatemia and hypercalcemia suggest intrinsic renal failure, which is an important differential for hypoadrenocorticism and carries a very different prognosis (5). Prompt improvement of azotemia as a result of the fluid therapy will allow ruling out acute renal failure (ARF) and should also increase the suspicion of hypoadrenocorticism.

Conclusive diagnosis will be achieved by the ACTH stimulating test prior to the administration of exogenous glucocorticoids due to suppression of the pituitary-adrenal axis (7). Basal plasma cortisol and 1 to 2hs (30 min in cats) after cosyntropin (0.5ug/kg/iv in dogs and 125ug/cat/iv) administration is measured. Most dogs with hypoadrenocorticism show baseline cortisol levels and post-ACTH stimulation cortisol levels ≤ 55.2 nmol/L or blunted response post stimulation (6).

A reconstituted vial of cosyntropin can be refrigerated for up to 21 days without loss of efficacy, utmost care should be taken to prevent bacterial contamination during reconstitution (6). Cosyntropin can also be stored as frozen aliquots (–20°C) in plastic syringes for up to 6 months in a frost-free freezer (6).

Treatment

An Addisonian crisis usually refers to an acute worsening of a chronic hydroelectrolytic disbalance. Mild to severe secondary acidosis and hypoglycemia are also common complications. The main objective of the emergency treatment is to prevent the severe sequels of tissue hypoxia, seizures and osmotic shifts over general and central nervous systems as a result of fluid and electrolytes imbalances. Expedite shock resuscitation and adequate correction of seizures, hydroelectrolytic and glucose balance are the corner stone of the therapeutic approach. Due to the possibility of SNC damage, in patients with >48hs of symptom presentation, it is very important to prevent plasmatic variations of osmolality larger than 3-4mOsm/L/h.

The main objective of the treatment is to restore normovolemia and tissue perfusion. Shock resuscitation should be performed within the first 30 min. End goal directed therapy with 10-15ml/kg/30min of lactated ringer should be started prompt. Once the patient is hemodynamically stable, fluid therapy should switch to NaCl0.45%/Dextrose 2.5% to prevent the risk of irreversible osmotic demyelination syndrome (hyponatremia >48 hs). Serum Na increases should be kept <10-12mEq/L/24hs and 18mEq/L/48hs. Should seizures arise, treatment should be far more aggressive considering that the risk of hyponatremic cerebral edema is greater than the potential presentation of a demyelination syndrome.

Generally, fluid therapy will be enough to correct the hyperkalemia. A slow infusion of calcium gluconate 10%, 0.5-1.0ml/kg/iv should be administered to treat severe arrhythmias (6). Dextrose (2.5%), or in combination with insulin 0.2U/kg/iv, followed by dextrose 5% iv CRI during 6 hs has proven to be effective to correct the hyperkalemia and hypoglycemia.

Fluid therapy generally resolves the mild to moderate metabolic acidosis (7). Sodium bicarbonate should only be administered at 1/4 of the calculated deficit over 2 to 4hs if a pH < 7.1 or HCO_3 < 12 mmol/L persists after 1 hour of fluid therapy and only to achieve a pH of 7.2 and the HCO3 to 12 mmol/L.

$NaHCO_3$ deficit = 0.3 × body weight (kg) × (24 - patient's HCO_3)

Dexamethasone 0.25-4.0 mg/kg during the first 3 hours of fluid therapy, will not only decrease the risk for demyelination, but also will address the deficit of glucocorticoids without interfering with the corticotropin test (prednisone, prednisolone, and hydrocortisone do interfere). Mineralocorticoids favor the rapid correction of the Na and they should not be used during the first 48 hours of therapy.

Long-term supplementation should consist of fluorocortisone 0.2mg/kg gradually increasing 0.05-0.1mg/24 hs until electrolytes have reached a stable concentration. Alternative therapy should incorporate DOCP 2mg/kg/25 days + prednisone 0.22mg/kg/day. Recommended DOCP dosages for cats are 12.5mg/cat/day, or methyl prednisolone 10mg/cat/3-4 weeks. During stressful events (surgery, traveling, etc.), prednisone therapeutic dosages should be increased 5-10 times.

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BASIC PERIODONTAL THERAPY

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ABSTRACT BODY:

BASIC PERIODONTAL THERAPY J.Krauss, Consultorio Odontológico Veterinario Dr. Krauss, Buenos Aires, Argentina

Introduction: The basis of periodontal therapy is fundamentally plaque control. The objective of this procedure is not only to clean and polish the teeth, but also to evaluate the periodontal tissues and the entire oral cavity. The concept of oral rehabilitation makes sense. Oral rehabilitation is a procedure that is performed under anesthesia and it is impossible to perform it without this requirement. Therefore, with the patient anesthetized and correctly intubated, we will detail the basic techniques and procedures.

Basic techniques and procedures:

Step 1: pre-surgical examination and consultation. The veterinarian should perform a complete physical and oral examination. The physical examination, in combination with preoperative tests, evaluates general health problems that may exacerbate periodontal disease or compromise anesthetic safety.

Step 2: Chlorhexidine Wash:

The oral cavity is a contaminated area and complete dental cleanings are slightly invasive. This means that dental cleanings often result in transient bacteremia, which is more severe in patients with periodontitis.

Step 3: Supragingival cleaning.

Large accumulations of calculus can be quickly removed with calculus forceps. However, this must be done very carefully to avoid damage to the teeth and gums. Supragingival scaling can be performed through mechanical or manual instrumentation, but is best performed using a combination of mechanical scalers. Mechanical scalers include sonic and ultrasonic types. The power should be set low and adjusted up to the minimum power required. The area of maximum vibration for ultrasonic scalers is 1-3mm from the tip. Do not use the sharp tip of the instrument, but the flat of the instrument, because the tip is not effective for calculus removal and may damage the tooth enamel. It is important to note that standard periodontal tips should not be inserted below the gingival margin. Use the instrument across the entire tooth surface with numerous overlapping strokes in different directions. Keep the instrument moving at all times to avoid damage to the teeth, and do not constantly use more than 15 seconds. The instrument should be kept in constant motion, running slowly over the tooth surface in broad, sweeping, overlapping motions. Plaque can be microscopically present on all tooth surfaces, regardless of whether the tooth appears clean, and therefore every square millimeter of every tooth surface must be treated.

Step 4: Subgingival plaque and calculus removal.

This is the most important step in dental cleaning, as supragingival plaque control is insufficient to treat periodontal disease. Subgingival scaling has classically been done by hand with a curette, but advances in ultrasonic and sonic tips now allow their use below the gingival margin. While some may obtain satisfactory results using ultrasonic scalers alone, it is generally recommended to use a combination of ultrasound (or sonic) and manual instrumentation for best results.

Manual subgingival scaling is a very demanding procedure from a technical point of view and it is suggested that the professional be trained in continuing education programs to hone their skills.

Step 5: Identification of residual plaque and calculation.

After scaling, it is recommended that the teeth be checked with an explorer, looking for rough areas that indicate small areas of dental pathology or residual calculus. Residual plaque and calculus can also be identified by using a plaque disclosing solution or by air drying the tooth surfaces (residual calculus will appear chalky in color).

Step 6: Polish

Dental scaling (both mechanical and manual) will result in microabrasion and roughening of the tooth surface, leading to increased plaque adherence. Polishing smoothes the surface of the teeth, which slows the adhesion of plaque.

Clinics may choose to use a commercially available polishing paste or make their own suspension of finely aerated pumice stone and chlorhexidine solution or water. These can be mixed in a dappen glass for each patient.

The polishing procedure is normally performed with a prophy rubber cup, on a low speed handpiece at a 90 degree angle (prophy angle). The handpiece should be used at a slow speed, not exceeding 3,000 RPM. Faster rotation will not improve the speed or quality of the procedure, and may result in overheating of the tooth. Also, it is important to use an adequate amount of polishing paste at all times. Using the prophy

cup without paste is not only inefficient; It can also overheat the tooth.

Step 7: Sulcal Wash

During the cleaning and polishing steps, debris such as calculus and prophylaxis paste (some of which are bacteria-laden) accumulate in the gingival sulcus (or periodontal pockets). The presence of these substances allows for continued infection and inflammation, and therefore gentle lavage of the sulcus is strongly recommended to enhance healing. Sulcal lavage is performed with a small-bore (22-25) blunt-tipped cannula. The cannula is gently placed in the groove and the solution is injected while slowly moving along the arches. can be used as a 0.12% chlorhexidine solution.

Step 8: Periodontal probing, oral evaluation and dental chart

This is a critically important step for complete dental prophylaxis, but unfortunately it is often poorly performed or omitted altogether. The entire oral cavity should be systematically evaluated using both visual and tactile senses. Periodontal evaluation should begin with pocket depth measurement. The only accurate method to detect and measure periodontal pockets is with a periodontal probe, as radiographs do not always diagnose pockets.

Step 9: registration in the dental file.

All abnormal findings should be recorded on the dental chart. Filling out the dental record is easier and more efficient if it is done with four hands. This means that one person assesses the mouth and dictates the pathology findings to the assistant who records it on the chart. Using the modified Triadan system will also greatly increase the efficiency of this step.

Step 10: Dental X-rays.

When available, dental radiographs should be taken of a minimum of each area of pathology noted on the dental exam. Numerous studies support full-mouth radiographs in all dental patients to further eliminate the chance of missing pathology.

Step 11: Treatment planning.

In this step, the practitioner uses all available information (visual, tactile, and radiographic findings) to determine the appropriate therapy. It is important to take into account the general health of the patient, the interest and willingness of the owner to carry out the care at home and all the necessary follow-up

Step 12 (optional): Application of a dental sealant.

A wax-based sealant that changes the electrostatic charge of teeth has been clinically shown to decrease plaque and calculus. After a prophylaxis, the teeth are dried and the product is applied according to the manufacturer's instructions. Continued applications are made by the client at home on a weekly basis.

A complete dental prophylaxis is a procedure that involves numerous steps.

Each step must be done correctly to achieve a positive result. Subgingival scaling is the most important step in prophylaxis.

REDUCIENDO EL MIEDO, LA ANSIEDAD Y EL ESTRÉS EN DERMATOLOGÍA

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ABSTRACT BODY:

INTRODUCCIÓN

Los problemas dermatológicos y de comportamiento están intimamente relacionados. Durante el desarrollo embrionario, tanto la piel como el sistema nervioso central (SNC) se desarrollan de la misma capa embrionaria, el ectodermo. Una vez que se ha compleado el desarrollo, la piel y el SNC comparten neurotansmisores y moléculas como, por ejemplo, la histamina. El SNC controla el comportamiento. Los problemas de comportamiento, especialmente aquellos que cursan con estrés, afectarán a la salud de la piel y los problemas dermatológicos podrán favorecer la aparición de conductas indeseadas.

Difererentes estudios epidemiológicos en medicina humana aseguran que más de el 70% de los pacientes ingresados por un problema de prurito tienen entre 1 y 6 problemas psiquiátricos. De todos los pacientes que acuden a la visita de dermatología, entre el 30 y 60% presentan problemas psicológicos concomitantes.

En perros, de 21 animales que presentaban pioderma recurrente, 10 tenían problemas de conducta y no mejoraban con el tratamiento solo de dermatología y 7 mejoraron con tratamiento de conducta. Finalmente, otro estudio clásico apunta a que el 70% de los pacientes con dermatitis acral por lamido presentaban, además, problema s de conducta.

PROBLEMAS DE CONDUCTA Y DERMATOLÓGICOS

Clásicamente, para entender mejor la relación entra la piel y los problemas de comportamiento, se han clasificado en cuatro grupos diferentes:

1. Problemas dermatológicos que se ven exacerbados por el estrés. P.e., la atopia.
2. Problemas de conducta que se deben a problemas dermatológicos. P.e. todas las patologías que cursan con prurito, especialmente si es crónico, pero también agudo, pueden causar probelmas de alteración del sueño y otras alteraciones relacionadas con el estrés como la agresividad (por aumento de la irritabilidad del animal).
3. Problemas de conducta primarios que inducen problemas dermatológicos. P.e., la dermatitis acral por lamido.
4. Problemas de hipersensibilidad cutánea que puede cursar con problemas de conducta asociados. Se describe especialmente el síndrome de hiperestesia felino.

Parece claro, por lo tanto, que es necesario prevenir y tratar las situaciones de estrés en los animales en general y, en particular, en aquellos que presentan problemas dermatológicos.

MANEJO DEL ESTRÉS EN LOS ANIMALES

Manejo del entorno

Previsibilidad – Ventajas de poder prever algo malo

Numerosos estudios, especialmente realizados en animales de laboratorio, demuestran que el hecho de poder prever una situación negativa reduce el estrés y sus consecuencias.

En un estudio clásico, se hicieron tres grupos de ratas. Todas ellas eran colocadas en aislamiento en una caja con suelo electrificado por el mismo tiempo. El primer grupo tratamiento recibía una descarga eléctrica siempre después de que se encendiera una luz en la caja, es decir, cuando se encendía la luz, las ratas sabían que iban a recibir una descarga eléctrica. En el segundo grupo, la luz que se encendía no permitía anticipar la descarga eléctrica ya que era un patrón aleatorio y la luz no se encendía cada vez que había una descarga eléctrica (ni viceversa). El tercer grupo era un grupo control. El estudio concluía que el hecho de poder predecir la descarga eléctrica reducía las consecuencias negativas del estrés ya que las ratas del grupo 1 tenían menos úlceras gastrointestinales que las del grupo 2 y 3 y, además, que la concentración de corticosterona de las ratas del shock predecible era igual que la del grupo control a las 24 hs del experimento, mientras que en el caso del grupo “impredecible” la corticosterona estaba significativamente más elevada.

Es importante señalar que estos efectos positivos no se producen porque el estímulo negativo (en este caso el electroshock) deje de serlo, sino porque entre cada evento negativo el animal puede “relajarse” y volver a su estado de estrés basal. Sin embargo, cuando el animal no puede predecir la situación negativa mantiene una situación de estrés más elevada de forma crónica.

Control del estímulo estresante

En unos estudios similares a los anteriores, diversos grupos de investigación en los años 70, demostraron

que “el control” de una situación negativa elimina los efectos negativos de dicho estímulo.

En estos estudios ponían a las ratas en jaulas electrificadas. A un grupo de animales les daban una palanca que permitía parar el electroshock cuando se producía y a otro no. Las ratas que tenían la palanca mostraban menos consecuencias negativas del estrés que las que no podían controlar la situación.

Sin embargo, el resultado más interesante se daba cuando a las ratas que inicialmente tenían la capacidad de controlar la situación las ponían en un segundo estudio. En este, a las ratas que tenían la palanca inicialmente, las dividían en dos grupos: Al primer grupo se le eliminaba la palanca, en el segundo grupo la palanca de control estaba en la jaula, pero no tenía ninguna función (solo estaba presente). En resumen, los dos grupos dejaban de tener el control de la situación, sin embargo, solo el grupo que no tenía la palanca empeoraba los niveles de estrés y sus consecuencias negativas. Lo que concluyeron los investigadores es que lo importante, no solo es tener el control, sino la sensación de tenerlo.

Capacidad de elección

Finalmente, una serie de estudios posteriores, han demostrado que obligar a un animal a realizar una actividad ante una situación de riesgo empeora el estrés. En estos se colocaban a los roedores ante una

Tabla 1. Principales psicofármacos utilizados en medicina del comportamiento.

Objetivo	Fármacos	Ventajas	Inconvenientes
Ansiolíticos de rápido y corte efecto	Benzodiacepinas (Alprazolam, cloracepato, etc.)	Efecto rápido Dosis dependiente Amnesia (evita generalizar la fobia) Efecto reversible (flumazenil)	Efecto paradójico (dosis bajas) Agresividad como efecto adverso en algunos casos (dosis bajas) Toxicidad hepática severa raramente descrita (diazepam) Tolerancia y dependencia
	Trazodona	Sedante Potente ansiolítico en administración única. Pocos efectos adversos	Demasiada sedación en ocasiones Precaución en epilépticos Tolerancia
	Gabapentina	Efecto rápido Muy pocos efectos adversos Efecto analgésico	
	Alpha-2-agonistas (dexmedetomedinay clonidina)	Produce ansiolisis con nula sedación	Presentación veterinaria de dexmedetomedina en gel para absorción en transmucosa oral es de formulación para perros Difícil administración en gatos
	Mirtazapina	Potente estimulante del apetito Pomada transdérmica Utilizado en problemas relacionados con ansiedad y estrés	Pocos efectos adversos.
Ansiolíticos de efecto lento	Antidepresivos tricíclicos (clomipramina, amitriptilina, etc.) – Son fármacos serotoninérgicos poco selectivos.	Efecto ansiolítico Efecto analgésico Antihistamínicos	Efecto anticolinérgico En ocasiones producen demasiada sedación Precaución en animales diabéticos y epilépticos Efecto tras 6-8 semanas de administración
	Inhibidores selectivos de la recaptación de serotonina (fluoxetina, fluvoxamina, sertralina, etc.) – Son fármacos serotoninérgicos muy selectivos.	Efecto ansiolítico Efecto analgésico Menos sedantes porque son más selectivos	Alteración del sueño REM Efecto tras 6-8 semanas de administración Precaución en animales epilépticos

situación estresante. A un grupo les obligaban a hacer ejercicio, mientras que al segundo grupo les daban la opción de poder hacerlo. Los animales del grupo que podían elegir hacer ejercicio (una elevada mayoría de individuos elegían hacerlo) y no eran obligados, tenía muchas menos consecuencias negativas del estrés.

En resumen, para que un estímulo estresante no tenga consecuencias negativas para el animal debe:

- Ser predecible.
- Ser controlable o que el animal creo que lo controla.
- El animal debe poder elegir cómo enfrentarse a esa situación.

Estos tres puntos serán la base del tratamiento de las situaciones de estrés en los animales.

Control farmacológico del estrés

Existen multitud de psicofármacos ansiolíticos que pueden utilizarse para el control de estrés. Se diferencian dos tipos de objetivos cuando se usan psicofármacos ansiolíticos en gatos:

- Ansiolíticos de efecto rápido y corto. El objetivo de estos psicofármacos es el control de situaciones fóbicas agudas y predecibles como, por ejemplo, a ruidos fuertes (tormentas, petardos, etc.), a viajes o, especialmente, a la manipulación veterinaria, entre otros. Existen varios fármacos que pueden utilizarse con este objetivo y cada uno tiene ventajas e inconvenientes (Tabla 1). Es decir, se usan para el tratamiento paliativo de situaciones fóbicas predecibles, no para su tratamiento curativo.
- Ansiolíticos de efecto lento. Se utilizan como fármacos ansiolíticos como coadyuvantes de protocolos de tratamiento en los que el objetivo es *curar* el problema de conducta. Es decir, se usan como coadyuvantes de protocolos de modificación de conducta (o modificación emocional, según el autor), protocolos de enriquecimiento ambiental o cambios en el manejo de los tutores, entre otros. Los más utilizados en son los antidepresivos tricíclicos (clomipramina, amitriptilina, etc.) y los fármacos inhibidores selectivos de la recaptación de serotonina (fluoxetina, fluvoxamina, sertralina, etc.). En los problemas en los que el prurito sea un punto clave, se recomendará usar los antidepresivos tricíclicos ya que tienen un efecto antihistamínico que no tienen los otros grupos.

Las feromonas también se usan con frecuencia para reducir el estrés, pero especialmente en gatos. Otros productos, como los nutracéuticos, han sido propuesto como alternativa en algunos casos, pero todavía faltan estudios que avalen su uso.

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IMMUNOLOGÍA DEL CÁNCER Y SUS AVANCES TERAPÉUTICOS EN LA MEDICINA VETERINARIA,EN DÓNDE ESTAMOS?

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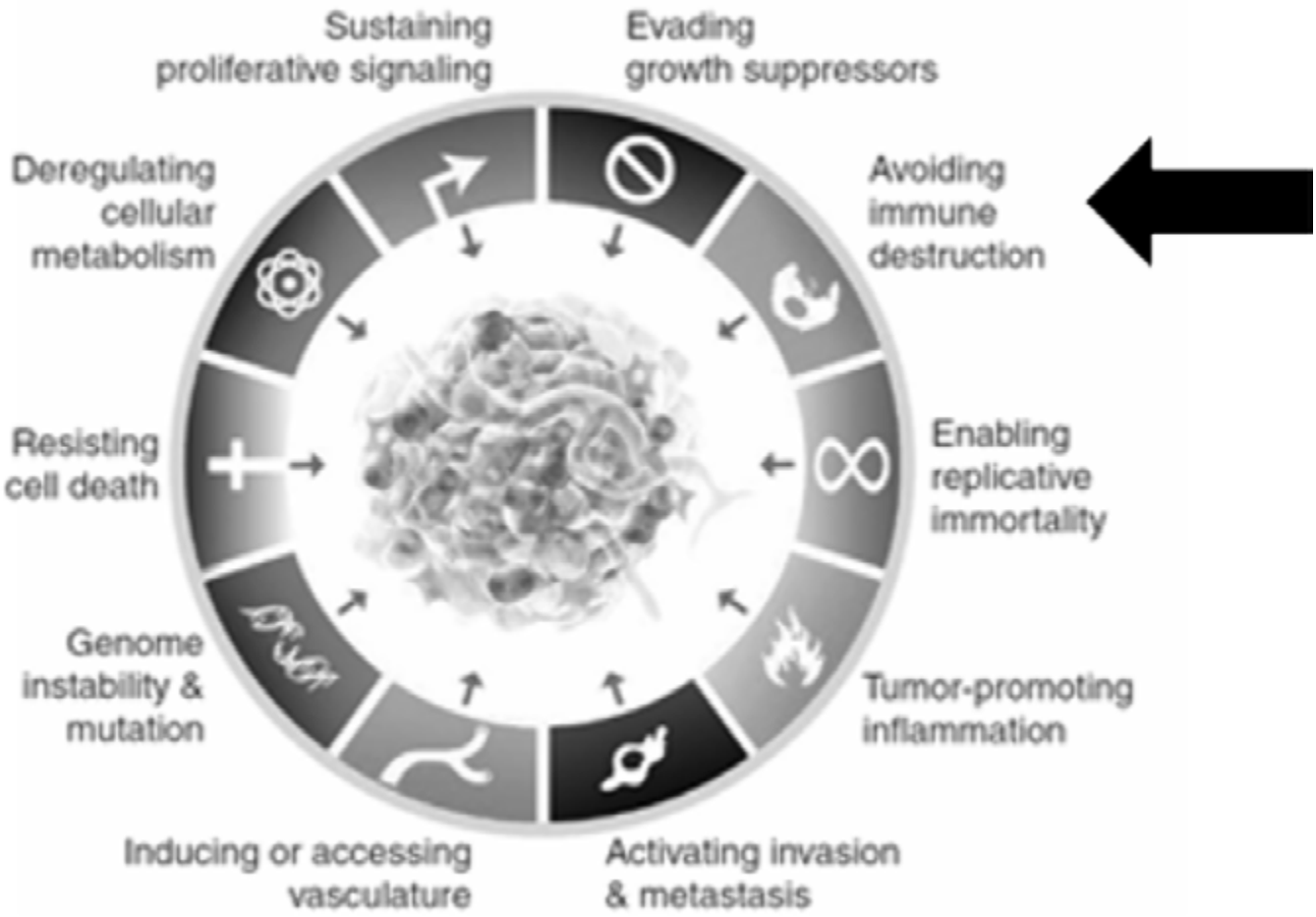
ABSTRACT BODY:

Uno de los desafíos terapéuticos más grandes de la oncología es el control del desarrollo y/o progresión de las metástasis a órganos distantes así como el control de las recidivas locales , gracias a la inmunoterapia se ha permitido tener un antes y después en el control neoplásico de tumores de comportamiento agresivo en estadios avanzados, esto se está logrando a los estudio avanzado del microambiente tumoral y el comportamiento del sistema inmunológico frente a las neoplasias.(1)

Para entender mejor como la inmunoterapia es aplicada en la oncología tenemos que comprender que el objetivo principal de todas las células neoplásicas es sobrevivir, proliferar, migrar; para que esto ocurra las células necesita de herramientas desarrolladas por ellas mismas o adoptan funciones propias del individuo



utilizándolas a su favor, ejemplo los componentes del sistema inmunológico.

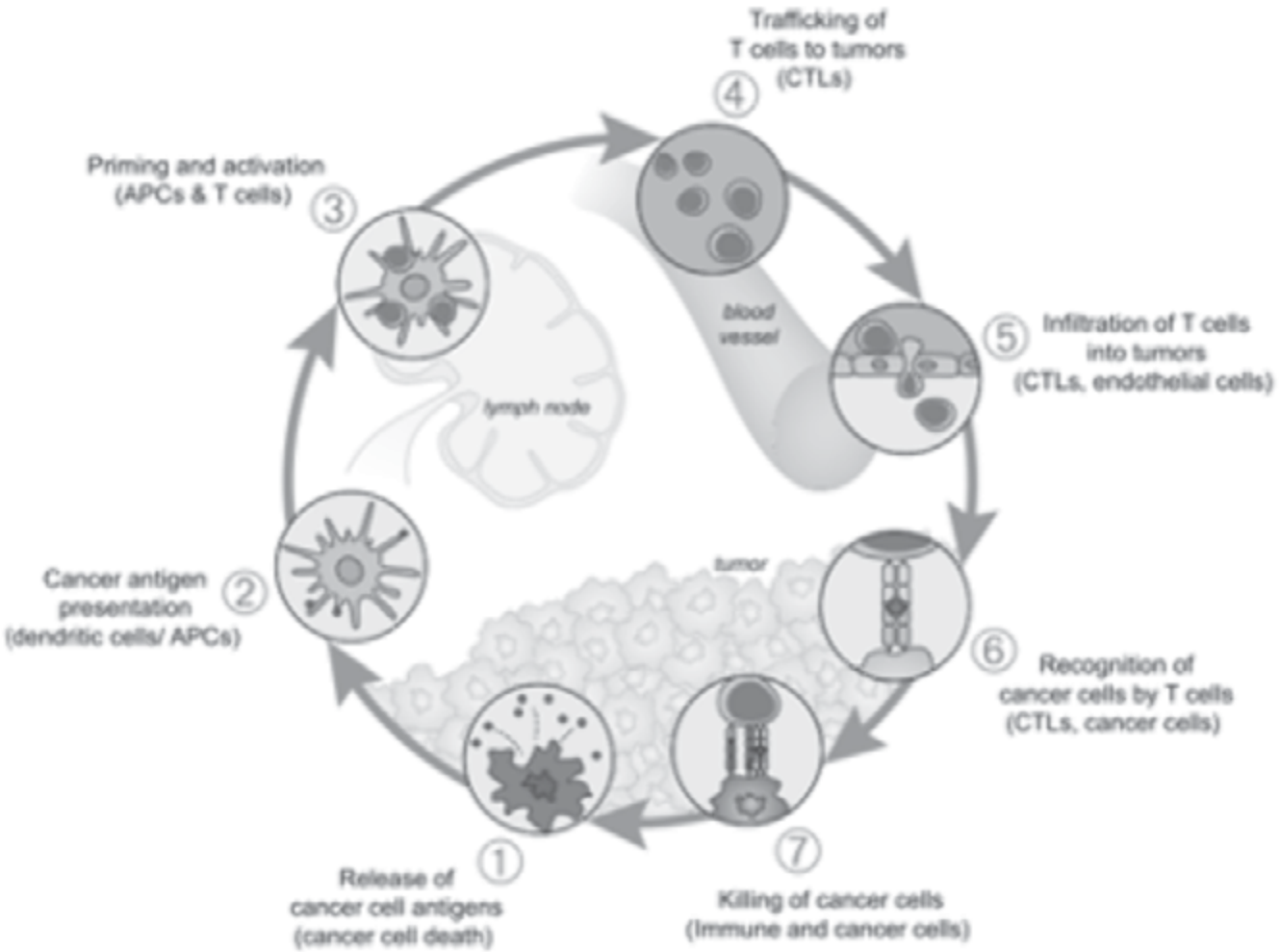
Estas herramientas son conocidas como “Hallmarks”, son 14 Hallmarks que han identificado, sin embargo esta vez nos centramos en: la evasión del sistema inmune.(2)

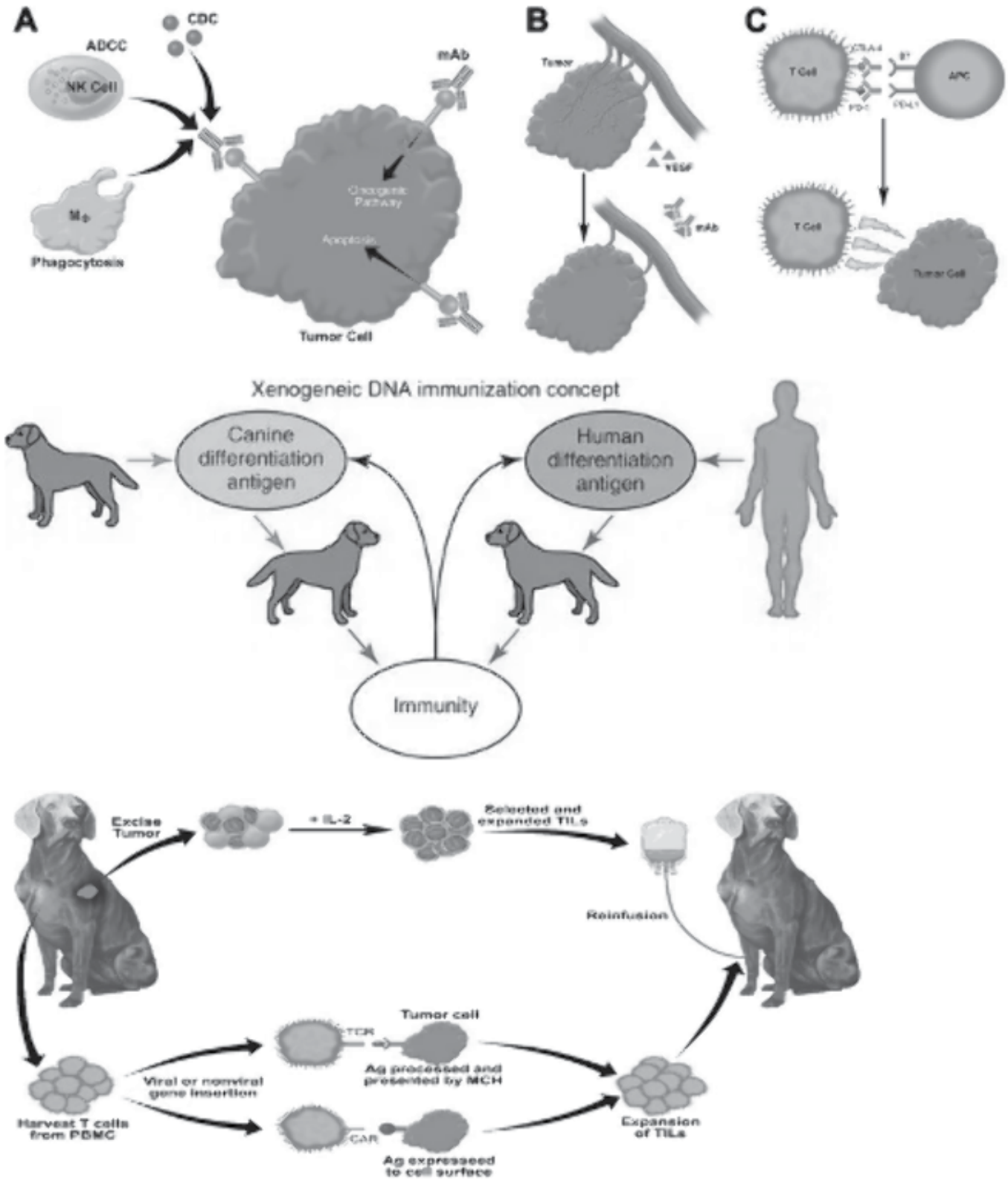
¿Qué es lo que ocurre normalmente cuando tenemos sistema inmunológico competente ?

El sistema inmunológico tiene la capacidad de erradicar patógenos (virales, bacterianos, fúngicos), controlar agentes inmunosupresores, y sobre todo, tiene la capacidad de detectar y eliminar células neoplásicas, esta capacidad es conocida como: INMUNOVIGILANCIA, compuesta por 3 fases: eliminación, equilibrio y escape.

Fase de eliminación: Capacidad de reconocer antígenos de células neoplásicas mediante la presentación de antígenos y acción de los linfocitos citotóxicos. Fase equilibrio: Fase donde el Sist. Inmune reconoce y controla el crecimiento tumoral además de mantener a las células tumorales silenciadas, es la fase más prolongada y silenciosa del cáncer que puede perdurar por muchos años y es unas de las razones por la cual los pacientes oncológicos son geriátricos. Fase de escape: Células tumorales logran evadir el sistema inmunológico por diversos mecanismos y comienza el desarrollo neoplásico (3,4).

En la inmunología del cáncer, se ha observado que la respuesta inflamatoria crónica favorece la tolerancia a la respuesta favorable al tumor. La actividad promotora tumoral favorece angiogénesis, invasión y metástasis suprimiendo sistema inmune adaptativo, adicionalmente los tumores son clasificados en: tumores fríos y calientes, lo que diferencia entre ellos es la cantidad de infiltración de linfocitos citotóxicos, otras células inflamatorias y linfocitos T regulatorios (Treg). Esta clasificación es importante para conocer como puede responder el paciente a inmunoterapia antineoplásica, es decir, que cuanto mayor infiltración de linfocitos T CD8 y otras células





inflamatorias y menor infiltración de linfocitos Treg en el microambiente tumoral son indicadores favorables para respuesta terapéutica a inmunoterapia; este tipo de evaluación es conocido como Inmuno-score, ya utilizado en algunas neoplasias humanas, como en el carcinoma colónico (5,6)

La inmunoterapia del cáncer es clasificada en 2 tipos: activa y pasiva. Inmunoterapia activa estimula el sistema inmunológico endógeno y es dividido en 2 grupos (específico y no específico). Inmunoterapia pasiva no estimula el sistema inmune y evita activarlo, por el contrario utiliza mecanismos específicos; y entre ambos grupos existe subgrupos con diversos tipos de tratamientos que son aplicados de acuerdo a cada tipo de neoplasia y su comportamiento tumoral. Por ejemplo, los inhibidores de “checkpoints”, es el tipo

de inmunoterapia pasiva más reconocida teniendo alto impacto en la oncología por alto suceso en el manejo de neoplasias en estado avanzado incluyendo el melanoma, carcinoma de células renales y neoplasia pulmonar de células pequeñas (7)

En medicina veterinaria, a pesar de no tener todas las opciones como existen en medicina humana, ya se tiene en el mercado algunas opciones como la vacuna Oncept ® aprovada por la FDA para tratamiento de melanoma oral, observandose resultados interesantes en estadio II y III. Los anticuerpos monoclonales (mba) para linfoma de células B (CD20) e células T (CD 52) (7,8)

Interesantemente, los CAR-T son un tipo de inmunoterapia utilizada en pacientes humanos con leucemias sin embargo, en veterinaria las investigaciones apuntan al uso de las células CAR-T para tratamiento de osteosarcoma apendicular y linfoma no-Hodking. Y así como ha sido demostrado que estadios avanzados de melanoma en el hombre con el uso de los anti-checkpoints se está obteniendo resultados interesantes, en la veterinaria investigaciones han demostrado que algunas neoplasias como melanoma oral, mastocitoma, tumor mamario tambien expresan PD-1 y CTLA-4 por lo que el desarrollo de nuevas inmunoterapias anti-checkpoints pueden traer resultados promisores en pacientes con neoplasias específicas en estados avanzados, sin embargo todas estas opciones de inmunoterapia están limitadas entre paises (9)

¿En latinoamerica que opciones de inmunoterapia tenemos? Especificamente en Brasil, en la Universidad de Campinas (UNICAMP) se ha desarrollado un tipo de inmunoterapia adaptativa no específica que es agonista de los Toll like receptors (TLR) 2 y 4 que activan la producción de INF-gamma promueve la activación de células TCD 8+, células dendríticas, células natural killer y macrofágos tipo 1 induciendo la muerte celular tumoral, adicionalmente se ha observado que disminuye la expresión de receptor kB factor nuclear activado (RANK) y su ligación (RANK-L) y consecuentemente previene o inhibe la progresión metastática, los tumores en estudio que se está observando resultados promisores en veterinaria son el melanoma oral canino y carcinoma urotelial alto grado (10)

En conclusión, los avances en el estudio del microambiente tumoral y el sistema inmunológico del individuo y su acción contra el cáncer ha permitido el desarrollo de nuevas estrategias terapeúticas que en conjunto con terapias convencionales han logrado obtener mayor tiempo de sobrevida en los pacientes con neoplasias en estado avanzados y gracias a la oncologia comparativa se está logrando desarrollar opciones de inmunoterapia en los animales, a pesar que en la medicina veterinaria existen diversas variables (presupuesto financiero, comportamiento heterogeneo tumoral entre razas, resultados variables de estudios clínicos) que limitan el desarrollo de nuevos fármacos ya tenemos grandes avances clínicos y medicamentos que posiblemente en el futuro se conviertan de uso comercial.

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HOW I PREPARE AND ADMINISTER FMT (FECAL MICROBIOTA TRANSPLANTATION) IN DOGS

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ABSTRACT BODY:

The intestinal microbiome is a complex system consisting of bacteria, fungi, archaea and viruses with extended interactions with its host. The constant interaction between gut microbiota and the intestinal innate immune system and the production of a vast array of metabolites (the gut metabolome) by theses microorganisms are both essential to their role in health and diseases. Overall, the gut microbiota provides essential benefits to the host health: locally they play a key role in the development and function of the gastrointestinal immune system, protect against enteropathogens, support the epithelial integrity and promote nutrient supply to the enterocytes. Moreover, and perhaps equally importantly, the gut microbiome and its metabolome also significantly impact the health of many other body systems beyond the GI tract.

Definition:

Fecal microbiota transplantation consists in transferring fecal microbiota from a healthy donor to a host with an abnormal intestinal microbiome. While the technique has been known and used for many centuries in various indications, it has gained popularity for the treatment of *Clostridiodes difficile* colitis and other diseases in people. The goal of FMT is to provide a healthy and viable sample of fecal microbiota in an attempt to repopulate the recipient's own microbiota. In addition, FMT also delivers essential metabolites such as secondary bile acids and fatty acids that may be lacking in the host.

Indications for FMT in dogs:

Unfortunately, there is only little published data on the use of FMT in dogs. One study evaluated the use of FMT as an adjunct to therapy in puppies with parvovirus infection. FMT was applied at patient admission and repeated every 48 h as needed. The results were encouraging since FMT lead to faster recovery from diarrhea and shorter hospitalization time. In another study, FMT was superior to antibiotics when evaluating recovery of normal fecal microbiota following an episode of acute diarrhea. However, clinical recovery was not faster in any of the 2 groups. Overall, there are several anecdotal reports but still only minimal data available from well-designed studies on the effects of FMT in dogs with various GI or other diseases, although studies on the value of the technique are ongoing.

Donor selection

The goal of donor selection is to identify healthy dogs

History and physical exam	Preferably between the ages of 1 and 10 years Preferably no travel history outside the local area No health issues in the last 6 or 12 months No history of chronic GI diseases, allergies and immune-mediated diseases Has not received any antibiotics in the last 12 months Regularly vaccinated according to existing guidelines Fed a balanced diet Not overweight or underweight (9-point body condition score between 4 and 6) Normal fecal consistency Deemed healthy on physical exam
Laboratory screening	Normal CBC and serum biochemistry Consider evaluation of basal cortisol, thyroxine Negative for parasite ova on fecal floatation, consider empirical deworming with a broad-spectrum anthelmintic drug Negative for Giardia oocysts on fecal floatation and ELISA fecal test, see above for empirical deworming Consider testing for fecal pathogens such as *Salmonella* spp., *Campylobacter* spp., etc.
Fecal microbiome evaluation]	Fecal dysbiosis index below 0

with feces that will not transmit any new disease to the recipient and hopefully benefit the recipients gut microbiome. Canine donors should be clinically healthy with a normal body condition score, not prone to digestive diseases such as vomiting and diarrhea, and screened for intestinal parasites (nematodes, cestodes and protozoa), and ideally for enteropathogenic bacteria (PCR). The author also evaluates the fecal dysbiosis index in order to check the health of donor's fecal microbiome. Moreover, fecal donors should not have received any antibiotics in the 3-6 months prior to collection (Table 1 summarizes several criteria of relevance)

From Chaitman J and Gaschen F: Fecal microbiota transplantation id dogs. Vet Clin Small Anim 51 (2021) 219–233

Preparation and administration:

Fresh feces should be processed within 6-12 hours of defecation. Numerous protocols can be used for preparation of FMT solution. The author adapted and currently uses a procedure used in human medicine. The feces are weighed and diluted with a volume of saline equal to 4 x the fecal weight in g, then thoroughly mixed prior to filtration through a tea strainer. Immediately following preparation, the fecal solution is administered to the recipient using a red rubber catheter in the proximal part of the descending colon via enema at a dose of 5 ml/kg. There is debate about the necessity to administer a mild sedative to recipients prior to the enema, however it may be useful, particularly in difficult dogs. In order to avoid rapid evacuation of the transplanted solution, recipients should be kept quiet for 30 min after administration. Some veterinarians recommend turning the recipient during the dwelling time. The need for repeat FMT depends on what disease is being treated. Dogs with chronic enteropathies do benefit from repeated FMTs while a single or two FMTs might be sufficient to help with acute diarrhea. Oral administration of FMT using gelatin capsules is another delivery method. Administration of fresh fecal solution is easiest under practical conditions and seems to warrant best survival of microorganisms. However, fecal solution can also be frozen at -20C after addition of 1 volume of pure glycerol solution to 9 volumes of fecal solution and stored for up to XXX months without major loss of viability.

Outlook:

FMT has gained popularity in the treatment of acute and chronic GI disorders of dogs in small animal practice. However, controlled prospective studies are required to evaluate the potential benefits of the technique in various canine diseases. It is possible that FMT might also have positive effects in the treatment of diseases primarily affecting organs outside the GI tract. Examples could include hepatic encephalopathy, obesity, behavioral disorders, etc.

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HOW I TREAT GASTROINTESTINAL ULCERATION

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ABSTRACT BODY:

Gastrointestinal (GI) bleeding is common in the veterinary intensive care unit (ICU) and is potentially life threatening. It is fairly common in dogs but occurs infrequently in cats. GI bleeding in dogs and cats leads to clinical signs that includes (but not limited to) lethargy, hematemesis, melena, hematochezia, pale mucous membranes, syncope, exercise intolerance, hyporexia and/or anorexia. The physical exam may reveal evidence of GI bleed (pale gums, melena on rectal exam, evidence of blood in the oral cavity) but absence of this finding does not rule out a bleed, especially an acute bleed)

Causes of GI bleed may include gastrointestinal neoplasia, thrombocytopenia and gastrointestinal ulcerative disease secondary to toxicity. Other possible causes include kidney disease (acute and chronic), hepatic insufficiency, intestinal diverticula, pancreatitis, intussusception, gastrointestinal foreign body, inflammatory bowel disease, hookworms, parvovirus and critical illness. Gastrointestinal ulcerations are the most common cause of GI bleed in dogs and cats. The severity of the bleed depends on the degree of mucosal erosion and arterial involvement.

History and Physical Exam An extensive history is very important when evaluating an animal with a suspected gastrointestinal bleed. Owners should provide a list of any medications administered to the patient (including over the counter medications), as well as any medications used by members of the household the pet may have gotten into. The owner may also be asked about any cutaneous masses (mast cell disease) that may have recently developed, as well as any signs that may suggest chronic gastrointestinal disease (inflammatory bowel disease, histoplasmosis). Common physical exam findings may collude pale mucous membranes, weak peripheral pulses, prolonged capillary refill time, evidence of melena or hematochezia on rectal exam, evidence of blood in oral cavity, petechiaeal hemorrhage, abdominal pain or collapse. It is important to note that these signs may not be present in every patient and their absence does not rule out gastrointestinal bleed.

It is helpful to attempt to localize the site of the GI bleed, as this helps determine differential diagnosis, diagnostic and therapeutic steps. Upper GI hemorrhage tends to be more profound than lower GI hemorrhage. Hematemesis and/or melena typically suggests upper GI hemorrhage while hematochezia suggests lower GI hemorrhage. However, the amount of time the blood remains in the GI tract determines the color of the stool. Hence if there is retention of blood in the colon, this could result in melena instead of hematochezia. On the other hand, severe acute upper GI bleeding may result in hematochezia if GI transit time is significantly decreased.

Diagnostic Testing Diagnostic tests to consider in the patient with suspected gastrointestinal hemorrhage include a minimum data base (CBC, Serum Chemistry panel, Urinalysis, Assessment of Coagulation). The CBC may reveal a microcytic hypochromic anemia (iron deficiency anemia). However, because it takes a while for iron-deficiency anemia to develop, a normocytic normochromic anemia may be present in cases of recent bleeding. Thrombocytopenia may be present in dogs (rarely cats) with GI bleeding secondary to immune mediated thrombocytopenia. The chemistry profile may reveal a high BUN-to-creatinine ratio (greater than 20) due to volume depletion and intestinal absorption of protein (digested blood). A high BUN-to-creatinine ratio is not expected in large bowel hemorrhage. The chemistry panel may reveal evidence of hepatic or renal dysfunction- which may predispose some patients to gastrointestinal ulcerations although this is still being investigated in patients with kidney injury. A coagulation profile may identify coagulopathies (rodenticide toxicity) although these cause GI hemorrhage uncommonly. However, any patient with significant gastrointestinal bleed may have slightly prolonged coagulation times due to consumptive loss of coagulation factors.

In equivocal cases of GI hemorrhage a fecal occult blood test (most of which rely on the peroxidase activity of hemoglobin) may be performed. While helpful for detecting occult GI hemorrhage, diets containing red meat or having high peroxidase activity, such as fish, fruits, or vegetables, can cause false-positive results. Animals should be fed a meat-free diet for at least 72 hours before a fecal occult blood test. The presence of peroxidase-producing bacteria within the GI tract also may cause false-positive results. Despite false-positive results a negative fecal occult blood test result does rule out significant GI hemorrhage.

Abdominal imaging may detect foreign material in

the gastrointestinal tract, neoplasia or evidence of free abdominal air (in cases of perforation of the gastrointestinal tract). Gastrointestinal ulceration may be identified on ultrasound exam although the absence of an ulcerative lesion does not rule out an ulcer. Endoscopy is the best way to assess the gastrointestinal tract for hemorrhage and ulceration but requires general anesthesia and may be cost prohibitive for some clients.

When significant gastric hemorrhage is suspected, passage of a nasogastric tube and aspiration of the stomach contents may confirm and help localize the site of GI hemorrhage. However, this procedure may cause discomfort, and false-negative results have been reported.

Treatment

Patients who present with evidence of hypovolemic shock should be fluid resuscitated. A packed red blood cell or whole blood transfusion may be required in patients that are clinical for the hemorrhage. Fresh frozen plasma may also be considered in patients with a coagulopathy, as well as a platelet transfusion in patients with thrombocytopenia.

Gastroprotectants are cornerstone for treatment of gastrointestinal hemorrhage. At standard doses, proton pump inhibitors (omeprazole, pantoprazole) are superior to histamine-2 receptor antagonists (famotidine, cimetidine) in treating dogs and cats with GUE. Proton pump inhibitors are most effective when administered at a dose of 1mg/kg IV or PO every 12 hours. However, histamine-2 receptor antagonists have the advantages of being more available, less expensive and they can be administered with food. In addition, while histamine-2 receptor antagonists at standard doses, do not meet the clinical goals (extrapolated from human patients) for treating acid related disorders in veterinary patients, a recent study showed a famotidine CRI was superior to pantoprazole in healthy dogs. In that crossover study, healthy Beagles on a famotidine CRI (1 mg/kg loading dose, followed by 8 mg/kg/day) had intragastric pH readings that were significantly higher than when they were treated with pantoprazole (1mg/kg IV q 12h). While the use of famotidine CRI is yet to be investigated in clinical patients, the author recommends considering it in animals with clinical signs of GUE due to its superior performance to pantoprazole. The use of a combination of proton pump inhibitors and histamine-2 receptor antagonists is not recommended.

The role of coating agents like sucralfate (0.5-1g/kg PO q 8h) and barium (0.5-1 mL/kg q 8-q12h) in patients with gastroenteritis is also controversial. In controlled studies, there have been no significant benefit of treatment in combining sucralfate with a histamine-2 receptor antagonist for the treatment of acute duodenal ulcers, stress bleeding or reflux esophagitis. Similar to the use of proton pump inhibitors and histamin-2 receptor antagonists, coating agents should only be considered in patients with clinical signs of gastrointestinal ulceration and erosion. Another recent comparative intravenous acid suppressant study compared a famotidine CRI ((1 mg/kg loading dose, followed by 8 mg/kg/day) to pantoprazole (1 mg/kg IV q 12 h) and esomeprazole (1mg/kg IV q 12 hr.) in healthy dogs. The results of the study showed that esomeprazole was superior to using a famotidine CRI or pantoprazole. However, there was still significantly better acid suppression in the famotidine CRI group, compared to the pantoprazole group. In that canine comparative study, pantoprazole did not meet the goals established for the treatment of gastroduodenal ulceration in people on all treatment days. Hence, the author's clinical preference is to utilize esomeprazole when available. When esomeprazole is unavailable, a famotidine CRI appears to be superior to pantoprazole for gastric acid suppression in dogs. A comparative oral acid suppressant study in cats also revealed that oral esomeprazole (1 mg/kg PO q 12) was superior to lansoprazole (1 mg/kg PO q 12) and dexlansoprazole (6mg/kg PO q 12).

Other considerations for the treatment of patients with gastrointestinal bleeding include analgesia, consideration for antibiotics when concerned about bacterial translocation. Surgery may be considered for patients with massive hemorrhage in order to resect the bleeding lesion and control the blood loss.

The prognosis of animals with Gi hemorrhage varies, depending on the underlying cause. Animals with moderate to severe bleeding requiring a transfusion generally have a poorer prognosis than those that have a mild bleed.

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DENTAL RADIOLOGY TECHNIQUES AND BASIC INTERPRETATION

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ABSTRACT BODY:

DENTAL RADIOLOGYSIMPLIFIED

Taking a dental radiograph:

Step 1: Patient positioning

Position the patient so that the area of interest is convenient to the radiographic beam. In general this is where the object is "up". For maxillary teeth, the patient should be in ventral recumbency. For mandibular canines and incisors the pet should be in dorsal recumbency. Finally, for maxillary cheek teeth, the patient should be in lateral recumbency with the affected side up. This being said, in our practice virtually all radiographs are exposed in lateral recumbancy. This takes some getting used to, but decreases the number of times a patient must be rolled when doing surgical or endodontic procedures.

Step 2: Film Placement within the patient's mouth

There is an embossed dot on the film. The convex side of this should be placed towards the x-ray beam. In most films, this side is pure white. The opposite or "back" side of the film will usually be colored (purple or green). Place the film in the mouth so that the entire tooth (crown and entire root surface) is covered by the radiograph. Remember, the roots of all teeth are very long. This is especially true of canine teeth, which are longer than you think. Always err on the side of having the film too far in the mouth to ensure you do not cut off the root apexes. The film should be placed as near as possible to the object (generally touching the tooth and gingiva) to minimize distortion.

Step 3: Positioning the beam head

There are two major techniques for positioning the beam head in veterinary patients. Both of these techniques are used daily in veterinary practice.

Parallel technique: This is where the film is placed parallel to the object being radiographed and perpendicular to the beam. This is how standard (large) films are taken. This gives the most accurate image. Unfortunately this is only useful in the lower cheek teeth in the dog and cat. This is due to the fact that these patients don't have an arched palate. The film cannot be placed parallel to the tooth roots because of the palate's interference. Therefore this technique is not always possible.

Bisecting Angle Technique: This is the most common type of dental radiograph taken in veterinary patients. This uses the theory of equilateral triangles to create an image that accurately represents the tooth in question. To utilize this technique, the film is placed as parallel as possible to the tooth root. Then the angle between the tooth root and film is measured. This angle is cut in half (bisected) and the beam placed perpendicular to this angle. This gives the most accurate representation of the root.

If this angle is incorrect, the radiographic image will be distorted. This is because the x-ray beam will create an image that is longer or shorter than the object imaged. The best way to visualize this is to think of a building and the sun. The building will create a 90 degree (right) angle to the ground. The bisecting angle in this case is 45 degrees.

Early and late in the day, the sun is at an acute angle to the building and casts a long shadow. In radiology this occurs when the angle of the beam to the object is too small and is known as elongation. At some point in the late morning and early afternoon, the sun is at a 45 degree angle to the building, which is the bisecting angle. This gives an accurate representation of the building height. As the sun continues up in the sky, the shadow shortens. This occurs in veterinary radiology when the angle is too great and is known as foreshortening. Finally, at noon, the sun is straight up from the building, which gives no shadow.

The **"Simplified Technique"** as developed by Dr. Tony Woodward does not utilize direct measurement of any angle, instead relying on approximate angles to create diagnostic images. There are only 3 angles used for all radiographs in this system 20, 45, and 90.

Mandibular premolars and molars are exposed at a 90 degree angle, maxillary premolars and molars at a 45-degree angle, and incisors and canines at a 20 degree angle.

To initiate any radiograph, place the film in the mouth and set the positioning indication device (PID) perpendicular to the film. For mandibular cheek teeth, this is the correct placement. For the maxillary premolars and molars, rotate the beam to a 45 degree angle. For the incisors and mandibular canines rotate 20 degrees. For the maxillary canines an additional rotation 20 degrees lateral is necessary to avoid superimposition of the first and second premolars.

Step 4: Setting the exposure

If you are using a machine where you set the exposure manually, you will need to set up a technique chart similar to one for a standard (large) unit. The good news is that there is only one variable that needs to be adjusted. \

If you are utilizing the computer controlled system, set the buttons for the species, size of the patient, and tooth to be imaged. If you have correctly set the machine and the image is incorrectly exposed, the easiest way to adjust is to change the *f* setting. By pressing this button, you will see the numbers go up on both sides. The one on the left is the *f* number and the one on the right is the exposure time. If you continue to press the button it will continue to increase the exposure until you reach 9 when it will markedly lower and the *f* number will go back to 1. If the radiograph is overexposed (too dark) lower the *f* number by 1. If it is underexposed (too light) increase the number by 1. Continue this process until you have the film that you want. Generally, the *f* number will be the same for all radiographs once you have discovered the correct setting for your machine start at that number in future sessions.

Step 5: Exposing the radiograph

Dental radiograph machines have a hand held switch to expose the radiograph. If it is possible, leave the room prior to exposing the radiograph. If it is not, stand at least 6 feet away at a 90 to 130 degree angle to the primary beam (meaning to the side or back of the tube head, not in front or behind). Once everything is set, press the button. It is important to remember, that these switches are “dead man’s”. This means if you let up during the exposure, it will stop the production of x-ray beams. On a standard unit, this will make a light radiograph, on a computer controlled one it will give an error message and you will need to start over. Make sure you hold the button down until the machine stops beeping.

CORNEAL ULCERS DOGS

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ABSTRACT BODY:

CORNEAL ULCERATION

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A corneal ulcer is a wound in the cornea that has the risk of contamination and extending to deeper corneal layers potentially resulting in corneal rupture.

When we diagnose a corneal ulcer, we must try to identify the cause, determine its severity, and select the appropriate therapy.

We can classify corneal ulcers into simple and complicated. The later can be divided into SCCED (spontaneous chronic corneal epithelial defect), melting ulcers, deep stromal ulcers, descemetocele, and corneal perforations.

The clinical signs include blepharospasm, ocular discharge, conjunctival hyperaemia, focal corneal oedema, miosis and aqueous flare.

The diagnosis is made by the presence of the clinical signs, by identifying fluorescein retention using cobalt light in a dark room.

Simple corneal ulcers

Simple corneal ulcers are superficial ulcers with a non-identifiable cause on examination. As a rule of thumb, they should heal within 2-6 days and doing so they cause minimal scarring.

Once diagnosed with the fluorescein test, prompt treatment should be started. We will always choose antibiotic cover. Fusidic acid BID is a good choice for a dog with normal anatomy. I am always more cautious for brachycephalic breeds, choosing a more broad-spectrum antibiotic such as chloramphenicol QID as even simple ulcers can deteriorate rapidly in these breeds. We should also provide cyclopegia to improve the reflex uveitis usually associated with the ulceration. We can use atropine bearing in mind that its use can decrease the tear production or cyclopentolate in cases where the tear production may be a concern. Artificial tears (i.e. hyaluronic acid) BID to QID can also be used. And some animals may need the use of an Elizabethan collar.

Remember that simple corneal ulcers can occur after general anesthesia and for this reason, frequent lubrication should be used in anesthetized patients.

Why do then simple corneal ulcers become complicated? They do because we have not identified the cause. Ectopic cilia, problems with the eyelid structure and function (i.e. lagophthalmia, entropion, facial paralysis), problems with the tear film and presence of a foreign body are common causes of initial simple corneal ulcers that can get complicated if not identified correctly. Or maybe we are dealing with a SCCED.

Complicated ulcers: SCCED (spontaneous chronic corneal epithelial defect)

Superficial and chronic corneal ulcer that can affect any breed of dog, but Boxers are overrepresented. It affects middle-aged dogs. The hallmark is the presence of a superficial corneal ulcer with non-adherent edges. The initiating event is likely trauma. It is not a bilateral disease however particularly in Boxers, a new SCCED may develop in the contralateral eye, typically within 24 months following a presentation of unilateral SCCED.

Histology of these corneas has shown the presence of a hyaline substance on the superficial stroma.

These ulcers do not resolve with medical treatment and are the only ulcers that we ever debride.

Here are some of the treatment options available:

- Cotton bud debridement (50%) + bandage contact lens (BCL): 58% success rate
- Epithelial diamond brush debridement (74-92% success). Beware of keratomalacia. 40-60 seconds duration of procedure
- Grid/punctate keratotomy (90% success)
- Superficial keratectomy (99% success). Scarring and pigmentation tends to occur. Not recommended as a first line of treatment
- Other: thermokeraroplasty, cyanoacrylate

My current treatment of choice is the epithelial diamond burr debridement.

Regarding the treatment, topical antibiotic such chloramphenicol QID is to be started after the debridement is performed. We also apply a single drop of atropine and give systemic non steroidals if the eye is particularly painful. The use and retention of a contact lens has been shown that significantly improves healing times.

Tetracyclines may be useful for the treatment of SCCEDs by mechanisms unrelated to matrix metalloproteinase inhibition. I do not tend to use

them either topically or systemically. Median time to epithelization was not significantly different between serum or saline treated eyes in one study.

Management of the carer's expectations is mandatory in these types of ulcers. Communication is key and they should be advised that more than one treatment may be needed.

Complicated ulcers: corneal melting

Corneal melting is an ocular emergency and to be treated appropriately, it needs to be correctly identified. In these ulcers breakdown of the collagen caused by matrix metalloproteinases occurs and it can be bacterial or sterile. Affected animals need to be hospitalised to receive intense medication (hourly drops). We must take a sample for cytology, culture and sensitivity. A broad spectrum antibiotic needs to be given such as a fluoroquinolone. This can be combined with gentamicin or chloramphenicol depending on the culture results. Remember cyclopegia and systemic pain relief (NSAIDS and opiods). We also need to add MMP inhibitors to the treatment regime. Examples of those are serum or plasma (my choice), tetracyclines and N-acetylcysteine. If the melting process is not arrested quickly, surgery should be considered.

Complicated ulcers: anterior and mid stroma

When the ulcerative keratitis progresses to deeper layers of the corneal stroma usually involves a secondary microbial infection that initiates the stromal destruction. If these stromal ulcers are non-progressive, they can be managed medically with treatment directed by the results of culture and susceptibility testing. The cytology will help us decide which antibiotic we should be starting (i.e., if we see rods we would start with a fluoroquinolone and if we saw cocci we would start with chloramphenicol). Remember the cyclopegia and the systemic NSAIDS. Sometimes these ulcers can end up getting deep or even perforating despite a good initial approach. Surgery is indicated when the depth is severe or when perforation has occurred. There are different techniques such as corneal transplant using frozen/ fresh corneas, corneoconjunctival transposition and the use of biosynthetic materials (i.e. Biosis, A-Cell). We should attempt to avoid ulcers to progress as scarring in dogs is severe and vision ends up getting significantly reduced, especially in bracycephalic breeds.

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DIAGNÓSTICO CITOLÓGICO DEL PACIENTE CON CÁNCER

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ABSTRACT BODY:

DIAGNÓSTICO CITOLÓGICO DEL PACIENTE CON CÁNCER

El cáncer en su conjunto es la causa de muerte más frecuente en animales de compañía geriátricos. Sin embargo, al hablar de cáncer incluimos más de 100 enfermedades con pronósticos y tratamientos diferentes que varían dependiendo no sólo del tipo histológico, sino del grado, localización o especie en la que se diagnostica. Por ello, el primer paso en el abordaje de los pacientes oncológicos, es determinar el diagnóstico concreto de la enfermedad.

En aquellos casos en los que el tumor es evidente, puede intentarse un diagnóstico mediante la citología. Aunque no sustituye a la histopatología, puede darnos un diagnóstico de forma rápida y barata en muchas ocasiones. Podemos realizarla a partir no sólo de masas, sino de efusiones, médula ósea o mucosas.

Aunque normalmente la citología se realiza por aspiración, en aquellos tejidos muy vascularizados, puede tomarse la muestra simplemente pinchando la masa con una aguja fina, sin aspirar, para intentar evitar la presencia de mucha sangre que dificulte la interpretación de la muestra.

La citología puede interpretarse en la clínica o ser enviada a un patólogo veterinario dependiendo de la experiencia del clínico y las características de la muestra. La primera distinción es entre muestra inflamatoria o neoplásica, aunque en algunos casos la muestra presenta una población mixta entre células inflamatorias y titulares, por lo que es difícil diferenciar entre neoplasia con inflamación secundaria y la inflamación con displasia celular; en estos casos, se recomienda la biopsia del tejido.

Si la muestra es tisular, se clasifica según su origen en epitelial, mesenquimal, de células redondas o de origen endocrino, y posteriormente se buscan signos de malignidad.

Tumores epiteliales

Los tumores de origen epitelial se inician en piel, mucosas o glándulas. Generalmente se obtienen muestras muy celulares y las células se observan en agregados, a veces muy cohesivos. En condiciones normales estas células son muy similares en morfología con tamaños celulares o nucleares parecidos. En tumores epiteliales, las diferencias entre las células suponen criterios de malignidad, por lo que una vez reconocido el tumor como epielial se buscan estos criterios de malignidad para diferenciar entre lesiones benignas o malignas.

Los criterios habituales son la diferencia en el tamaño celular (anisocitosis), nuclear (anisocariosis), presencia de nucléolos prominentes (que pueden verse en diferente número, tamaño o forma), incremento del ratio núcleo/citoplasma, presencia de mitosis anormales o multinucleación en los casos donde no es normal (hay que recordar que algunas células pueden presentar varios núcleos de forma habitual, como algunos hepatocitos, que presentan dos núcleos, o como los osteoclastos, con múltiples núcleos).

En ocasiones, hay carcinomas bien diferenciados que son difíciles de identificar mediante citología. Un caso frecuente son los carcinomas hepatocelulares. El carcinoma hepatocelular puede ser pleomórfico pero generalmente está bien diferenciado y las diferencias con los hepatocitos normales son muy sutiles. En estos casos, algunos cambios que sugieren malignidad son la pérdida de cohesión de los hepatocitos, la presencia de células rotas con núcleos intactos o células con tres o más núcleos.

Tumores mesenquimales

Estos tumores se originan en tejido conectivo. La celularidad de los aspirados suele ser más baja que en tumores epiteliales o de células redondas pero a veces se obtienen muestras muy celulares. Si con un aspirado con aguja fina no se obtiene muestra suficiente pueden usarse agujas de mayor calibre y aspirado con jeringuillas de 5-10 cc para hacer vacío.

En citología los tumores mesenquimales se pueden reconocer por la forma fusiforme, alargada o irregular de sus células, con citoplasmas alargados o con bordes mal definidos. Los núcleos pueden ser redondeados pero generalmente son ovoides o alargados. No es posible sin embargo diferenciar los diferentes tipos de sarcoma en la mayoría de los casos. Una vez identificado el tumor como de origen mesenquimal se buscan criterios de malignidad, que como en el caso de los tumores epiteliales, consisten en diferencias en la morfología celular.

Tumores de células redondas.

Este grupo de tumores es donde podemos obtener mayor información con la citología ya que puede

llegarse a un diagnóstico concreto en la mayoría de los casos. Los tumores de células redondas incluyen los linfomas, mastocitomas, tumores de células plasmáticas, tumor venéreo transmisible y tumores histiocíticos. Algunos melanomas puede también tener una morfología celular redondeada aunque pueden presentar también aspecto mesenquimal o epitelial.

Linfomas: el diagnóstico citológico de linfomas de células grandes se basa en la identificación de una población homogénea y mayoritaria de linfoblastos (de mayor tamaño que un netrófilos) en linfonodos, o su presencia en tejidos extranodales (piel, riñones en gatos).

Mastocitomas: Este tumor frecuente no solo se puede diagnosticar por citología identificando las células con abundantes gránulos púrpura en el citoplasma, sino que se han descrito criterios morfológicos celulares que se asocian a mastocitomas de alto grado, como son la pobre granularidad, la multinucleación (tres o más núcleos) o las mitosis.

Tumores de células plasmáticas: Estos tumores pueden presentarse en forma de tumores localizados (plasmocitomas, en piel, cavidad oral…) o como enfermedad sistémica (mieloma múltiple en médula ósea). Desde el punto de vista citológico, pueden ser células plasmáticas bien diferenciadas, con la característica zona del aparato de Golgi, que se identifica de aspecto claro al lado del núcleo celular, o como células redondas muy anaplásicas, con múltiples criterios de malignidad.

Tumor venéreo transmisible: con células redondas con vacuolas en el citoplasma.

Tumores histiocíticos: Las enfermedades histiocíticas caninas incluyen un grupo de tumores con comportamiento clínico muy variable, y enfermedades no tumorales, cuya presentación clínica es muy similar a las neoplasias, pero con un tratamiento y pronóstico completamente diferente. En este grupo de tumores se incluyen los histiocitomas, histiocitosis cutánea, sarcoma histiocítico o el sarcoma histiocítico hemofagocítico. El diagnóstico citológico puede ser difícil y es necesario tener en cuenta factores clínicos como la presentación, reseña del animal, etc.

Tumores endocrinos

Los tumores endocrinos pueden diagnosticarse mediante citología pero hay que tener en cuenta algunas particularidades. En general la muestra es altamente celular pero puede haber mucha contaminación con sangre (es frecuente en el caso de tiriodes). Las células pueden estar agrupadas en racimos poco cohesivos, sin diferenciar límites citoplasmáticos, y es frecuente encontrar núcleos sueltos sin citoplasma asociado, denominados núcleos desnudos. En estos tumores, a pesar del comportamiento biológico maligno (carcinomas) no es frecuente encontrar criterios de malignidad. Los tumores de las glándulas de saco anal citológicamente se presentan como este tipo de tumores.

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ABSTRACT BODY:

Las hernias perineales se producen por un fallo del diafragma pélvico, lo que permite la protusión del contenido abdominal o pélvico entre dicha estructura y el recto.

Las hernias o roturas perineales se observan más frecuentemente en perros machos enteros, aunque también han sido descritas en machos castrados, hembras y en el gato. La etiología es todavía incierta aunque se asocian con cambios degenerativos en los músculos del diafragma pélvico. Con la pérdida del soporte lateral se produce una desviación rectal progresiva. Cuando se produce de forma unilateral esta alteración se conoce con el nombre de saculación, mientras que a la afección bilateral se la llama dilatación. Los divertículos rectales reales, con la mucosa rectal sobresaliendo a través de la musculatura rectal, son extremadamente raros. Las hernias pueden complicarse con la inclusión de grasa pélvica o peritoneal, asas de intestino delgado, próstata, quistes prostáticos o paraprostáticos y, en casos graves, por retroflexión de la vejiga. [1]

En la etiopatogénesis de las hernias perineales se incluyen distintos factores aunque ninguno ha sido completamente corroborado. Estos incluyen la prostatomegalia, la disminución de receptores para los andrógenos en los músculos elevador del ano y coccígeo, la saculación rectal (más probable que sea secundaria a la herniación), la colitis y el corte de cola.[1]

Anatomía perineal

El diafragma pélvico lo componen:

- Músculo elevador del ano
- Músculo coccígeo
- Fascia externa
- Ligamento sacrotuberoso
- Músculo glúteo superficial

Las estructuras que se verán implicadas en una hernia perineal podrán ser la vejiga de la orina, el recto, próstata, grasa retroperitoneal, yeyuno o colon,…

Diagnóstico y aspectos clínicos

Las hernias perineales suelen aparecer casi exclusivamente (93%) en machos caninos no castrados. Su aparición en hembras es casi siempre de causa traumática.

En gatos son poco frecuentes

Suele presentarse en animales de edad adulta (a partir de 5 años) y pueden ser unilaterales (60% de los casos) o bilaterales (40%).

Los signos clínicos más frecuentes son un abultamiento en la zona perineal, tenesmo y estreñimiento [2].Pueden añadirse otros como disuria, vómitos, letargia y pérdida de peso

El tratamiento de la hernia perineal deberá ser siempre quirúrgico. El tratamiento médico es sólo para apoyar al tratamiento quirúrgico. Este tratamiento médico podrá ser: dieta baja en residuos, laxantes, castración ó antiandrógenos

La castración está indicada junto con la herniografía ya que se ha demostrado que esta reduce la recurrencia.

Cuando la hernia es muy crónica, la dilatación de la ampolla rectal es muy grande o se ha producido retroflexión de la vejiga, debemos realizar laparotomía para fijar la vejiga, el colon y los conductos deferentes.

CONSIDERACIONES PREOPERATORIAS

En la visita previa a la cirugía planteamos un perfil geriátrico y hacemos un examen rectal para valorar el grado de dilatación, la presencia de estructuras dentro de la hernia y si debemos hacer técnicas alternativas para evitar las recidivas.

Pautaremos enemas preoperatorios dos días antes y el día de antes con productos comerciales.

En el momento de la cirugía vaciar las heces de manera manual, colocar una gasa y realizar una sutura en bolsa de tabaco. Cateterización de la uretra si realizamos la resolución de hernia perineal bilateral.

Durante la cirugía colocamos Cefalotinas: 15 – 30 mg/kg/I.V. repetir c/ 2h durante la cirugía

Protocolo anestésico

Debemos tener en cuenta que se trata de animales geriátricos, una anestesia epidural con lidocaína o ropivacaína más un analgésico opiáceo sin preservante, morfina, 0,1 mgr/Kg es interesante. Respecto al dolor, metadona durante la cirugía y en el posoperatorio inmediato, robenacoxib al final de la cirugía y en los días posteriores apoyado por metamizol como analgesia de rescate ya que si el animal presenta dolor y si realiza esfuerzos para defecar puede producirse un desgarro de los puntos,.Tipo de cirugía: se trata de una cirugía de tercio posterior por lo que puede ser de gran

ayuda realizar una anestesia EPIDURAL. La epidural la podremos realizar con una mezcla de anestésico local (lidocaína o ropivacaina) y un analgésico opiáceo sin preservante, morfina, 0,1 mgr/Kg

TÉCNICAS QUIRÚRGICAS

La bibliografía describe varias técnicas para la resolución de las hernias perineales:

- ELEVACIÓN MÚSCULO OBTURADORINTERNO [3] (éxito 80%)
- TRANSPOSICIÓN MÚSCULO SEMITENDINOSO[4]
- TRANSPOSICIÓN MÚSCULO GLÚTEO SUPERFICIAL (éxito 64%)
- IMPLANTES SINTÉTICOS
- FASCIA LATA

La castración siempre estará recomendada como técnica complementaria para reducir las recidivas. La herniorrafía convencional suturando las estructuras que han permitido la hernia aboca al fracaso inmediato. Como ya he comentado valorar colopexia y cistopexia dependiendo del grado de dilatación de la ampolla rectal.

Elevación del músculo obturador interno[3]

Técnica quirúrgica:

- Colocación del animal en decúbito esternal con la cabeza más ventral que la zona perineal, las extremidades posteriores colgando y sus nervios femorales protegidos. Previamente hemos introducido una gasa por el ano y hemos cerrado el mismo con una bolsa de tabaco.
- Realizar una incisión en la piel desde la zona lateral de la base de la cola hasta el aspecto ventral de la hernia pasando por la parte medial de la apófisis isquiática. Disecar de forma roma el saco herniario si lo hay y exponer el contenido. Reducir el contenido después de su inspección.

Una vez reducida la hernia identificar y preservar la arteria, vena y nervio pudendo

- Localizar el esfínter anal externo, el ligamento sacrotuberoso y el músculo obturador interno. Una vez localizados incidir el borde caudal del origen del ms. Obturador interno desde su aspecto medial hasta la parte más lateral y elevarlo desde el isquion. Incidir el ligamento del músculo obturador.

Al disecar el músculos obturador interno: no dañar nervio obturador (aspecto caudal del agujero obturador)

Al seccionar tendón: no dañar nervio ciático

Desplazar el músculo obturador interno hacia el defecto. Realizar puntos sueltos con material sintético no absorbible. Empezar posicionando el elevador del ano y el músculo coccígeo con el esfínter anal externo (aspecto dorsal). Posteriormente realizar puntos de sutura entre el obturador interno y el esfínter anal externo (aspecto medial) y el elevador del ano y el músculo coccígeo (aspecto lateral)

5. Cerrar el tejido subcutáneo y piel de forma convencional, yo prefiero dejar un Penrose durante una semana.

Complicaciones y pronóstico

La complicación más frecuente es la dehiscencia de la sutura o la infección en la incisión quirúrgica, con una incidencia descrita de 6-26%. El drenaje, lavado y una terapia antimicrobiana adecuada son generalmente curativos.

Otras complicaciones:

- La incontinencia fecal debido a la pérdida de la función del esfínter anal externo puede producirse si los nervios rectales caudales o pudendos son dañados durante la herniorrafia. Por ello tras la disección del contenido herniario buscaremos el paquete pudendo gracias al pulso de la arteria. La neuritis se puede recuperar pasadas unas semanas y el esfínter se puede reinervar desde el lado contralateral. El daño nervioso bilateral puede resultar en una incontinencia fecal permanente. No anudar las suturas hasta haberlas realizado todas, evita daños neurológicos.

- La parálisis ciática es muy rara NUNCA debemos manipular lateral al borde del isquion, por ello suturas en el ligamento sacrotuberoso sólo las realizaremos si no hay más remedio. La compresión del nervio se caracteriza por dolor y cojera en el lado afectado. Si se detecta, las suturas deben ser retiradas inmediatamente ya sea revisando la reparación original o mediante un abordaje caudolateral al nervio ciático.

- La eversión/prolapso rectal puede producirse inmediatamente después de la cirugía. La lubrificación con gel de anestésico local y la reducción manual, combinado con una sutura en bolsa de tabaco temporal, es generalmente todo lo necesario. Cuando el prolapso rectal recurre deberá realizarse una colopexia incisional con el fin de prevenir más prolapsos. También será necesario establecer la causa del tenesmo y tratarlo[1].

- Las fístulas rectocutáneas son complicaciones raras pero pueden producirse si el material de sutura ha penetrado de forma inadvertida la mucosa rectal o por reacción a las mallas.

- La recurrencia es poco frecuente (10- 20%) cuando se ha realizado de forma correcta la transposición del músculo obturador. Ante cualquier duda apoyarse en la colopexia y cistopexia. Hay autores que entienden la hernia perineal como un proceso bilateral y tras

la corrección del lado afectado realizan una sutura preventiva en el lado contrario.[5]

Serie de fotos en:

https://bit.ly/3A1VzwL

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ORTHOPEDIC DIAGNOSIS OVERVIEW: GAIT, CLINICAL EXAM & IMAGING CHOICES

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ORTHOPEDIC DIAGNOSIS OVERVIEW: GAIT, CLINICAL EXAM & IMAGING CHOICES

Introduction

Gait abnormalities are among the most reported medical conditions in dogs and cats. And although lameness is one of the most ostensible signs of pain in the dog and cat, it is not the only sign. Localization of the pain can be extremely challenging during the orthopedic exam but it is critically important to help guide the use of diagnostics, achieve a diagnosis, and prescribe treatment. A sequenced, repeatable, and consistent approach to the orthopedic patient is essential to achieve consistent, accurate orthopedic diagnosis. Failure to perform any of the required steps will skew your findings and create error and misdiagnosis.

Step 1: Observation

Before commencing the more interactive components of the orthopedic examination and during the collecting or confirmation of the owner's patient history, observation allows you to see changes that may not be apparent when in close contact with the animal. Watching the patient stand, rise, sit and lay down within the examination area allows a preliminary 'feel' for the patient's overall demeanor, body control and recognition of any significant visible issues. Confirming some of your early observations with the owner to assess if they have seen the same abnormalities or have others to add allows a deeper understanding of the lameness issue.

Step 2: Tools

In general, the most consistently utilized tools are your visual assessment, palpation, patience and a well-trained veterinary assistant.

Step 3: Gait Exam

After preliminary visual assessment of the patient within the exam room it is crucial that you watch the patient move. Distances can vary but a minimum of twenty feet passes often allows you to see present lameness, determine the limb affected and grade the severity. Repeating the passes several times allows a more consistent assessment and negates the initial patient's exuberance and excitement. Following gait exam the affected limb(s) should be stated and the Grade of lameness (1-4 or 1-9 depending on personal preference) recorded. Classifying the lameness as mild, moderate, severe or non-weight bearing (Grades 1-4) allows initial and follow-up exams to be compared and contrasted to term the lameness as static, resolving or progressive over time.

Step 4: Standing & Prone Examination

There are some simple techniques for this portion of the examination that make a difficult task more manageable and will result in more accurate findings while avoiding patient and owner distress and veterinary team trauma.

a.) Always use a high quality, non-slippery floor mat for medium and large patients and a well- padded raised exam table for small patients.

b.) Have the owner stand or sit directly in front of the patient to allow the patient to see the owner but minimize the physical contact the owner has with the patient during the exam.

c.) Always have a trained, capable veterinary assistant restrain the patient for the exam, never attempt to perform the exam without restraint or with the owner providing restraint.

d.) Always commence your complete orthopedic exam with good body contact between yourself and the patient. Maintain physical contact with the patient throughout the exam to allow the patient to know where you are and not feel under random attack.

e.) Perform a sequential, thorough and repeatable physical examination with care to examine all the limbs and all the joints in a standard fashion.

f.) Use an orthopedic exam recording form to document clinical findings and use a member of your staff to record your findings, both normal and abnormal as you perform the exam. This will limit errors of omission in terms of the exam and in documenting relevant findings and create a good medical record.

g.) Following the standing exam, it is often helpful to perform a prone examination of the limbs. Placing a patient into lateral recumbency can be challenging but can be performed by the clinician and assistant quickly and smoothly if they work together. Wrestling the patient into submission must be avoided.

The advantages to evaluating the animal in lateral recumbency include the ability to take the animal out of its normal standing position, which may make some dogs more docile. It also may reduce the overall limb muscle tone that results when standing and allows the examiner to detect more subtle conditions of joint laxity.

Physical examination is the best tool we have and is often the most challenging. Your physical exam should not become abbreviated and truncated. This leads to errors in patient assessment which in turn leads to issues with correct diagnosis. In some cases, patients may need some chemical sedation or isolation away from the owners to allow better and safer patient exam. Owners may resist a suggestion to muzzle, isolate or sedate their pet and a calm but firm approach to these situations must be developed by the team to avoid emotional escalation from both sides.

Step 5: Limb Analysis

The aim and principal sequence of each limb exam is the detection of primary and secondary abnormalities, the determination and recording of their severity and a determination of any inter-relationship between them. The determination of muscular atrophy is important and should not be missed. Gentle simultaneous grasping and palpation of paired muscle groups allows a comparison and contrast between left and right sides. A standing test of value is the palpation for joint effusion and peri-articular remodeling. Hock, stifle, carpal and elbow effusion and thickening can all be assessed most accurately with the patient standing. Documenting mild, moderate, and severe effusion and remodeling allows both the current active and historical duration of joint abnormality to be broadly determined. Determining each joints range of motion (ROM), documenting decreases in ROM and the presence and severity of concurrent discomfort and pain is crucial to avoiding examination errors. The use of 'within normal limits' (WNL), 'no palpable abnormality' (NPA) and 'no visual abnormality' (NVA) ensures your medical record documents normality according to the examiner at the time of exam.

Prior to the transition from pelvic to thoracic limbs by the examining team, it is wise that the sacral, lumbar and associated pelvic limb nerve tracts are examined. The thoraco-lumbar junction, the cervico-thoracic junction, the thoracic limb nerve tracts and cervical spine all must be included in the examination protocol as the presence of a primary or secondary spinal or neuropathic pain focus must not be missed. Spinal and neuropathic pain is common in companion animals and will factor into both diagnostic decisions and treatment plans.

Step 6: Diagnostic Imaging

Once the site(s) of patient discomfort and dysfunction is localized by examination, it is time to consider imaging. Way too frequently the patient is imaged before localization which results in excessive chemical and physical manipulation of the patient, excessive imaging effort by the veterinary Team, inconclusive diagnostic efforts, and increased confusion regarding the actual cause of lameness and dysfunction. It is never a good idea to image widely without focus in the hopes of making a definitive diagnosis. The choice of imaging modalities is dictated by condition and modality availability. The most common and widely used technique is radiography. The most poorly practiced and incorrectly performed technique is also radiography. The advent of digital radiography has done nothing to change this reality, it may indeed have exacerbated it. The ability to collect digital images in rapid-fire succession without a demonstrable immediate cost to the practice (in comparison to the use of flat film in the past) has lead to a proliferation of studies with multiple images of incorrectly positioned patients that are difficult to interpret and lend themselves to misdiagnosis and error. The key to excellent radiography is a sedated, contained, well-positioned patient with appropriately exposed well collimated orthogonal views of focal anatomic areas. Although the means to achieve this end may vary the goals do not. Deviating from these key goals decreases the quality of the study achieved and decreases the clinicians chance to use that study to determine the definitive diagnosis, stage the pathology present in terms of radiographic severity and determine the relative health of other adjacent or possibly involved secondary sites. In addition, or in some cases instead of radiography, the use of computer tomography (CT) and magnetic resonance (MR) as cross-sectional imaging modalities has increased dramatically in the past twenty years in both large and small animal practice. Ultrasonography and radionucleotide gamma camera imaging (technetium scans) are also useful in musculoskeletal disease although advanced training and progressive experience in musculoskeletal ultrasound is a must and technetium scanning is not routinely available in many areas. The respective choice of advanced diagnostic imaging is dictated by the anatomic area being evaluated, the availability of the modality to the individual clinician and the client's financial commitment limitations. Careful selection of advanced imaging options often requires contribution from a specialist veterinarian will extensive experience in the modality being selected.

RECOMENDACIONES SOBRE VACUNACIÓN DE PERROS PARA LATAM

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ABSTRACT BODY:

RECOMENDACIONES SOBRE VACUNACIÓN DE PERROS PARA LATAM

La comprensión del concepto de vacunas esenciales y no esenciales es fundamental para determinar el protocolo de vacunación de un perro. Las **vacunas esenciales** son aquellas que todo perro, independientemente del lugar que viva o de su estilo de vida, debe recibir. Las vacunas esenciales contienen el virus del moquillo (CDV), el adenovirus canino (CAV) y el parvovírus canino (CPV2). En los países en que la rabia canina es endémica, la vacuna antirrábica también se considera esencial. Las **vacunas no esenciales** son las que se consideran para animales cuya ubicación geográfica o estilo de vida los pone en riesgo de infecciones específicas. Ellas no son necesarias para todos los perros y no deben utilizarse cuando no hay pruebas de la existencia de una enfermedad en la región, o cuando no existe riesgo de exposición. Las vacunas no esenciales incluyen las vacunas contra la leptospirosis, contra algunos microorganismos del Complejo Respiratorio Infeccioso Canino (CRIC), tales cuales el virus de la parainfluenza (CPiV) y la *Bordetella bronchiseptica* (Bb), y contra la leishmaniasis. Algunas vacunas son consideradas **no recomendadas** para perros, porque no existe suficiente evidencia científica que justifique su uso. Estas incluyen la vacuna que contiene el coronavirus entérico canino (CCoV) y la vacuna contra la giardiasis.

La vacunación esencial de cachorros debe comenzar entre las 6 y 8 semanas de edad, vacunándose cada 3 a 4 semanas hasta las 16 semanas de edad o más, con vacunas que contengan de preferencia virus vivos modificado (VVM). El objetivo de administrarse las dosis hasta las 16 semanas o más es para que se tenga mayor seguridad de que se ultrapasó la interferencia de los anticuerpos maternos. La vacunación esencial puede comenzar a las 4 o 6 semanas de edad con vacunas que contengan CDV y CPV2 de alto título, desarrolladas para la utilización en cachorros a partir de 28 días (si está disponible), antes de cambiar a la vacuna esencial trivalente a las 8 semanas o más. Se debe administrar una última vacuna esencial de VVM a los 6 meses de edad para asegurar que todos los cachorros reciban, al menos, una dosis de vacuna capaz de conferirles inmunidad, en caso de que aún existan anticuerpos maternos a las 16 semanas. El desarrollo de la inmunidad protectora no depende del número de vacunas esenciales de VVM administradas al cachorro, sino de cuándo son administradas.
En el caso de los perros, hay abundantes pruebas científicas que respaldan la revacunación con vacunas esenciales de VVM con una frecuencia no superior a 3 años. La serología podría usarse para monitorear la inmunidad protectora (para CDV, CAV y CPV2) y para ayudar a la toma de decisiones sobre los intervalos de revacunación. Si bien las autoridades reguladoras de los países de América Latina exigen la revacunación anual con vacunas esenciales internacionales de VVM, registradas en otros lugares para su uso a intervalos de 3 años, esta práctica se considera inapropiada.
El aumento de la frecuencia de vacunación con las vacunas esenciales de VVM no confiere una mayor protección a un perro. Sin embargo, el aumento del número de perros que se vacunan, adecuadamente, es mucho más importante para garantizar la protección mediante la inmunidad de rebaño, que vacunar a cada animal con mayor frecuencia. Las vacunas contra la rabia canina deben ser administradas a partir de las 12 semanas de edad y la revacunación debe realizarse de acuerdo con las recomendaciones de los prospectos y las leyes del país.

Los profesionales de América Latina y sus asociaciones nacionales deberían ejercer presión sobre la industria para acceder a vacunas caninas internacionales que contengan sólo los componentes esenciales (CDV, CPV2 y CAV) o los componentes no esenciales (CPiV, Bb y Leptospira). Esto permitiría la administración de las vacunas esenciales de VVM cada 3 años y la vacunación anual con vacunas no esenciales para perros en riesgo. Actualmente, la mayoría de las vacunas disponibles en los países de América Latina contienen antígenos esenciales de VVM (CDV, CAV, CPV2) combinados con antígenos no esenciales (Leptospira, Bb) y antígenos no recomendados, como el CCoV.

Aunque no hay duda de que las leptospiras infectan a los perros de América Latina, existen raros estudios científicos sobre los serovares infectantes. El problema es que la gran mayoría de los estudios no se basan en el aislamiento y cultivo de las leptospiras, sino en la serología (MAT). La vacuna contra la leptospirosis se debe administrar de acuerdo con las recomendaciones del fabricante, generalmente empezando a las 8 o 9

semanas de edad, con administración de dos dosis, con un intervalo de 2 a 4 semanas, y revacunación anual.

En cuanto a las vacunas no esenciales para el CRIC, tenemos vacunas inyectables, intranasales y orales. Las vacunas inyectables necesitan de 2 dosis, con intervalo de 3 a 4 semanas, y se administran, generalmente, a partir de las 8 semanas de edad. Las vacunas intranasales y orales necesitan de una sola dosis, siendo que las intranasales pueden utilizarse en cachorros de tan solo 3 semanas de edad, mientras que las orales a partir de las 8 semanas de vida. Para todas, se requiere una revacunación anual. Las vacunas administradas por vía mucosa inducen a una respuesta local y más rápida que las inyectables. La vacunación por vía intranasal probablemente genera una inmunidad protectora superior en relación a las vacunas orales, aunque los estudios publicados, que abordan esta cuestión, no siempre coinciden. La vacunación intranasal puede tener el beneficio añadido de contener hasta 3 antígenos (CPiV, Bb, CAV-2), mientras que las vacunas orales tienen la ventaja de ser fáciles de aplicar, pero contienen un solo antígeno (Bb).

Con relación a la leishmaniasis, actualmente en América Latina solo hay dos vacunas. El protocolo de vacunación de los cachorros incluye 3 dosis, administradas con 3 semanas de diferencia, y un refuerzo anual. Dependiendo de la vacuna, se puede utilizar en perros a partir de los 4 o 6 meses de edad. Los perros adultos que nunca han sido vacunados reciben el mismo protocolo de los cachorros. Las vacunas contra la leishmaniasis solo deben considerarse para los perros que viven en áreas endémicas, donde están en riesgo de ser infectados. Es importante señalar que los perros vacunados pueden infectarse y transmitir la infección, aún estando asintomáticos, de modo que las vacunas contra la leishmaniasis canina son una herramienta adicional para proteger a un perro que ya utiliza insecticidas tópicos, preferentemente en forma de collares.

No se recomienda el uso de la vacuna para el coronavírus entérico canino (CCov) una vez que este virus se considera de menor importancia clínica como patógeno entérico primario, causando solo una diarrea leve en los cachorros. La enfermedad entérica más grave se produce cuando, además del CCoV, hay una coinfección por CPV2. Los estudios publicados han demostrado que las vacunas de CCoV no reducen la infección vírica o la excreción fecal del virus. Además, la vacuna inyectable contra el CCoV no provoca un aumento de la concentración de anticuerpos IgA específicos en las heces, lo que se cree que es la base de la protección inmunológica. Igualmente, no se recomienda la utilización de la vacuna contra Giardia porque no hay suficientes pruebas científicas que justifiquen su uso. La evidencia de que la vacuna puede prevenir la eliminación de quistes, o la infección, es débil. La enfermedad en los perros no es mortal, raras veces es zoonótica y responde bien a los medicamentos.

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STATE-OF-THE-ART LECTURE: LEISHMANIASIS OR LEISHMANIOSIS? TWO TERMS, ONE HEALTH

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ABSTRACT BODY:

The World Health Organization defines One Health as "an approach to designing and implementing programmes, policies, legislation and research in which multiple sectors communicate and work together to achieve better public health outcomes. Leishmaniases are a group of diseases caused by protozoa belonging to the genus Leishmania, that affected humans and different animal species. The diseases can be categorized into two types according to the primary reservoir hosts of the human infection: zoonosis and anthroponosis. Zoonosis refers to an infectious disease of animals that can be transmitted to humans, and anthroponosis refers to a naturally occurring infectious disease among humans. The majority of the Leishmania species are involved in zoonotic transmission. Leishmania parasites are transmitted by phlebotomine sand flies, and the disease is zoonotic in most settings. These diseases have been reported both, in the "Old World" and "New World", with different clinical manifestations. From a clinical point of view, human leishmaniasis is often divided in 3 distinct forms: visceral leishmaniasis (VL), with the involvement of internal organs, cutaneous leishmaniasis (CL), characterized by single or multiple cutaneous lesions, and mucocutaneous leishmaniasis in which there is the partial or total destruction of mucous membranes of the nose, mouth and throat (Rocha et al., 2022). This last form is rarer than the first two and usually circumscribed to few Countries of Latin America and Ethiopia. VL is caused by parasites of the *Leishmania donovani* complex (*L. donovani* in the Old World and *L. infantum* in both the Old and New Worlds) being responsible for causing a severe disease which is lethal when untreated. While *L. donovani* transmission is anthroponotic, also if some infections have been detected in different animals (Kushwaha et al., 2021), the life cycle of *L. infantum* is mostly zoonotic. Due to the complex relationship between human, animal hosts, parasites, and sand fly vectors, the transmission of Leishmania spp. is intricate. Moreover, vector-borne diseases are influenced by environmental changes and socioeconomic factors such as poor housing and sanitary conditions, malnutrition, or population movement. Dogs are considered the main domestic reservoirs of VL caused by *L. infantum*. CL, caused by several species of Leishmania and responsible for considerable morbidity in endemic foci. While the estimated annual incidence of VL is of 50,000 to 90,000 cases, CL incidence is of 600,000 to 1 million cases. The clinical classification between visceral, cutaneous and mucocutaneous forms is not applicable to canine leishmaniosis (CanL) that is characterized by a broad spectrum of clinical signs, ranging from asymptomatic/subclinical infection, to severe, fatal disease condition. The latter is associated to elevated humoral T-helper 2 unbalanced response, leading to diffuse immune complexes deposition in several organs The severity of clinical picture is often characterized by a large variety of skin lesions which has been associated with the ability to transmit *L. infantum* back to the sand fly population (Gizzarelli et al., 2021). Occasionally, dogs may host Leishmania species different from *L. infantum*: *L. major and L. tropica* in the Old World and several *Leishmania species* in Latin America, mainly *L. brazilensis*. For all these species, the role of the dog as reservoir is under investigation. The clinical signs caused by the Leishmania species different from *L. infantum* consist of variable cutaneous and or mucocutaneous lesions. Interestingly, *L. infantum* may cause the same skin lesions without damage to the other organs, both in humans and dogs.

Climate change and man-made alterations of the environment such as deforestation also have a significant impact on leishmaniasis. A warming climate can affect the movement of vectors and reservoir animals. For this reason, Leishmaniases constitute a perfect medical model that should be approached on One Health context. The term leishmaniosis has been proposed to indicate the animal disease, mainly the well-recognized canine and feline leishmaniosis (CanL and FeL) caused by *Leishmania infantum*. On the contrary, the term Leishmaniasis is used to describe the human diseases. Taking the complexity and of the Leishmaniases epidemiological factors into account, it is not useful to distinguish between human and animal diseases that are worldwide considered re-emerging in endemic areas and emerging in non-endemic areas also for the increasing human influence

on the environment. The One Health approach is the essential key to controlling the disease, the rapid advancement of various diagnostic tests in recent decades has improved early detection and accurate species identification. Considering the relationship between zoonotic diseases and spillover events, the use of synanthropic animals as sentinels is vital for surveilling zoonotic leishmaniases in the human population. Future efforts must be directed towards the development of cost-effective and more accurate, accessible tests designed for field use in remote areas of endemic regions (Hong et al., 2020).

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VETERINARIO- PACIENTE - FAMILIA : TRÍADA ESENCIAL PARA EL BIENESTAR ANIMAL

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ABSTRACT BODY:

VETERINARIO-PACIENTE y FAMILIA . TRIADA ESENCIAL PARA EL BIENESTAR ANIMAL

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Como veterinarios comenzamos tratando animales de compañía y al poco tiempo nos damos cuenta que tratamos familias.

La importancia de comprender el vínculo humano/ animal en la Clínica diaria.

La rama de la ciencia que se dedica al estudio de los vínculos entre los animales y el ser humano es la antrozoología.

Somos una especie social con emociones, por lo que formamos vínculos emocionales con otros seres humanos y animales.

Si nos ponemos en contacto con un animal , y repetimos el encuentro, de manera espontánea se desarrolla un vínculo .

Este vínculo se describe como una relación mutuamente beneficiosa entre personas y animales que es esencial para la salud y el bienestar de ambos.

Es un vínculo profundo y fuerte y cualquier circunstancia que genere un sentimiento de miedo , estrés o dolor en un animal de compañía, indefectiblemente generará la misma sensación en su tutor .

En algunos casos es exacerbado y deriva en lo que se denomina antropomorfismo (todos iguales al ser humano) . Derivando muchas veces en problemas serios como por ejemplo exceso de alimentación y obesidad

En el otro extremo …está el maltrato animal , el descuido, la crueldad y el abandono

1 de cada 100 perros o gatos que atendemos en el consultorio sufre maltrato.

En los hogares donde hay maltrato animal probablemente haya maltrato infantil, y los niños maltratados en el hogar, son más propensos a maltratar los animales y a volverse violentos con los seres humanos.

El abandono ,miedo y el dolor son las sensaciones mas terribles que un animal puede experimentar. El trauma que generan puede quedar grabado como una impronta en el cerebro que no se olvida.

Como veterinarios tenemos que estar atentos y considerar de manera proactiva el maltrato animal como un diagnóstico diferencial .

Los veterinarios somos , y debemos esforzarnos por ser los principales defensores del bienestar de los animales .

Bienestar

El bienestar animal es un tema emotivo , y puede significar diferentes cosas para diferentes personas .

El bienestar contempla la salud física y la salud emocional de un animal en el contexto en que vive y muere .

En particular, cómo se siente el animal , como es su vida .Es feliz?

Que implica ser feliz para un animal?

Maximizar el bienestar animal para nuestros pacientes permite mejores resultados clínicos y quirúrgicos, y mejores relaciones veterinario-paciente-propietario. Por el contrario, el estrés, el miedo, la ansiedad y/o el dolor en nuestros pacientes animales pueden tener profundos efectos clínicos, tanto físicos como psicológicos.

En el consultorio veterinario, la falta de lenguaje verbal genera una tensa expectativa por parte de nuestro paciente , que no sabe y no comprende lo que va a suceder en ese momento.

Si somos capaces de comprender el lenguaje corporal y percibir como se sienten ,transmitir tranquilidad con nuestra forma de abordaje para minimizar esas sensaciones , podemos generar un mejor vínculo , inspirando confianza en nuestro paciente y en el tutor ,esencial para llegar al diagnóstico .

Los animales responden directamente al medio que los rodea.Hay que evitar o minimizar las posibles causas de estados emocionales negativos en la clínica.

Nuestro paciente es un ser animal , que tiene sentimientos . Para poder evaluarlo , debo tener en cuenta como se siente

Las diferentes especies tienen sus popias emociones .Mediante la observación podemos llegar a comprender como expresan esas emociones .

Ejercitar la paciencia . La consulta no debe ser apurada

, el tiempo que dedicamos a la charla previa , que en general no tiene que ver con el problema en sí, permite que el tutor baje su nivel de estrés .Es un buen momento para explicar cual será la forma de abordaje que llevaremos a cabo y que el animal reconozca el lugar y tome confianza .

El tutor siente la necesidad de conocer y comprender , y necesita sentir que es reconocido y comprendido. De esa manera colaborará con el tratamiento.

De todas las emociones vividas en la Clínica , la peor es el miedo Los animales pueden adaptarse al dolor , pero no al miedo .

Los perros y gatos , se acuerdan de todo lo negativo y lo positivo. Tienen memoria.

La Comunicación

La comunicación se realiza en relación con las personas y los animales.VET y el tutor , VET y su paciente y entre el Tutor y su animal .

La comunicación , es un intercambio verbal y no verbal.

El consultorio, probablemente es el medio de comunicación mas importante para el veterinario.

Podemos ser los mejores clínicos, los mejores cirujanos, grandes especialistas, pero, si la comunicación con el tutor falla, es posible que no comprenda lo que le decimos y no pueda seguir nuestro consejo.

La comunicación es un arte.Y actualmente en plena era de la comunicación , estamos perdiendo lo esencialque es saber escuchar .

El conocimiento , la percepción , la empatía y la forma de transmitir el mensaje verbal y no verbal nos permitirán una buena comunicación.

Para que la interacción sea clara, los tres elementos , pensamiento, emoción y cuerpo deben estar en perfecta armonía.

Cuando las personas comprenden un problema en profundidad , se inclinan a aplicar el conocimiento a la práctica.

Lo importante es escuchar lo que está diciendo , e interpretar lo que esta sintiendo.

Como dar una mala noticia

Es muy difícil dar una mala noticia , la mayoría de las personas, no está preparada para recibir malas noticias.

Comprender la angustia que experimenta una persona cuando siente la posible pérdida de su ser tan querido , y saber acompañarlo es una parte ineludible del trabajo veterinario.

Podemos ayudar a disinuir la pena según como comuniquemos . Hay que preparar a la familia para que comprendan como se desarrolla la enfermedad y darles herramientas para tomar desiciones al final de la vida

Fin de la vida

Nuestra misión como médicos es cuidar y proteger a los animales sanos y enfermos con el respeto y la dignidad que merece todo ser vivo, y brindar la mejor atención a un animal que sufre(enfermo) sin olvidar que la vida de los seres vivos tiene un límite natural e inexorable.

Poder humanizar el morir (como proceso y la muerte como momento) es respetar la vida.

Poder transmitir que la vejez no es una enfermedad, y que por lo tanto no es curable. Y ayudarlos a evaluar si la vejez en una terapia intensiva puede generar una situación de estrés y extrema soledad porque a veces se pueden brindar cuidados especiales en el caso de los animales añosos en el domicilio .

Tenemos que dejar de pensar como médicos que hay que luchar contra la muerte que es un proceso natural, sino saber acompañar en el buen morir y la muerte con dignidad

LIDER EN LA VETERINARIA,DEBE ENAMORARSE?

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ABSTRACT BODY:

En un mundo que avanza tan rapido con constantes cambios, los participantes del sector pet tienen que adaptarse a ello. Aprovechando así las nuevas tendencias, actuando muy rápido en lo que sucede y sacar el mayor beneficio para sus organizaciones. Muchas veces es el propio veterinario emprendedor que inicia su propio negocio, sin importarle temas organizacionales, sin saber que significa ser lider, o pensando que ser jefe es lo mismo. Este veterinario tomará el control de su organización de manera administrativa y médica, haciendo lo que puede, con los mismo pensamientos y creencias con los que vive el día a día. El líder esta obligado a optimizar tiempos y recursos, a sacar su organización adelante, haciendo que los clientes esten bien atendidos y que su entorno sea feliz. Cuando una persona se enamora de algo o alguien, sea bueno o sea malo, le destinará recursos, siendo aquí el tiempo. Este tiempo que le ofrece a un momento que ya pasó, le quitará oportunidades a uno próximo, que vendrá solo o en su mejor efecto, siendo pro activo, éste lo buscará y creará. El lider, dueño, emprendedor y muchas veces trabajador de su organización muy frecuentemente se enfrasca en situaciones que lo marcarón y ya sucedieron, viviendo así de sueños. Estos por más que hayan sido buenos o malos, ya tienen que quedar atrás y enfocarnos en lo que podemos crear. Ya que vivimos en un mundo que cambia tan rápido y en esas nuevas situaciones, se presentar nuevas oportunidades que podemos descubrir para hacer crecer nuestra organización.

El fin de toda organización que se crea, es de producir riqueza para los accionistas, mediante ello, se tejeran circunstancias en el medio que será ayudar y hacer crecer a nuestro colaboradores, atenciones adecuadas, clientes felices con ganas de volver y mascotas con mejor estilo y calidad de vida. Haciendo las cosas bien, se logrará hacer girar ese circulo, cada vez mas grande y más rapido, llegando así al objetivo de la producción de riqueza como consecuencia de hacer bien las cosas, adaptandonos y aprovechando lo que nos brinda los nuevos comportamientos que adopta el lider.

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HOW I DIAGNOSE HEART FAILURE IN DOGS AND CATS RADIOGRAPHICALLY

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ABSTRACT BODY:

Radiography remains important for overall screening of the thorax, assessment of the lungs for edema or diseases that can have similar clinical signs to that of cardiac disease such as bronchitis, and for assessment of the pulmonary vasculature, tracheal diameter, and lung volume. A minimum of two correctly exposed and positioned orthogonal views performed on inspiration is required. A left lateral and a dorsoventral are most commonly used, however, this varies by institution. I currently perform a 4-view examination for any and all thoracic radiographs. DV radiographs allow the best assessment of the caudal lung lobes for signs of cardiogenic edema due to left heart failure. Most important is that the SAME views are performed each time so that they can be compared. Heart patients will be radiographed many times during their illness to assess therapy and recognize signs of decompensation and similar positioning is critical.

Cardiomegaly

The diagnosis of heart failure begins with recognition of cardiomegaly and shape changes of the cardiac silhouette. The pulmonary vasculature and caudal vena cava are also scrutinized for distention in both left and right heart failure cases. Endocardiosis is a degenerative disease that affects the AV valves (mitral and tricuspid) causing insufficiency (regurgitation) usually in older, small breed dogs and along with dilated cardiomyopathy are the most common causes of left heart failure leading to pulmonary edema. Depending on the phase of decompensation (failure) the left atrium gradually increases in size, the trachea is displaced dorsally as the entire heart enlarges, the stem bronchi are displaced dorsally, the left auricle enlarges (DV view 2-3 o'clock), the pulmonary veins dilate (congestion), and the lungs progress from an interstitial pattern to a coalescing alveolar pattern that is usually centrally located but can be atypical in location (ventral or only right-sided). Signs of right heart failure may also occur.

Diagnosing heart disease in cats is challenging since heart murmurs are not always present and or if they do they are not always associated with functional or structural changes in the heart. Clinical signs can be nonspecific. Furthermore, some of the most common myocardial diseases may not result in size or shape changes radiographically. However, thoracic radiography assessment for cardiac disease is important and includes the heart size and shape, the size and shape of the pulmonary arteries and veins as well as the lung opacity. Lateral and ventrodorsal views of the thorax are adequate for assessing a cat for cardiac disease and heart failure. The width of the heart on the lateral image should be no more than 2-2.5 intercostal spaces. The vertebral heart score can alternatively be used and is useful as a quantitative measure of size when assessing serial radiographs over the course of disease and treatment.

To perform the vertebral heart score, measure the distance between the ventral aspect of the carina and cardiac apex using the digital calipers of the imaging software or use a piece of paper on analog film images. Then, take a second measurement perpendicular to the first at the widest point of the heart. Compare the length of these two lines with the thoracic vertebral length starting at the cranial endplate of Th4. Count the vertebral bodies that correspond to the length of the two lines and add them. This is the VHS. Normal values in dogs are 8.5-10.5 and in cats are 7.5 +/- 0.3.

The paired pulmonary arteries and veins must be scrutinized for distention and can help to support a diagnosis of heart failure. The cranial lobar artery should be equal to slightly smaller than the vein. The vein should not be greater than ¾ of the width of the proximal third of the 4th rib in the lateral view. In the DV image, the caudal lobar vein should make a square shape where it crosses the 9th rib. An enlarged vein will have an increased width where it crosses the rib and have the shape of a wide rectangle.

Cor Pulmonale

The term cor pulmonale refers to right heart enlargement due to lung disease that causes a disturbance of the pulmonary blood flow (dirofilariasis, obstructive or restrictive respiratory disease) and pulmonary hypertension.

Radiographically, the right heart and main pulmonary artery are enlarged. The peripheral pulmonary arteries can become tortuous and changes of the pulmonary parenchyma can be seen (mainly increased opacities, mixed patterns).

Developmental disease of the pericardium: The cardiac silhouette becomes round like a soccer ball (DV view mainly). The pulmonary vasculature will be reduced in size, the lung will appear more lucent and

the vena cava will be a larger diameter than the aorta. The liver may become enlarged and loss of serosal abdominal detail due to ascites may occur. Heart-based tumors and pericarditis appear similarly in radiographs. Echocardiography is required to confirm a heart-based or right atrial/auricular tumor.

Radiographic findings of right heart failure

Right heart enlargement or generalized cardiomegaly is present and there is an increase in the diameter of the caudal vena cava where the caudal vena cava diameter is greater than the descending aorta. Hepatosplenomegaly, pleural and peritoneal effusion are the main findings.

Radiographic findings in left heart failure

Pulmonary venous congestion: The veins will have a larger diameter than the arteries.

First, interstitial edema occurs: the perihilar pulmonary vessels become ill-defined. Later, these changes occur in the periphery of the lung (unstructured interstitial pattern).

Alveolar pulmonary edema: As the interstitial pattern becomes worse, coalescing patchy areas of soft tissue opacities will be recognized the usually progress to become alveolar. The right caudal lobe may be more affected than the left. In some breeds such as the Doberman, an ill-defined bronchocentric pattern can develop in heart failure that is atypical.

The presence of lung opacities in a cat with suspected heart disease or signs of cardiomegaly increases the likelihood of cardiogenic pulmonary edema. The opacities can range from interstitial to severe air space localizations and can be perihilar or multifocal. A patchy uneven distribution is common in cats. Pleural effusion in the cat can be a sign of left heart failure and the pathophysiology is poorly understood and controversial in the literature. Anatomical drainage of the pleural via the thoracic duct is the current theory. A recent study has shown that a normal left atrial size on thoracic radiographs does not rule out the presence of left-sided congestive heart failure in cats with clinical signs of respiratory distress. Approximately half of the cats in failure in this study had left atrial enlargement and the other half did not.

HOW I TREAT … CANINE PERIANAL FURUNCULOSIS

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ABSTRACT BODY:

Inflammatory perianal disease (canine perianal fistulas/furunculosis)

Canine perianal furunculosis or canine perianal fistulas is a chronic inflammatory disease of the perianal and peri-rectal tissues characterized by the development of ulcers and ulcerative-fistulous tracts. It is a severe disease, that causes gastrointestinal, systemic, and behavioral disorders in the affected dogs. The disease affects dogs 4 to 8 years old, and males and females are equally affected. 80% of the cases are reported in German and Belgian shepherd dogs, but other breeds are also affected (setter, collie, Chihuahua,). About one-third of affected dogs suffer also from or have a history of atopic dermatitis. The reason for consultation is usually dyschezia, increased number of defecations or perianal pain. In the dermatologic examination, the affected dogs present one or several perianal ulcers, from 1 mm to several cm in diameter, and fistulous tracts up to 5 cm deep. Rarely true fistulas are present. Considering this point, it would be probably better to name the entity inflammatory perianal disease (IPAD). A mucopurulent or purulent-hemorrhagic discharge and areas of scarring and hyperpigmentation are also common. Behavioral changes (aggressiveness), dyschezia, tenesmus, constipation-diarrhea, and weight loss are also common clinical signs.

The diagnosis is clinical, based on history and clinical picture. Clinical pathology shows non-specific alterations, usually not very marked.

Although several surgical techniques have been reported (tail amputation, cryosurgery, wide surgical resection including anal sacs), all have limited efficacy and post-surgical complications are common. Currently, medical management of the disease is recommended.

Medical treatment

My treatment for a case of canine perianal fistulae is as follows:

- Ciclosporin 7,5 mg/kg/12h. This dose is maintained until the ulcers close. After that, the dose is tapered and treatment with topical tacrolimus is started. If there is no improvement with this dose of ciclosporin, an evaluation of the serum levels of ciclosporin 2h after administration is indicated.
- Prednisone 1mg/kg/24 can be added for 1-2 weeks if the inflammation is very severe (optional, to accelerate the improvement).
- Washing of the perianal region with a chlorhexidine solution or shampoo.
- Tacrolimus 0.1% cream. Once the ulcers are epithelized, tacrolimus cream is applied twice daily on the perianal skin, after the washing.
- Pain management. Tramadol or any other painkiller medication should be prescribed until the lesions are healed.
- Diet change. Some authors recommend that all dogs go through a hypoallergenic diet (8 weeks). I only recommend the diet if there are other clinical signs suggesting the possibility of a food adverse reaction (pruritus, erythema in other body sites, otitis). In most cases, I recommend a diet high in fiber.
- Antimicrobial treatment. If the dog presents clinical and cytologic signs of pyoderma, a systemic antibiotic/antimicrobial can be prescribed (especially if the lesions are not improving with the chlorhexidine washes). It is recommended to perform culture and sensitivity testing for selecting the most adequate antibiotic.

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BASIC EXTRACTION TECHNIQUES

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ABSTRACT BODY:

DENTAL EXTRACTIONS MADE EASIER

Step 1: **OBTAIN CONSENT**

NEVER extract teeth without owner consent (preferably written), no matter how bad the problem, or how obvious the decision is. Make sure that you have a valid daytime number (or numbers) for the client and inform them they must be available during surgery hours. Consider loaning pagers to clients for the day, as this author has found this to be a very effective means to contact clients. If the client cannot be reached and prior consent was not obtained, DO NOT PULL THE TOOTH. Document the problem, recover the patient, and reschedule the work. Remember, the tooth can always be extracted later, but it cannot be put back in!

Step 2: **DENTAL RADIOGRAPHS**

Dental radiographs should be exposed on all teeth prior to extraction. Dental radiographs are invaluable resources for the practiconer. Radiographs allow the practitioner to determine the amount of disease present, any root abnormalities or ankylosis. Help with radiographic interpretation is available while the patient is under anesthesia at www.vetdentalrad.com. In addition, the radiographs will serve as evidence for the extraction in the medical record. Radiographs should also be exposed post-extraction to document complete removal of the tooth.

Step 3: **PAIN MANAGEMENT**

Extractions are surgical procedures and are moderately to severely painful for the patient. Depending on patient health, a multimodal approach (combination of opioids, NSAIDs, local anesthetics) should be employed, as this provides superior analgesia. Preemptive analgesia is proven to be more effective than post-operative, and it is therefore important to administer the drugs BEFORE the painful procedure.

SINGLE ROOT EXTRACTIONS

Step 4: **INCISE THE GINGIVAL ATTACHMENT**

This is accomplished with a scalpel blade (number 11 or 15), elevator, or luxator. The selected instrument is placed into the gingival sulcus with the tip of the blade angled toward the tooth (this will help avoid going outside the bone and creating a defect or cutting through the gingiva). The blade is then advanced apically to the level of the alveolar bone, and the instrument is carefully worked around the entire tooth circumference.

This step is very helpful as the gingival attachment contributes approximately15% of the retentive strength of the periodontal apparatus. More importantly, however, this procedure will keep the gingiva from tearing during the extraction procedure.

Step 5: **ELEVATE THE TOOTH**

Elevation is the most dangerous step in the extraction procedure. Remember that you are holding a sharp surgical instrument and working in an area of numerous critical and delicate structures. There have been many reports of eyes that have been gouged and lost by extraction instruments as well as at least one confirmed fatality due to an elevator puncturing a patient's brain. The index finger is placed near the tip of the instrument to avoid causing iatrogenic trauma in the event of instrument slippage or encountering diseased bone. In addition, the jaw should be gently held with the opposite hand to provide stability and avoid mandibular fracture.

First, select an instrument which matches the curvature and size of the root. There are numerous instruments available including the classic elevator, the luxating elevator, and the winged elevators. Classic elevators and winged elevators are used in an "insert and twist" motion to tear the periodontal ligament, whereas luxators are used in a rocking motion during insertion to fatigue as well as cut the periodontal ligament. Luxators can be GENTLY twisted for elevation, but they are not designed for this and can be easily damaged when used in this manner.

Elevation is initiated by inserting the elevator or luxator firmly yet gently into the periodontal space. The insertion should be performed while keeping the instrument at about a 10 to 20 degree angle toward the tooth, to avoid slippage. Once in the space between the bone and the tooth, the instrument is gently twisted with two-finger pressure. This is not to say that the instrument should be held with two fingers, rather the entire hand should be used to hold the instrument. Twist only with the force that you could generate when holding with two fingers. Hold the position for 10-30

seconds to fatigue and tear the periodontal ligament.

It is important to note that the periodontal ligament is very effective in resisting intense, short forces. It is only by the exertion of prolonged force (i.e. 10-30 seconds) that the ligament will become weakened. Heavy stresses only serve to put pressure on the alveolar bone and tooth which can result in the fracture of one of these structures, so it is important not to use too much force.

After holding for 10 to 30 seconds, reposition the instrument about 1/8 of the way around the tooth and repeat the above step. Continue this procedure 360 degrees around the tooth, each time moving the elevator apically as much as possible. Depending on the level of disease and the size of the tooth, a few to several rotations of the tooth may be necessary.

The key point to successful elevation is PATIENCE. Only by slow, consistent elevation will the root loosen without breaking. It is always easier to extract an intact root than to remove fractured root tips.

Step 6: **EXTRACT THE TOOTH**:

Removing the tooth should only be attempted after the tooth is very mobile and loose. This is accomplished by grasping the tooth with the extraction forceps and gently pulling the tooth from the socket. Do NOT apply undue pressure as this may result in root fracture. In many cases, especially with premolars, the roots are round in shape and will respond favorably to *gentle* twisting and holding of the tooth while applying traction. This should not be performed if there are root abnormalities (significant curves, weakening) seen on the pre-operative radiograph.

It is helpful to think of the extraction forceps as an extension of your fingers. Undue pressure should not be applied. If the tooth does not come out easily, more elevation is necessary. Start elevation again until the tooth is loose enough to be easily removed from the alveolus.

Step 7: **CLOSURE OF THE EXTRACTION SITE**:

This is a controversial subject among veterinary dentists, and thus some texts recommend suturing only in large extractions, other authors (including this one) recommend suturing almost all extraction sites. Closure of the extraction site promotes hemostasis and improve post-operative discomfort and aesthetics. It is always indicated in cases of larger teeth (e.g. canines, carnassials), or any time that a gingival flap is created to allow for easier extraction. This is best accomplished with size 3/0 to 5/0 absorbable sutures on a reverse cutting needle. Closure is performed with a simple interrupted pattern with sutures placed 2 to 3 mm apart. It is further recommended to utilize one additional throw over manufacturer's recommendations to counteract tongue action.

In regards to flap closure, there are several key points associated with successful healing. The first and most important is that there must be no tension on the incision line. If there is any tension on the suture line, it will not heal. Tension can be removed by extending the gingival incision along the arcade (called an envelope flap) or by creating vertical releasing incisions and fenestrating the periosteum. The periosteum is a very thin fibrous tissue which attaches the buccal mucosa to the underlying bone. Since it is fibrotic, it is inflexible and will interfere with the ability to close the defect without tension. The buccal mucosa is very flexible and therefore will stretch to cover large defects. If there is no tension, the flap should stay in position without sutures.

EXTRACTION OF MULTI ROOTED TEETH:

Section all multi-rooted teeth into single rooted pieces. The roots of almost all multi-rooted teeth are divergent and this will cause the root tips to break off if extractions are attempted in one piece. Root fracture can occur even if a tooth is relatively mobile to start with. With mobile teeth, the sectioning step alone often allows for simple extraction.

The best tool for sectioning teeth is a bur on a high-speed air driven hand piece. Besides being the quickest and most efficient tool for the job, it also has air and water coolant that will avoid overheating the tooth. Many different styles of burs are available, however this author prefers a cross-cut taper fissure bur (699 for cats and small dogs, 701 for medium dogs and 702 for large breeds).

The best way to section the teeth is to start at the furcation and work towards the crown of the tooth. This method is used for two major reasons. First, it avoids the possibility of missing the furcation and cutting down into a root, which subsequently weakens the root and increases the risk of root fracture. In addition, this method avoids the possibility of cutting through the tooth and inadvertently damaging the gingiva or alveolar bone.

After the tooth has been properly sectioned, follow the above steps for each single rooted piece. In some cases, the individual tooth pieces can be carefully elevated against each other to gain purchase.

NON ULCERATIVE CORNEAL DISORDERS IN CATS

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ABSTRACT BODY:

NON ULCERATIVE CORNEAL DISEASE IN CATS

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Bullous keratopathy

It is a rare disease that causes acute corneal oedema caused by lamellar separation and disorganisation. The cornea gets a conical shape, and it loses its normal profile. It can be focal, geographic, or complete. The cause is unknown. The progression of the corneal oedema tends to be quick leading to perforation that could end up leading to enucleation. It is a condition that tends to be unilateral however both eyes can be affected. It affects young animals however older cats can also be affected.

The ocular signs are non-specific with lacrimation, blepharospasm and conjunctival hyperaemia. An oedematous bulla can appear within hours and can deteriorate quickly. Pockets of oedema are usually visible.

The main differential diagnosis is a melting ulcer in any breed of cat. In Manx cats that suffer from progressive oedema, the advanced stage of the disease is also described as a bullous keratopathy.

The cause is unknown however there are several theories that have been proposed:

- Defect in Descemet's membrane and endothelium
- Primary or hereditary endothelial dystrophy
- Endothelial dysfunction

In human ophthalmology the cause is a break in Descemet's membrane.

When it comes to histology there is no evidence of inflammation. Cytology and culture are negative and the concurrent systemic diseases appear to be coincidental. It appears that the use of systemic cyclosporine is a risk factor for the development of the disease.

Treatment

If the defect that forms is small, we could attempt to treat with medical treatment using antioedema drops (sodium chloride 5%) and antibacterial drops. If we are dealing with a large defect, tectonic support will be needed. In these cases, many techniques have been described but our choice would be a nictitating membrane flap.

Eosinophilic keratoconjunctivitis

It is an infiltrative and progressive corneal disease that can affect cats of any age however adult cats around 4-6 years of age are usually more predisposed. This disease has also been reported in horses, dogs, and rabbits. In 66% of cases is initially unilateral but tends to end up being a bilateral disease.

Regarding the aetiopathogenesis there has been an association with Feline Herpesvirus-1 reported but this remains unclear.

The ocular signs are initial perilimbal vascularisation and white nodular deposits that progress to form a vascularised mass surrounded by corneal oedema. The classical location is dorsolateral, but any part of the cornea can be affected.

The clinical presentation is characteristic: ¨cottage cheese¨ like deposits. They are accompanied by blepharospasm, white ocular discharge and conjunctival hyperaemia. Concurrent dermatological disease has not been reported. Most of these cases have an associated superficial corneal ulcer.

The diagnosis is made by the clinical appearance and by corneal cytology. Only with one eosinophil found on cytology we can confirm the diagnosis.

The treatment will be local immunosuppression. Prednisolone acetate 1% or dexamethasone 0.1% every 8-12 hours is a good starting point. We need to achieve a maintenance dose and again, carer education is very important. Management rather than cure is likely to be achieved in these cases. Cyclosporine, megestrol acetate (topical and systemic) and subcutaneous triamcinolone have also been reported. Do we also give systemic antivirals if we suspect FHV-1 could be involved?

Corneal sequestrum

It has also been described in dogs and horses. The clinical appearance is of brown decolouration of the affected stroma. The colour can progress from ambar to black and the depth can progress from superficial to deep.

The aetiopathogenesis is speculative. It is thought that a corneal insult starts the process and that cell apoptosis contributes. Ulcerative keratitis, lagophthalmia, entropion, trichiasis, tear film

abnormalities, grid keratotomy and FHV-1 are all predisposing factors. There are certain breeds such as Persian, Burmese, Siamese, Himalayan and domestic short hair that are predisposed. The brachycephalic breeds have lagophthalmos, medial entropion and trichiasis. More attention is being recently paid to a disfunction of the tear film.

The corneal pigmentation characteristic of this disease is not melanin.

The clinical signs are typical. It is usually unilateral, and it brachycephalic breeds can also be bilateral. It tends to be an oval or round area in the axial/paraxial cornea. Ambar colouration extends to all areas. Depth is difficult to calculate with the slit lamp and sometimes, using high resolution ultrasound may be helpful. Signal reflectivity and its absorption can limit depth assessment in OCT.

Cats with corneal sequestrum show blepharospasm, ocular discharge, conjunctival hyperaemia, variable vascularisation, and a superficial ulcer on the edge of the sequestrum.

Histopathology shows that the epithelium is absent over the sequestrum and that it is poorly attached on the edges. Coagulation necrosis is observed.

Treatment involves resolution of the cause and resolution of the sequestrum. Treatment for sequestrum is most times surgical. A superficial keratectomy is the technique of choice, and a graft may or may not be needed depending on depth. Types of grafts are corneoconjunctival transposition, amniotic graft, BioSis, A-Cell, corneal transplant, etc. The choice depends on surgeon's choice. It is thought that if residual pigmentation is left after the keratectomy, the chances of recurrence increase.

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ESTADIO CLÍNICO:ES UN TUMOR LOCALIZADO O HAY METÁSTASIS?

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ABSTRACT BODY:

ESTADIO CLÍNICO: ¿ES UN TUMOR LOCALIZADO O HAY METÁSTASIS?

Cuando diagnosticamos una neoplasia en nuestros pacientes, debemos ofrecer a los propietarios información sobre el pronóstico y las opciones terapéuticas disponibles. Esta información depende, además del tipo concreto de neoplasia, del estadio clínico (extensión de la enfermedad tumoral en el cuerpo). Además de los estudios necesarios para conocer el estadio clínico, es necesario conocer el estado general del paciente, mediante la historia clínica, examen físico y analítica. La historia debe incluir medicaciones, enfermedades o cirugías previas, especialmente de cualquier masa o tumor que se haya extirpado y revisar la histopatología. El examen debe ser completo, incluyendo examen rectal. En casos en los que se valore una amputación, el examen ortopédico y neurológico es imprescindible para valorar si el paciente es un buen candidato. La analítica sanguínea y de orina nos ayudará a conocer la función renal y hepática en animales que pueden necesitar múltiples anestesias (radioterapia) o recibir medicaciones hepato o nefrotóxicas. Una vez completados la historia clínica, el examen físico, analíticas y el estudio de la extensión del tumor puede ofrecerse información pronóstica y valorar la terapia más efectiva. Conocer el estadio clínico previo al tratamiento, nos permite además, tener un valor inicial para poder valorar la respuesta a la terapia.

La cirugía ha sido tradicionalmente el tratamiento local de los tumores. Ahora hay disponibles otras terapias locales como la radioterapia, electroquimioterapia o ablación por radiofrecuencia pero antes de recomendar estas técnicas, hemos de estar seguros de que no hay enfermedad a distancia. En el caso de terapias sistémicas, a la quimioterapia tradicional se han unido en los últimos años la quimioterapia metronómica o los inhibidores tirosin quinasa. Para poder valorar la respuesta de estos tratamientos (remisión parcial o completa, enfermedad estable, o enfermedad progresiva) es necesario conocer en el momento que se inicia la terapia, la extensión de la enfermedad para poder valorar la eficacia de estos tratamientos.

Así pues, en todos los casos oncológicos, además del **diagnóstico** del tumor, es necesario conocer el **estadio clínico** de cada paciente, que puede hacer que dos animales con el mismo tumor reciban tratamientos distintos si tienen estadios clínicos diferentes. Las pruebas necesarias en cada caso, dependen del tumor primario y el comportamiento biológico esperado para esa neoplasia.

Aunque hay clasificaciones específicas para cada grupo tumoral, en general la clasificación se basa en los sistemas **TNM**, que incluyen datos del tumor primario (**T**), la afección de los nódulos –ganglios- linfáticos (**N**), y la presencia o ausencia de metástasis (**M**) a distancia.

Una vez diagnosticado, mediante citología o histopatología, el tumor primario debe medirse y registrar su tamaño y localización. En la mayoría de tumores, el **tamaño** es un importante factor pronóstico. Junto con el tamaño, la **localización** del tumor ofrece, en algunos tumores, importante información pronóstica: el melanoma oral canino tiene mejor pronóstico si se localiza en mandíbula rostral.

Para el estudio de algunos tumores son recomendables pruebas de imagen avanzadas como la tomografía. En tumores de la cavidad nasal, la extensión del tumor se utiliza para conocer el estadio clínico. Otro factor a tener en cuenta es la **invasión de los tejidos adyacentes**.

Igualmente importante es la evaluación clínica de los ganglios linfáticos para poder determinar la extensión de la enfermedad tumoral. El riesgo de metástasis a ganglio en el momento del diagnóstico depende del tipo tumoral. Más de la mitad de los casos de melanoma oral canino, un tercio de los carcinomas de células escamosas orales en gatos, o un 20% de los fibrosarcomas orales caninos, tienen metástasis ganglionar en el momento del diagnóstico. El tamaño de los ganglios, ya sea mediante el examen físico o medido en pruebas avanzadas como la tomografía, no sirve como indicador de metástasis y el diagnóstico es citológico o histológico.

Para la evaluación de los ganglios es necesario conocer a qué ganglios drena cada área corporal, recordando que no siempre es al ganglio más cercano. Algunas zonas cutáneas drenan a ganglios abdominales como la zona perineal o la zona del sacro y articulaciones coxofemorales (1).

La valoración del tamaño no es definitiva en la mayoría de los casos: el hecho de que el tamaño sea normal no descarta la presencia de metástasis ganglionar. Por

otra parte, si el tamaño está aumentado, no significa obligatoriamente que haya metástasis. Aunque el método definitivo para diagnosticar metástasis es mediante la extirpación del ganglio y su análisis histopatológico, el aspirado del ganglio y citología muestran una buena sensibilidad y son generalmente el método que usamos.

El diagnóstico de metástasis se basa en la identificación de células de una población distinta a la linfoide pero en casos de células que pueden encontrarse de forma normal en los ganglios, como ocurre con los mastocitos, el diagnóstico es más complicado. Se ha propuesto (2) para el mastocitoma la probable metástasis (al menos un portaobjetos, con más de 3 focos de 2-3 mastocitos agregados y/o 2 focos de más de 3 mastocitos) y la metástasis cierta (presencia de agregados de células pobremente diferenciadas, con pleomorfismo, anisocitosis o anisocariosis, y/o más de 5 agregados de más de 3 mastocitos). Criterios similares se usan si el ganglio se analiza mediante histopatología.

Finalmente, debe evaluarse la presencia de metástasis a distancia. Aunque la diseminación metastática puede afectar a distintos órganos, los pulmones son los que se ven afectados en un mayor número de casos. La radiología de tórax, con tres proyecciones (laterales derecha e izquierda y otra ventrodorsal) es un método sensible para la detección de lesiones tumorales. Al compararlo con la tomografía, entre el 80 y el 95% de los casos con metástasis confirmada en TAC fueron detectados con radiología convencional. La principal diferencia es el número de nódulos detectados con cada técnica (mayor con TAC). Independientemente de la técnica utilizada, es importante que las imágenes sean valoradas por un radiólogo con experiencia (3). Cuando se evalúa el tórax por metástasis, un diagnóstico diferencial importante es la presencia de osteomas, lesiones benignas que se ven con frecuencia en perros mayores, con razas predispuestas como el bóxer. Son lesiones mineralizadas de menos de 3 mm que se observan en el parénquima pulmonar.

En el caso del mastocitoma, las metástasis a distancia afectan a hígado o bazo y no a pulmón. Más de un 30% de los mastocitomas de alto riesgo tienen metástasis en hígado o bazo en el momento del diagnóstico, pero la sensibilidad de la ecografía para diagnosticar metástasis de mastocitoma en hígado o bazo es muy baja y la recomendación es aspirar estos órganos independientemente del aspecto ecográfico en los pacientes de riesgo.

Distintos tumores tienen un comportamiento biológico distinto y en los carcinomas de mama hay diferencias en los órganos afectados dependiendo de que el carcinoma sea inflamatorio o no inflamatorio. En carcinoma no inflamatorio puede metastatizar a ganglios, pulmón, hígado, riñón y hueso, mientras que el inflamatorio metastatiza con frecuencia a vejiga de la orina y tracto reproductivo (4).

Los tumores mamarios son los tumores que con más frecuencia metastatizan a hueso, aunque otros tumores como los músculoesqueléticos o los uroteliales pueden hacerlo. Independientemente del tipo histológico, las vértebras y el húmero parecen ser los huesos afectados más frecuentemente. Los osteosarcomas metastatizan con frecuencia a huesos distintos de donde se localiza el tumor primario.

En resumen, para poder ofrecer información sobre el pronóstico, opciones de tratamiento y poder valorar la respuesta, y saber si hay otras lesiones, neoplásicas o no, es imprescindible conocer el estadio clínico en nuestros pacientes oncológicos. La calidad de las pruebas diagnósticas (radiografías, ecografías o citologías) y su interpretación (radiólogos, patólogos clínicos) son de gran importancia a la hora de la toma de decisiones clínicas por parte del oncólogo.

La presencia de metástasis no significa que no puedan tratarse algunos de estos casos. Por ejemplo, en mastocitomas de bajo grado, la extirpación de linfonodos con metástasis se asocia a un buen pronóstico, con una sobrevida de 4 años en el 90% de los casos.

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HERNIA DIAFRAGMÁTICA

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ABSTRACT BODY:

Una hernia diafragmática se define como aquel defecto en la pared diafragmática que permite el paso de contenido abdominal a cavidad torácica. El hígado suele ser el primer órgano en herniarse seguido del tracto gastrointestinal y el bazo.

Las principales causas de hernia diafragmática en pequeños animales son los traumatismos indirectos y contusos sobre el diafragma[1], aunque en animales jóvenes debe sospecharse de hernias diafragmáticas congénitas (un 5-10% de los casos de hernia diafragmática son congénitos).

Existen razas predispuestas a estas hernias peritoneo-pericárdicas como el Braco de Weimar mientras que los perros machos fértiles de 1 a 3 años son los más afectados por las hernias traumáticas

Tipos de hernias diafragmáticas:

Los tres tipos de hernias diafragmáticas son:

a. **Hernia Pleuroperitoneal:** Las traumáticas son las más habituales donde las vísceras abdominales se desplazan al espacio pleural. Las congénitas corresponden a un fallo en la fusión de la membrana pleuroperitoneal con el canal pleuroperitoneal durante el desarrollo del diafragma, es hereditaria autosomal recesiva en muchos casos los animales afectados nacen muertos o mueren a las pocas horas de nacer por estar asociado a anomalías pulmonares importantes, si sobreviven, el defecto suele ser amplio y a veces difícil de cerrar[2].

b. **Hernia Peritoneopericárdica:** Son las hernias diafragmáticas congénitas más frecuentes en las que se produce una comunicación entre el abdomen y el saco pericárdico. Se piensa que es debida a una lesión prenatal o defecto en el desarrollo del septo transverso y pliegues pleuroperitoneales. Pueden estar asociadas a otras anomalías congénitas como defectos de la pared abdominal craneoventral, defectos esternales caudales, defectos pericárdicos e intracardíacos (defecto en el septo ventricular). Esta combinación de anomalías congénitas suele ocurrir esporádicamente en perros y gatos, afectando más a razas como Weimaraner, Cocker Spaniel , Dachshunds y Collies. Se ha descrito también en gatos y el proceso puede venir acompañado de riñones poliquísticos. Cuando aparecen síntomas, en perros son más típicos los signos digestivos, mientras que en gato prevalecen los síntomas respiratorios[2]. Un artículo reciente indica que los resultados son excelentes tras la resolución quirúrgica, con un 97% de supervivencia[3].

c. **Hernia de Hiato:** Es una hernia, usualmente congénita, debida al defecto del ligamento frénico-esofágico que permite el desplazamiento de la unión gastroesofágica hacia el interior de la cavidad torácica. Existen tres tipos de hernia de hiato, la de hiato axial, la paraesofágica y la combinada. En la primera y más frecuente, la parte abdominal del esófago y parte del fundus gástrico se desplazan hacia el tórax a través del hiato esofágico. En la segunda, la parte abdominal del esófago y el esfínter esofágico caudal permanecen en una posición fija, pero una porción del estómago se desplaza dentro del mediastino adyacente a la porción torácica del esófago. La hernia de hiato puede pasar desapercibida y si hay síntomas suelen ser debidos a esofagitis, por reflujo y si la cosa se complica puede darnos penumonía por aspiración. Las razas más predispuestas a sufrir este tipo de hernia son las braquicefálicas y los Sharpei[2]. Se ha asociado este proceso como causante del reflujo gastroesofágico en perros braquicefálicos, aunque en un reciente estudio se habla que puede existir el reflujo por una incompetencia del esfínter sin que haya hernia hiatal[4].

Signos clínicos

Los signos clínicos pueden variar en función de las vísceras herniadas. Los casos crónicos pueden ser asintomáticos, siendo su diagnóstico muchas veces un hallazgo clínico.

Los principales síntomas son respiratorios (tos, disnea e intolerancia al ejercicio). Los síntomas digestivos pueden ser también importantes debido a la herniación parcial o total del tracto gastrointestinal (vómitos, anorexia, diarrea, pérdida de peso, dolor tras la ingestión de comida). Los signos cardíacos (principalmente signos de insuficiencia cardíaca derecha) y neurológicos (encefalopatía hepática si el hígado es el órgano herniado) aparecen con menos frecuencia.

Las hernias de hiato no causan signos respiratorios pero sí síntomas debidos al reflujo gastroesofágico:

esofagitis, megaesófago secundario e hipomotilidad esofágica.

El diagnóstico definitivo se realiza mediante un estudio radiológico y/o ecográfico.

Consideraciones preoperatorias y anestésicas

Según un estudio antiguo[5], las hernias diafragmáticas traumáticas tenían mayor mortalidad cuando la cirugía se realizaba antes de las 24 horas con un 33% de fracasos, mejorando la supervivencia hasta el 90% si se retrasaba la cirugía tres semanas. Si la cirugía se producía tras 1 año de la lesión, había hasta un 67% de fracasos. Actualmente se considera que el tratamiento quirúrgico se pospondrá hasta la estabilización del paciente, pero no debe demorarse innecesariamente si el paciente está estable porque pueden derivarse complicaciones. No se han asociado peores supervivencias valorando el tiempo del trauma a la cirugía, del trauma al ingreso y del ingreso a la cirugía[6].

En este estudio sí que hay mayor mortalidad para mayor duración de la anestesia y del procedimiento quirúrgico. Los animales con lesiones concurrentes de tejidos blandos tenían 4,3 veces más probabilidades de mortalidad que aquellos sin lesiones de tejidos blandos. Los animales con lesiones ortopédicas y de tejidos blandos concurrentes tenían una probabilidad de mortalidad 7,3 veces mayor que aquellos sin lesiones ortopédicas. Los animales que dependían del oxígeno tenían 5,0 veces más probabilidades de mortalidad que los que no lo eran[6].

Si el estómago es una de las vísceras herniadas, la cirugía se realizará lo antes posible ya que una distensión gástrica aguda en el tórax puede provocar una crisis respiratoria que puede resultar fatal.

En cuanto a las consideraciones anestésicas, debemos tener en cuenta que son pacientes que presentan o pueden presentar distrés respiratorio, por lo que consideraremos la opción de sedar al animal antes de anestesiarlo para aliviarle el estrés, y en el momento de la inducción anestésica, ésta será lo más rápida posible mediante agentes inyectables.

TÉCNICA QUIRÚRGICA

El abordaje para la resolución de las hernias diafragmáticas se realizará siempre a través de la cavidad abdominal, aunque algunos autores han aconsejado un abordaje a través del noveno espacio intercostal, conociendo el lado donde se localiza la hernia porque permite una mejor exploración de la maltrecha cavidad pleural[7].

Pasos:

1. Colocar al paciente en decúbito dorsal y preparar la zona abdominal de forma aséptica bastante más craneal que su apófisis xifoides.

   Realizar una laparotomía media craneal de xifoides hasta sobrepasar la cicatriz umbilical y explorar la cavidad abdominal cuidadosamente. Retirar el ligamento falciforme para mejorar la observación del defecto[8].
2. Las vísceras herniadas se movilizarán delicadamente hacia cavidad abdominal. Si es necesario, agrandaremos el defecto diafragmático. (hernias incarceradas.
3. Si existieran adherencias, diseccionar cuidadosamente los tejidos de las estructuras torácicas, reseccionando o desbridando el tejido necrótico si fuese necesario.
4. Explorar detalladamente las vísceras para descartar lesiones graves en ellas.
5. En el caso de hernias peritoneopericárdicas, no cerraremos el saco pericárdico.

Una vez recolocamos las vísceras, procedemos a cerrar el defecto del diafragma.

6. En general no se requiere reavivar los bordes del defecto. Cerrar el defecto de dorsal a ventral mediante un material de sutura preferiblemente no absorbible, o absorbible de reabsorción lenta como la polidioxanona. Podemos realizar un patrón de sutura discontinuo mediante puntos recurrentes horizontales o en cruz, pero otros autores aconsejan un patrón contínuo.
7. Previamente al cierre del defecto recomendamos la colocación, a través del mismo defecto, de un tubo de palometa acoplado a una llave de tres vías y una jeringa, para restaurar la presión negativa intratorácica una vez cerrada la hernia
8. Comprobar la estanqueidad de la sutura llenando la cavidad abdominal de suero salino fisiológico estéril hasta sumergir la totalidad de la sutura y verificar que no existen fugas.
9. Aspirar el suero y cerrar la laparotomía media de forma convencional.

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THE FRACTURE CHALLENGE ~ WHAT TO DO AND WHEN

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ABSTRACT BODY:

THE FRACTURE CHALLENGE ~ WHAT TO DO AND WHEN

As fracture repair has progressed in small animals over the past fifty years the concept of the surgeon working as carpenter vs. gardener has driven many of the changes to the repair strategies we currently employ. Our ability to select and modify our stabilization plan according to the specific individual patient challenges that we face has broadened our choices, improved patient-specific care and increased our success rate. It has also introduced a bigger technical challenge to the individual surgeon. With an increase in options comes a need for surgeons to become educated, familiar, and experienced in a broader range of techniques and implants.

The initial step in deciding what is the best way forward surgically with a fracture patient is the accurate and timely assessment of the patient's overall status. Fracture repair is rarely an emergency, but it is urgent. If the patient is stable and the fracture is closed, and patient pain status is controlled then repair within 4-6 days of the trauma is an acceptable time frame. At initial presentation the patient should be carefully assessed for pulmonary, urinary, gastrointestinal, vascular, and neurological status. This allows the assessment of patient suitability for fracture repair, presence of concurrent injury, determination of fracture management, and ideal timing of surgery. Stable patients have a very different set of issues than unstable patients and differentiation of patient type is crucial to avoiding anesthetic complications and sub-optimal results.

Fracture patients must be initially assessed clinically in terms of the owner and patient compliance and the pet's pain threshold. This is followed by mechanical fracture assessment to determine the intrinsic stability of the fracture site and therefore the stabilization challenge ahead. The biologic fracture assessment includes the patient age, health status, soft tissue condition at the fracture site, area of bone involved and its relative challenge, the degree of concurrent traumatic disruption to the patient, and the required extensiveness of surgical approach.

The choice and timing of fracture repair technique varies greatly between patients. In cases with open fracture and unremittent poorly controlled patient pain, fracture repair is urgent and should be tackled within 6-12 hours. The rest of the fracture patients are often able to wait for surgery if necessary, providing the fractured limb is supported by external coaptation as indicated, the patient is well supervised and cared for until the procedure, and the patient's fracture pain is adequately managed. Patient owners must be well informed during the assessment and decision-making process and the veterinarian performing the procedure must speak openly and honestly to the owners about their pet's medical status, the risks, costs and the owner's required commitment in time and money. The predicted functional outcome of the patient must also be considered by the surgeon and communicated directly to the owner to allow them to understand what their pet's functional prognosis is.

The choice of technique and implant system has evolved greatly over the past 50 years. The addition of external skeletal fixation frames (ESF) to our initial foray into skeletal bone plating allowed us significantly more flexibility on our choices. The degree of soft tissue trauma present, the exposure of bone in open fractures and the ability to disrupt the skeleton's soft tissue surround less during repair all combined to elevate the ESF option to general use.

Mirroring the human bone implant trend, the intramedullary interlocking nail (ILN) was introduced to allow a more stable, less invasive approach to the diaphyseal fracture. Originally, in companion animal fracture repair, an intramedullary pin was combined with cerclage wire in an attempt to create stability. It was often unsuccessful. Pin migration, cerclage wire slippage and overall loss of construct stability opened the door to non-union, delayed union and malunion. The ILN sought to utilize the biomechanical advantage of the intramedullary implant in combating bending forces whilst providing resistance to compression, distraction, and rotation at the fracture site with additional interdigitating screws or bolts. Although there were specific challenges that needed to be overcome with patient size, bone canal diameter and the screw or bolt nail interface, the continued evolution of this implant has been a successful one.

The evolution of original bone plating into locking

plate technology was an important one for bone-plate construct stability. The ability to build a more reliable and force-resistant construct that required less overall implantation of screws and resisted biomechanical forces more reliably further progressed our options and improved our fracture repair success rates. As we have become more familiar with the locking plate systems and their performance, we have continued to evolve the implantation techniques. Open reduction internal fixation (ORIF), which had been our primary bone repair technique, evolved with the more recent focus on minimally invasive fracture repair techniques. Minimally invasive plate osteosynthesis (MIPO) technique and the sub-technique of minimally invasive percutaneous plate osteosynthesis (MIPPO) has allowed veterinary orthopedists to seek more rapid healing with less morbidity. The increased availability of intra-operative imaging and the development of fluoroscopic protocols has further improved the accuracy of surgical repairs but has increased the complexity of the surgical theatre and the investment that is required to follow the MIPO trend.

Along with the evolution of fracture implant systems has come a continued progression in fracture site management. Original ORIF surgical techniques involve wide exposure of the fractured bone, removal of fracture blood clots, painstaking reconstruction of the bone column, and placement of a stabilizing bone plate. The development of the 'open but do not touch' (OBDNT) surgical philosophy was followed closely by biologic osteosynthesis under the minimally invasive orthopedics (MIO) banner. The key principles of biologic osteosynthesis is the indirect reduction of fracture ends via traction, positioning and closed manipulation of the fractured limb, the use of flexible fixation, the avoidance of biological damage by the surgical team and a decreased reliance on bone grafting to overcome fracture site disruption. The overall aim is to produce the best biological conditions for healing rather than absolute stability of the bone-implant construct.

In summary, the fracture patient must be assessed carefully, categorized into an appropriate category that includes patient, trauma and fracture type and surgically treated in an appropriate fashion that maximizes success, limits complications, and allows the patient to recover the best functional status possible. Surgeon's must do no harm due to poor choices or lack of care, must consider all options carefully and completely, should consider the need for outside guidance and decide if they are indeed the person who should perform the procedure for this patient at this time.

RECOMENDACIONES SOBRE VACUNACIÓN DE GATOS PARA LATAM

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ABSTRACT BODY:

RECOMENDACIONES SOBRE VACUNACIÓN DE GATOS PARA LATAM

La comprensión del concepto de vacunas esenciales y no esenciales es fundamental para determinar el protocolo de vacunación de un gato. Las **vacunas esenciales** son aquellas que todo gato, independientemente del lugar que viva o de su estilo de vida, debe recibir. Estas vacunas protegen contra los agentes infecciosos que pueden causar enfermedades graves o la muerte, especialmente en los gatitos. Las vacunas esenciales contienen el parvovírus felino (FPV), el calicivírus felino (FCV) y el herpesvírus felino (FHV). En los países en que la rabia es endémica, la vacuna antirrábica también se considera esencial. Las **vacunas no esenciales** son las que se consideran para gatos cuya ubicación geográfica o estilo de vida los pone en riesgo de infecciones específicas. Ellas no son necesarias para todos los gatos y no deben utilizarse cuando no hay pruebas de la existencia de una enfermedad en la región, o cuando no existe riesgo de exposición. Las vacunas no esenciales para LATAM incluyen las que contienen el virus de la leucemia felina (FeLV) y la *Chlamydia felis*. Algunas vacunas son consideradas **no recomendadas** para gatos, porque no existe suficiente evidencia científica que justifique su uso. Estas incluyen la vacuna contra la peritonitis infecciosa felina (PIF), pero que no se encuentra disponible en América Latina.

La vacunación esencial de gatitos debe comenzar entre las 6 y 8 semanas de edad, vacunándose cada 3 a 4 semanas hasta las 16 semanas de edad o más, con vacunas que contengan, de preferencia, virus vivos modificado (VVM). El objetivo de administrarse las dosis hasta las 16 semanas o más es para que se tenga mayor seguridad de que se ultrapasó la interferencia de los anticuerpos maternos. Esto se debe a que, en los últimos años, se han acumulado evidencias científicas que indican que una minoría considerable de gatitos tiene cantidades importantes de anticuerpos maternos contra algunos de los componentes de la vacuna, incluso hasta las 20 semanas de edad. Por ello, se debe administrar una última vacuna esencial de VVM a los 6 meses de edad, para asegurar que todos los gatitos reciban, al menos, una dosis de vacuna capaz de conferirles inmunidad, en caso de que aún existan anticuerpos maternos a las 16 semanas o más. Por lo tanto, el número de vacunaciones primarias con vacunas esenciales estará determinado por la edad en que se inicia la vacunación y el intervalo de revacunación elegido. Se ha demostrado que las vacunas de VVM contra el FPV proporcionan una inmunidad duradera y robusta a una gran mayoría de los gatos vacunados, cuando se utilizan de acuerdo con el protocolo arriba mencionado. Por consiguiente, se recomienda la revacunación contra el FPV cada 3 años. Es poco probable que la vacunación más frecuente que cada 3 años, con vacunas de VVM que contienen el FPV, proporcione alguna mejoría en el grado de protección; y, todavía, puede aumentar el riesgo de reacciones adversas. Por el contrario, las vacunas contra el FCV y el FHV no proporcionan una protección tan sólida o duradera como las vacunas contra el FPV. La inmunidad que confieren estas vacunas no puede prevenir la infección, ni el desarrollo del estado de portador. Sin embargo, para los gatos que llevan un estilo de vida de “bajo riesgo” (es decir, gatos sin acceso al exterior de la casa y que no visitan criaderos) se considera que la vacunación cada 3 años proporciona una protección suficiente. Para los gatos con mayor riesgo de infección por FCV o FHV (por ejemplo, gatos con acceso al exterior, o gatos que visitan regularmente un criadero), se recomienda la revacunación anual. En algunos países, es posible adquirir vacunas que contienen solo el FCV y el FHV, de modo que se pueden utilizar vacunas trivalentes cada 3 años (FPV, FCV, FHV) y una vacuna bivalente (FCV, FHV), si es necesario, en los demás años. Desafortunadamente, tales vacunas no están, hasta ahora, disponibles en todo el mundo.

Se puede utilizar la serología para monitorear la inmunidad protectora para el FPV y, por consiguiente, ayudar a la toma de decisiones sobre los intervalos de revacunación. No obstante, la serología no puede predecir la protección contra el FCV o el FHV. La correlación entre anticuerpos circulantes y la protección contra la infección por FCV y FHV es menos robusta que la presencia de inmunidad local de mucosa y de inmunidad mediada por células. Por esa razón, un resultado negativo en la serología para FCV y FHC no indica, necesariamente, la ausencia de protección.

Las vacunas contra la rabia canina deben ser administradas a partir de las 12 semanas de edad y la revacunación debe realizarse de acuerdo con las

recomendaciones de los prospectos y las leyes del país.

El uso de las vacunas para la leucemia felina debe ser determinado por el estilo de vida y el riesgo de la exposición de los gatos, así como por la prevalencia de la infección en el local. Muchos expertos felinos creen que a pesar de que la prevalencia de la infección por el FeLV se ha reducido notablemente en muchas partes del mundo, debido a programas de control exitosos, en las áreas geográficas en las que la infección por el FeLV sigue siendo frecuente, cualquier gato de menos de 1 año con riesgo de infección debe recibir la vacunación. Se debe aplicar dos dosis de la vacuna, dadas con 3 a 4 semanas de intervalo, no empezando antes de las 8 semanas de edad, con revacunación 1 año después de la última dosis. Los gatos adultos que nunca han sido vacunados necesitan también recibir 2 dosis de la vacuna contra el FeLV, con intervalo de 3 a 4 semanas. La revacunación de los gatos con riesgo de exposición debe ser anual, o cada 2 años, dependiendo de la vacuna utilizada. Solo se debe vacunar a los gatos negativos para el FeLV.

La vacunación para *C. felis* se usa, principalmente, como parte de un régimen de control para ambientes con muchos gatos, donde se ha confirmado infecciones con manifestación clínica de la enfermedad. El protocolo de vacunación consiste en administrar la dosis inicial a partir de las 9 semanas de edad, y una segunda dosis 3 a 4 semanas después. Los gatos adultos nunca vacunados contra *C. felis* también necesitan de 2 dosis separadas por 3 a 4 semanas. Se indica el refuerzo anual en los gatos con riesgo de exposición.

Los profesionales de América Latina y sus asociaciones nacionales deberían ejercer presión sobre la industria para acceder a las vacunas felinas que contengan solo los componentes no esenciales (FeLV o *C. felis*). En América Latina hay una escasez o falta de vacunas monovalentes no esenciales; por ejemplo, en algunos países las vacunas de *C. felis* solo están disponibles en combinación con los componentes esenciales (FPV, FCV y FHV), y la vacuna contra el FeLV sólo está disponible en combinación con los cuatro antígenos anteriores. Por lo tanto, existen vacunas para felinos de 3, 4 y 5 componentes, pero pocas o ningunas monovalentes no esenciales.

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PREVENTIVE AND THERAPEUTIC STRATEGY FOR LIMITING LEISHMANIA INFECTIONS IN DOGS AND CATS

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ABSTRACT BODY:

Infected dogs are considered the main urban reservoirs for *Leishmania infantum* parasite. Infectiousness to competent phlebotomine vectors has been associated with many factors, the main being the severity of the disease exhibited by infected dogs (1). Serological and specific PCR test that detect *Leishmania infantum* infection in dogs appear weakly useful to provide information on the capacity of infected dogs to be infectious, in particular to evaluate the dog's infectiousness during and after the use of preventative and/or therapeutic compounds against *Leishmania* parasite. The evaluation of dog's infectiousness remains still difficult due to the need of xenodiagnosis as the only valid method to assess it. Preventative and therapeutic strategies appear the only methods useful to limit the spread of parasite from infected dogs to other animal reservoirs and to susceptible human population. The treatment of infected dogs is allowed all around the Europe and many Countries of the world, however it appears not able to achieve a 100% sterile cure in dogs and it can cause drug resistance. For this reason WHO strongly suggests to avoid the use of drugs used for the treatment of humans for veterinary purposes. The first-line treatment for CanL is represented by the use of second-line leishmanicidal drugs for humans (N-methylglucamine antimoniate [MA] and Miltefosine [MIL]) in combination with Allopurinol [AL]. Despite its proven efficacy, MA has some drawbacks, including the costs, the parenteral administration and the numerous reported side effects. MIL used at the registered dosage of 2 mg/Kg/die, is a good alternative, it is administered orally and this is widely preferred by owners. This molecule has a low impact on renal function and can be used in dogs with severe proteinuria, one of the most frequent clinicopathological alteration reported in CanL. However, adverse reactions associated with MIL treatment have been reported too, mainly vomiting, diarrhea, abdominal pain and loss of appetite that tend to regress spontaneously within a few days after starting treatment probably because dogs become habituated to the medication. Allopurinol is administered orally too, in combination of one of the two mentioned drugs. The time course of treatment, both for MA and MIL, is 4 weeks. After the first month, the length of allopurinol treatment depends on the severity of the disease, the clinical and parasitological response to treatment and the individual tolerance to this drug. Due to its mechanism of action, Allopurinol could cause massive xanthine crystalluria. In this case, the drug should be discontinued after the evidence of no reduction of crystalluria with low purine diets or by reducing the drug's dosage, to avoid the risk of urolithiasis.

Few Leishmania vaccines have been marketed during the last 20 years, the main limitation of which is that they do not prevent the establishment of infection. Leishmania vaccines may reduce the infectiousness to sand flies [2], however vaccinated dogs should be considered always as a source of infection for dogs and for immune response in dogs in order to either i) prevent the establishment of an initial infection, or ii) control its progression towards severe disease and iii) promote the abrogation of Leishmania transmissibility by vaccinated dogs if they become infected (Gradoni, 2015). Protection from CanL strongly depends on the ability of the dog immune system to respond since control requires balanced levels of tissue-specific immunity to not cause immune-mediated tissue damage and subsequent disease. The current licensed vaccine (one in Brazil - Leish-Tec® (Ceva Saúde Animal, Brazil), the second in Europe - LetiFend® (LETIPharma, Spain) have only the ability to limit the progression of the infection to disease or to decrease the severity of the disease. This is due to the complexity of the immune response of the naturally infected dogs against the parasite, which can result in immunological resistance or susceptibility profile and different clinical forms of the disease. Mathematical models have suggested that canine vaccination have limited to no effect on the infection incidence in humans, compared to insecticide impregnated collars.

Topical insecticides with proven efficacy against sand fly bite are considered the best tool to limit canine leishmaniosis infection. These compounds combine two positive effects against transmitting insects: a) the killing effect, the capacity to determine the death of sand flies after the contact with protected dogs, b) the anti-feeding effect, the capacity to avoid the bite.

Several topical formulations, such as collars, spot-ons or sprays, containing synthetic pyrethroids with proven anti-feeding (excito-repellent) and insecticidal effects against phlebotomine sand flies aere currently employed all around the world (3). After being applied to the dog's skin, their active ingredients spread over the entire surface of the body and the hair coat. Despite their large scale application, insecticide products are characterized by some limits mainly due to the not constant good compliance of the dog's owners, mainly for their cost and the regularity of application. Although discouraged by manufacturers a combination of devices is commonly used by dog's owners, being the association of collar plus spot-on the most frequently employed. Preventive measures against sand flies are typically adopted to protect healthy dogs from *L. infantum* infections, less so to avoid parasite spreading by CanL sick dogs, for which anti-leishmanial therapies may lead to decreased infectiousness to vectors, but have only temporary efficacy. Preventive and therapeutic measures against CanL should be considered an integrated program for the control of this zoonosis, both in dogs living or travelling into endemic areas. Future researches should investigate new anti-Leishmania drugs for specific use in the animals, effective vaccines for limiting the possibility of infection, low cost new insecticides and a less complicated method to assess the infectiousness of infected individuals.

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WHERE IS THIS COUGH COMING FROM?

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ABSTRACT BODY:

Why is this patient Coughing?

Cough is one of the body's defense mechanisms to prevent the entry of noxious materials into the respiratory system, and to clear potentially harmful debris from the lungs and respiratory tract. It is normally associated with other protective processes such as bronchoconstriction and mucus secretion, both of which may aid in the effectiveness of the cough. Cough is an indicator of airway or pulmonary disease, although it also may occur as a primary sign of other thoracic disorders. Cough receptors are in the larynx, pharynx and large airways. They are absent in the alveoli, alveolar sacs, and pulmonary parenchyma. As a result, diseases of the lung parenchyma may not stimulate the cough reflex to the degree that disorders within the airways do. This is teleologically understandable because even a vigorous cough would not move gas fast enough to cause turbulence and shearing forces at the airway wall in the smaller airways. Cough at this level, therefore, would be ineffective. Cough is not a localizing sign being associated mostly with diseases of the lower respiratory tract (trachea, bronchi, and lungs). Patients with upper respiratory tract disease may, however, cough if secretions are being drained into the larynx and trachea.

What is causing this cough?

Cough in dogs is usually caused by tracheal collapse, chronic bronchitis, compression of the left main stem bronchus by a large left atrium, congestive heart failure or pneumonia. Other causes of cough include: tracheitis, traumatic or mechanical problems (e.g. foreign body, irritating gases), parasitic disorders (e.g. lungworms, heartworm), extraluminal compression of airways, and neoplasia. In cats, cough is usually a sign of pneumonia, heartworm disease, asthma, or lungworms.

The *history* may help establish the etiology for the cough. In dogs, nocturnal coughing is generally associated with cardiac disease, psychogenic causes, or tracheal collapse. Patients with tracheal collapse also may start coughing after drinking water or during periods of activity in the house. Cough due to respiratory disease is more likely to occur during the day. Tracheal diseases and diseases affecting the large bronchi usually have a goose-honk cough that can be initiated by excitement or by pulling on the collar and leash.

The *physical examination* assists in identifying the reason for the cough and is particularly helpful in differentiating cough respiratory or heart disease. Patients with heart failure usually have fast heart rates with soft crackles, whereas patients with primary lung disease tend to have pronounced sinus arrhythmia with crackles that are usually louder and of higher pitch, than the ones resulting from heart failure. Wheezes also can be present, particularly in patients with disease of the trachea and large bronchi. Crackles are a non-specific sign of small airway disease. Maneuvers that stimulate the patient to take deep breaths may help in lung auscultation because abnormal lung sounds (especially crackles) may be missed if the lung is not adequately expanded. Closing the nostrils for 10 to 15 seconds usually will force the patient to inspire deeply.

Good quality *chest radiographs* are of paramount importance in evaluating a patient with cough. They provide an initial overview of thorax and great for planning and monitoring treatment. On many occasions the diagnosis can be made by visualization of a tracheal collapse, large left atrium, pulmonary masses, or radiographic patterns suggestive of pulmonary edema, pneumonia, bronchial disease, or heartworm disease. There is a limited number of ways that the heart and lungs can respond to injury and produce radiographic patterns. Even when radiographs do not reveal the diagnosis, they help in narrowing the differentials, and determining the next diagnostic step. *Computer tomography (CT)* is the most accurate non-invasive technique to evaluate large and small airways, and pulmonary parenchyma. It has higher sensitivity than radiographs providing more detail and better anatomic delineation of abnormalities seen in radiographs. It can also be helpful in cases where no changes were observed in radiographs. Unlike radiography, CT can differentiate soft tissue from fluid and allow for better visibility of internal soft tissue architecture.

B-type natriuretic peptide (*BNP*) is a functional cardiac biomarker that increases in the blood when cardiac stretch and pressure increase. Increases in BNP are used diagnostically as an indicator of serious heart disease or congestive heart failure. NT-proBNP is

higher in dogs and cats with respiratory disease from heart failure than in dogs and cats with respiratory distress from other causes. Occasionally, BNB can be increased in dogs and cats with primary respiratory disease and pulmonary hypertension due to the structural changes in the right ventricle. Heart failure is extremely unlike in dogs and cats with BNP within the reference range.

Bronchoscopy provides direct inspection of trachea and bronchial tree. General anesthesia is required, but the risks involved with the technique are outgained by the valuable information that can be obtained. Abnormalities in the lumen (e.g. tracheal or bronchial collapse, external compression), mucosa (e.g. edema, inflammation, excessive mucus), or submucosa (e.g. hyperemia, nodules) can be directly observed with the bronchoscope. Additional advantages of bronchoscopy include the ability to perform bronchoalveolar lavage and removal of foreign bodies during the procedure.

Although imaging techniques and direct visualization are useful in identifying and localizing the disease process responsible for the coughing, they do not always provide an etiologic diagnosis and biopsies are not yet routinely performed. For these reasons, *cytologic evaluation* is often necessary to establish an etiologic diagnosis or to characterize the type of inflammation present. Samples are usually obtained via endotracheal wash, bronchoalveolar lavage, or fine needle aspiration of the lung. Samples obtained by tracheal wash and bronchoalveolar lavage can also be used for bacterial and fungal culture in case of a suspected primary or secondary infection. Fine needle aspiration of the lungs is usually indicated for the diagnosis of masses. Based on the cytologic examination, the nature of inflammatory process (e.g., eosinophilic, neutrophilic) can be determined. On some occasions, a specific diagnosis can be obtained (e.g. when etiologic agents can be recovered or specific neoplastic cells identified).

Is a dog with heart murmur coughing due to heart or lung disease?

Coughing dogs with heart murmur constitute a diagnostic and therapeutic challenge. Old dogs, particularly of small breeds, are commonly affected by chronic respiratory diseases and mitral regurgitation, both of which can result in coughing. Dogs with heart disease may cough due to left-sided congestive heart failure or due to compression of the left main stem bronchus. Patients that are coughing because of compression of the left main stem bronchus and are not in heart failure have history and clinical signs like patients with chronic respiratory disease.

The history and physical examination are very helpful in the determining the reason for the cough in many cases. Dry, hacking coughs are usually associated with large airway disease (e.g. tracheobronchial collapse, tracheobronchitis, or compression of the left main stem bronchus). More subtle coughs may result from parenchymal injury (e.g. pulmonary edema, pneumonia). Cough in heart failure patients tends to be worse at night and the cough may be accompanied by pink nasal discharge or sputum. Cough in patients with respiratory disease is usually worse during the day and may be accompanied by mucopurulent nasal discharge. Dogs with heart failure are frequently thin with severe weight loss (a notable exception are some Dobermans with dilated cardiomyopathy). Patients with primary respiratory disease, on the other hand, tend to be overweight.

On physical examination, dogs with left-sided congestive heart failure usually have a fast heart rate, whereas dogs with respiratory disease or left main stem bronchus compression have normal heart rate with pronounced sinus arrhythmia. Presence of a gallop rhythm suggests diastolic dysfunction and heart failure. Crackles may be presented in patients with heart or lung disease, although patients with primary lung disease tend to have louder, high-pitched crackles. Cyanosis can be observed in both primary lung disease and congestive heart failure. Cyanosis in a patient that is not ill strongly suggests that the cyanosis is caused by a respiratory problem. Dogs cyanotic from heart disease usually dye or recover with therapy in a short period of time. Long term cyanosis will be accompanied by polycythemia. Dogs with chronic respiratory disease, therefore, have increased hematocrit, whereas dogs with congestive heart failure have low-normal to low hematocrit due to fluid retention. Further diagnostic tests such as chest radiographs, echocardiogram, and tracheal wash may be necessary to establish if the cough is due to pulmonary or heart problem. Careful interpretation of the information obtained in the history and physical examination, however, will allow the veterinary practitioner to arrive at the correct diagnosis with reasonable assurance in many cases.

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AVANCES Y TÉCNICAS DE APRENDIZAJE EN LA CARRERA DE MEDICINA VETERINARIA

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ABSTRACT BODY:

Advances and learning techniques in veterinary medicine

The students should construct and integrate knowledge, so I would like to show three fields that we have been working in the last years: Biological foundations, Structure and Function and Simulation. The first two aim at the understanding of the necessary science that supports practice and the third is directed at the development of clinical skills that are the blocks of practice. The general goal is to have graduates that are skilled, although not experts, practitioners.

To use the advances and learning techniques in veterinary medicine, you cannot ignore the basic tools for their use, that is why today we will make some summaries of these tools that are used daily bases.

I. Simulation. - Situations that are relevant, and that the student must learn are simulated, scenarios can be used. In the simulation, the important thing is to make clear “How to do something”, you come to do things, not to see. It is to represent real life situations in a controlled environment. Simulation models: Skills (isolated and integrated - memory develops, can be an active procedure, teacher feedback is immediate) Scenarios (simple and interprofessional - it is to integrate knowledge, attitude, and skill - feedback and debriefing occurs at the end of the class).

The difference between feedback (the teacher tells how things are, it is one to one) and debriefing (it is in a group, students talk with students, it is a group construction by students for students). The simulation becomes more complex over time. Students should start with simple skills (replicate situations they see), integrated ones (they are more complex and require making decisions) Simple scenarios, knowledge must be put in context, instead interprofessional scenarios, soft skills must be developed (stress management, communication, respect, teamwork, etc.)

The scenario processes are:

1. Briefing 25%
2. Scenario 25%
3. Debriefing 50%

At this point the facilitator should respond with questions so that the students arrive at the answers. Do not be punitive, always positive, closed answers should be always avoided.

II. Biological Foundations. - The methodology is common to any classroom Methodological proposals: You can work in stations, or everyone does the activities, in a short time and measured.

Team based learning (groups of 5 to 7):

1. Previous work
2. Individual work, then in group
3. General and facilitated discussion
4. Application of concepts
5. Closing discussion

METHODOLOGIES: Normally used in the classroom Jigsaw group: Experts on topics are trained and then they join in heterogeneous groups and feed on what they have learned, at the end they return to the original group to give the feedback. (Pieces are the people) Odd one out: cards are given, and you must find what is abnormal or rare Carousel: you assign yourself a task or proposal and go to other groups and present the ideas of the task or proposal and then return to your original group. Know Want to know Learned: Learning process where questions are asked about what they want to know and what things at the same time, there is interaction with the teacher, the students came to class prepared with the appropriate information. Puzzle: Topics that mesh to achieve an end (cause / effect) - build concepts (pieces are the concepts) Gamification, games on concepts. The type of teaching depends on the ACHIEVEMENT you want for the session.

III. Structure and Function. - You must have clear documentation; this also serves for accreditation and continuous improvement.

Rules:

1. Student Centered
2. Clear goals
3. Details must be concrete

Model:

1. Enable learning
2. Soft skills
3. Relevant learning
4. Curricular integration (horizontal and vertical)

The class plan is a document managed by the teacher, it should be detailed in it about what to do and important points, available resources. The more

detailed the better, it should be clear what the student requires (relevance).

Beginning.

1. Integration between the disciplines of Structure and Function

2. Integration between the disciplines of Structure and Function and clinical practices

3. Active collaborative learning

4. Use of technology and living anatomy

5. Relevant learning Structure and Function Moments: 1. Opening 2. Autonomous work (40% to 60% of the session – under teacher supervision) 3. Logout

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HOW I TREAT CANINE LYMPHANGIECTASIA: HOW LOW IS LOW FAT

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ABSTRACT BODY:

Intestinal lymphangiectasia is characterized by marked dilatation and dysfunction of the intestinal lymphatic vessels, which then leak protein-rich lymph into the intestinal lumen, resulting in protein-losing enteropathy (PLE) (1). It can be primary/idiopathic (Yorkshire Terriers being a predisposed breed) or secondary to lymphatic obstruction (due to intestinal inflammation, neoplasia, fibrosis, thoracic duct obstruction, or right sided heart failure). Typical clinical signs are diarrhoea, weight loss, and polyphagia. Less common ones are lethargy, and anorexia. Vomiting can also be present. Ascites and oedema can appear if the hypoalbuminemia is severe enough. Classical laboratory abnormalities (2) include lymphopenia, panhypoproteinemia, hypocholesterolemia, and hypocalcaemia (partially attributed to the low albumin, but can also be due to calcium and vitamin D malabsorption). Also, hypomagnesemia has been suggested to result in secondary hypoparathyroidism and consequently contribute to hypocalcaemia (3). Hypomagnesemia is common in malabsorption syndromes, especially in Yorkshire Terriers (4).

The leakage of lymph promotes oedema and inflammation, which can result in malabsorption and subsequent malnutrition. Fat absorption can be severely affected, and fat-soluble vitamins can become deficient over time. These animals are also prone to coagulation disorders, due to possible vitamin K malabsorption and due to the loss of coagulation factors. Antithrombin III can be lost through the lymph, which promotes a thromboembolic state. Measuring fat-soluble status in a clinical setting is difficult, but parenteral supplementation would be indicated if clinical signs which could be attributed to vitamin deficiencies do not resolve with treatment.

Management of lymphangiectasia includes addressing the underlying disease if present, implementing dietary modifications, and treating symptomatically as needed.

Dietary fat restriction is the cornerstone of the treatment of this disease. The level of fat restriction will depend on the severity of the disease and on the fat level of the current diet of the patient. In general, restriction well below that found in a commercially available canine maintenance diet is necessary.
Fatty acids, especially long-chain ones, considerably increase the flow in lymphatic vessels(1). Therefore, dietary fat restriction results in decreased lymphatic flow and subsequent decrease in lymph leakage. There are some papers supporting the use of very low fat, highly digestible homemade diets in the management of PLE (5).

The use of medium chain triglycerides (MCT) has been proposed to provide energy since it was hypothesized that they are absorbed through the portal vein. However, there is a lack of clinical data and they do not provide any essential fatty acid, whose requirements need to be met.

FEEDING PLAN

Diet choice: A complete diet history is needed to identify the fat intake associated with clinical signs. In the author's experience, halving the fat intake is a good start, and, in patients fed relatively high fat diets, there are commercial intestinal low-fat diets that might work well. Commercial intestinal low fat diets have fat levels of 16-22% fat calories. As a comparison, maintenance diets can range from 20 to 35% calories (can be even higher, especially in wet diets or some diets marketed as low carb). Some veterinary diets, like convalescence ones, can be as high as 50-60% fat calories. Raw diets can be even higher, some reaching 70% fat calories or more.

However, if the patient is eating an average fat diet, these diets are less likely to be successful, especially in primary cases. If the lymphangiectasia is secondary to other enteropathies (such as IBD), it is reasonable to try a limited ingredient elimination diet (based on novel ingredients or hydrolysed protein), choosing the one lowest in fat. Alternatively, obtaining a homemade diet with both novel ingredients and low in fat from a veterinary nutrition specialist is indicated. Highly digestible diets are indicated in all cases. Omega 3 supplementation (EPA and DHA) can be considered, but there is very limited clinical data in chronic enteropathies, and its provision increases fat intake.

Feeding amounts: The energy requirement of each patient must be met, these can be calculated via formulas (6) or using label information, but those are associated with error. Obtaining the current calorie intake from the diet history is preferrable. The calorie goal (kcal/day) is then divided by the energy density of the diet of choice. In all cases, the feeding amounts should be adjusted up or down in 10% intervals to

achieve the weight and body condition goals.

Feeding method: multiple meals per day (at least 2-3) are indicated to reduce the intestinal workload. Free feeding can be considered in patients with low body condition and picky appetite.

Follow up includes clinical signs, nutritional assessment (7), bloodwork, and others (imaging, for example) as needed. The feeding plan will be adjusted accordingly, including diet and daily allowance changes.

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HOW I TREAT FELINE RENAL DISEASE WITH DIET

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ABSTRACT BODY:

INTRODUCTION

Chronic kidney disease (CKD) is a common disease in cats. It is reported to occur in up to 5% of the larger feline population[1] and up to 30-40% of cats over 10 years of age.[1-3] This disease is progressive and irreversible and therapy is directed toward controlling clinical signs and slowing progression.[4] Commercial therapeutic diets designed for the management of CKD are available worldwide and are the cornerstone of medical therapy. Research in cats with naturally-occurring CKD has demonstrated that consuming a therapeutic diet formulated for renal disease can slow progression, reduce the risk for developing a uremic crisis, and increase survival time compared to cats consuming a maintenance diet[5-8] but concerns from owners about certain aspects of therapeutic diets (such as ingredient or protein levels) may lead to delayed dietary treatment with negative impacts on overall health and longevity.

DIETARY MANAGEMENT OF RENAL DISEASE

General dietary management of renal disease involves restricting the protein, phosphorus, and sodium contents of the diet while enhancing the levels of water-soluble vitamins (i.e., B-vitamins) relative to adult maintenance foods. Scientific evidence also supports the protective effects of higher concentrations of EPA in the diets of cats with compromised renal function.[9] The degree of protein and phosphorus reduction will vary by stage of disease. Cats with International Renal Interest Society (IRIS) stage 1 or 2 may need just need a reduction compared to their previous diets while more advanced stages 3 and 4 will require feeding a diet that is at or even slightly below minimum requirements recommended by National Research Council (NRC) recommended intakes.

For cats with decreased tubular function resulting in more dilute urine concentrations without proteinuria and only mild elevations in renal parameters on serum biochemistry tests, reduction in dietary phosphorus relative to current intake is warranted. Over-the-counter (OTC) diets marketed as "Senior" have variable levels of dietary phosphorus, but a few OTC brands are actually developed for dogs and cats with early uncomplicated renal compromise. Nutrient levels should be verified through the manufacturer directly before advising a change to a particular OTC diet for a cat with IRIS stage 1 renal disease. For cats with IRIS stage 2-4 disease, there are a number of commercially-available renal therapeutic wet and dry diets that meet the profile recommended dietary profile for these individuals. OTC cat foods are too high in dietary phosphorus and should not be fed to cats with documented IRIS stage 2 or above renal disease. A number of more recent studies have demonstrated progressive and more rapid renal damage when fed OTC higher phosphorus foods.[10,11] Additionally, home-prepared diets should be used with caution and only under the guidance of trained Veterinary Nutritionists (veterinarians with specific expertise in clinical nutrition for dogs and cats) as many recipes developed by well-intended lay-people, general practice veterinarians, and even non-veterinarian nutritionists may not be appropriate for long-term feeding.[12]

For animals with glomerular disease, loss of plasma proteins (such as albumin) in the urine can result in the progression of renal damage, and the presence of proteinuria is considered a negative prognostic indicator in feline CKD.[13] In dogs with proteinuria reducing dietary protein intake to decrease glomerular capillary pressure and subsequently lessen proteinuria is a mainstay of therapy.[14] The relationship between a reduction in protein intake and a reduction in proteinuria is presumed to occur in cats as well though specific dietary studies have not been published to date.

SUMMARY

Dietary and medical management of CKD in cats has the same therapeutic goals: minimizing clinical signs related to uremia, avoiding dystrophic renal mineralization, reducing proteinuria, maintaining lean muscle mass, and improving quality of life parameters. In the face of reduced renal function at any stage, a reduction in dietary protein and phosphorus should be made but the degree of change can vary with the individual patient's need.

There is no one perfect diet strategy and recommendations should be tailored to the individual's medical history, food preferences, and concurrent disease states to ensure patient acceptance and owner compliance. An individual cat's needs may also change throughout the course of disease and monitoring

body weight, body condition and musculature, and biochemical and urinary parameters should be done regularly.

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SURGICAL EXTRACTION TECHNIQUES

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ABSTRACT BODY:

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Introduction: Surgical extractions involve gingival flaps and the removal of bone to expose tooth roots. This facilitate the operation by extracting a small amount of buccal cortical bone. This technique is very useful for the extraction of canines, maxillary fourth premolars and mandibular first molar, as well as teeth with root malformations and/or retained roots. It is very important to obtain the consent of the owner before performing the dental extraction.

Before performing the dental extraction, dental X-rays should be made to determine if there is ankylosis, or any other important information that guides us about the next procedure. Radiographs provide solid evidence in the medical record too.

First step: A gingival flap is created This flap can be a horizontal flap across the arch (a wrap-around flap) or a flap with vertical releasing incisions (a complete flap). Gingival attachment is released with a periosteal elevator along the arch. This includes the tooth(s) to be extracted and its margin may extend to adjacent teeth. The gingiva is released to or below the level of the mucogingival junction (MGJ) and the flap is connected by incising the gum in the interdental spaces. The advantage of this flap is that the blood supply is not interrupted and there is less suturing.

The most commonly used flap includes one or more vertical release flaps. incisions. This method makes it possible to create a much larger flap than (if handled properly) will increase the defects that can be covered. the upright incisions are created at the line angle of the target tooth, or a mesial tooth and distal to the target tooth. The incisions should be made slightly apically divergent (wider at the base than at the gingival margin). Furthermore, it is important that the incisions be created full thickness, in one motion. A full-thickness incision is created by incising all the way to the bone, and the periosteum is thus held with the flap. Once created, the entire flap is gently reflected with a periosteal elevator. Care must be taken not to tear the flap, especially at the muco-gingival junction.

Second Step:

A portion of the alveolar table between 1/3 to 1/2 in length is removed, corresponding to the length of the root. If the tooth is multirooted, all its roots should be sectioned to form single-rooted teeth. To carry out this maneuver as a whole, a turbine or chisel and a surgical hammer can be used.

Using a high-speed drill and bur (#2–#4 round bur), the bone is cut following the previously identified root edges (mesial and distal edges) from the crestal bone apically toward the flap of soft tissue. These cuts along the edge of the roots can be made quite deep. If made deep, these cuts destroy the root-to-bone attachment along the cuts, reducing the overall attachment of the tooth. With these two cuts made, they are joined apically through the root. This now forms a continuous cut from the crestal bone mesially to the crestal bone distally, a “U” shaped cut. This “U” shaped cut accomplishes the second purpose of hard tissue surgery: the buccal/labial bony plate over the root is now separated from the skull/mandible, and no longer participates in root support. Alternatively, the bone plate can be completely and carefully burred away. When making this and other cuts, be careful not to destroy too much root tissue; this will make the root fragment brittle and difficult to remove in one piece. These hard tissue cuts are made around all root segments that require removal.

Third Step:

We proceed to close the created defect but before a procedure called periosteal fenestration is performed. The periosteum is a very thin fibrous tissue that joins the buccal mucosa to the underlying bone. Since the periosteum is fibrotic, it is inflexible and interfere with the ability to close the defect without tension. the buccal mucosa however, it is very flexible and will stretch to cover large flaws. Due, the periosteal incision takes advantage of this attribute. the fenestration must be done at the base of the flap, and it must be very superficial like the periosteum is very thin. This step requires careful attention, so as not to cut through or cut the entire flap. After fenestration, the flap should stay in desired position without sutures. If this is not the case, then tension is still present and further release is necessary prior to closure. Once the release is accomplished, the flap is replaced and sutured in place to close the extraction socket thus allowing primary healing. Generally, a 4-0 sized material is used with a swaged on reverse cutting needle. A cutting needle is vital, as it will pass through the gingiva more easily, reducing the risk of pulling through the flap. Gentle tissue handling is very important here.

The points that could generate failure in the maneuver would be the following:

- lack of visualization of the tooth (x-ray and surgical exposure)
- impatience
- Too much horizontal force
- Too much vertical force
- Too much rotational force
- Too much grip force
- Insufficient bone removal
- Lack of isolation of each root

The consequences of a non-well procedure are a longer operative time to complete the extraction and more trauma for the animal. If the roots of the teeth are left in the animal, most will abscess and be lost due to inflammation. If complications occur, the oral surgeon risks legal consequences with financial implications. All of these problems can be avoided if the oral surgeon follows each step and does not take shortcuts.

KERATOCONJUNCTIVITIS SICCA

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KERATOCONJUNCTIVITIS SICCA (KCS)

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Introduction

A very important ocular layer that tends to be forgotten is the tear film. Its composition is complex, but it can be divided into layers that mix amongst them:

- Lipid layer: produced by the Meibomian glands
- Aqueous layer: produced by the lacrimal and the third eyelid glands
- Mucoid layer: produced by the globet cells of the conjunctiva
- Gycocalix is the internal layer that ¨links¨ the tear film with the corneal epithelium

To have a good tear film distribution we must have good eyelid anatomy, normal globe movement and a full and an intact blink.

The tear film contributes to the nutrition, oxygenation, immunity, lubrication, and cleaning of the corneal surface.

Tear film deficiency

This can be of a qualitative (lipid or mucoid layer) or a quantitative (aqueous layer) origin. We could also have problems with the distribution of the tears owing to lagophthalmia, incomplete blinks or eyelid abnormalities.

Several causes of the quantitative keratoconjunctivitis sicca have been described such as:

- Iatrogenic: removal of the nictitating membrane, radiation, drug related (i.e. sulphonamides, atropine), anaesthesia
- Viral: distemper
- Neurogenic
- Congenital: i.e. Yorkshire Terrier
- Endocrine: hypothyroidism, diabetes mellitus
- Other: chronic conjunctivitis, eyelid problems
- Immune-mediated: the more common cause

Clinical signs

Although the common presentation of keratoconjunctivitis sicca is the chronic form, the acute one can also occur. These animals present with acute pain, corneal ulceration that could be a melting process, a descemetocele, or a corneal perforation.

The more common chronic presentation tends to present with cases that are potentially diagnosed with recurrent conjunctivitis episodes. A Schirmer tear test will show in these cases a low result (remember it should be more than 15 mm/min). Some breeds like West Highland Terrier, Cavalier King Charles Spaniel, English Bulldog etc are predisposed to suffer from keratoconjunctivitis sicca. The ocular signs in these chronic cases consist of a dull corneal reflex, mucoid/mucopurulent discharge, +/- blepharospasm, +/- corneal ulceration, corneal vascularisation and pigmentation and recurrent/chronic conjunctivitis.

Diagnosis

We must perform the Schirmer tear test in all dogs with recurrent conjunctivitis, with corneal ulceration (contralateral eye) or corneal changes. Remember to place it on the lower lateral eyelid with direct contact with the corneal surface. It measures the basal and the induced component of the tear production. The STT values in dogs are:

- >= 15 mm/min= normal
- 11-14mm/min= early or subclinical
- 6-10mm/min= mild to moderate
- >=severe

Remember that the values must be accompanied by the ocular signs. If you are not sure about the presence or not of KCS, it is advisable to repeat the test within a few days. The tear production fluctuates over 24 hours. It can also be affected in stressed animals or animals that are receiving medication. Some cases can have a temporal decrease on the STT production or can be borderline cases. Monitoring would therefore be advised for them.

Presence of meiboimitis or a globet cell deficiency could affect the qualitative characteristics of the tear film leading to a qualitative problem.

The treatment available for KCS can be divided into lacrimostimulant and lacrimomimetic agents. It is important to educate the clients so they can understand that management rather than cure will be achieved.

The *lacrimostimulants* commonly used are cyclosporin and tacrolimus. They are both immune-modulators that supress T-cells. Different concentrations can be formulated but following the cascade the commercial

Optimmune, which is 0.2% cyclosporin, should be started. Treatment every 12 hours is usually given and, although an immediate effect can be seen in most cases, waiting a few weeks for the treatment to take effect is usually recommended. Tacrolimus may be effective in dogs nonresponsive to cyclosporine. Cyclosporin can also be use as episcleral implants in dogs that are difficult to medicate.

There may be a potential association between chronic corneal inflammation and topical immunosuppressive therapy in the development of corneal squamous cell carcinomas in dogs.

Treatment with topical cyclosporin, corticosteroids and tacrolimus have been associated with clearing of corneal pigmentation however the clinical efficacy of these treatments is unknown.

There are many *lacrimomimetics* available in the market. Viscoelastic substances with mucinomimetic properties include sodium hyaluronate, chondroitin sulfate, and 1%–2% methylcellulose preparations. These lacrimomimetic formulations should serve as effective viscoelastic and protectants for the ocular surface.

Surgery in the form of parotid duct transposition (PDT) is considered when medical treatment fails. It is a procedure that should be carefully discussed with the carers as it is not risk free. *Rhodes and colleagues* concluded that PDT is a successful procedure based on clinical findings and in terms of owner perception.

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PROTOCOLOS DE QUIMIOTERAPIA EN LA CLÍNICA DIARIA

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ABSTRACT BODY:

PROTOCOLOS DE QUIMIOTERAPIA EN LA CLÍNICA DIARIA

La quimioterapia es el uso de sustancias químicas (muchas de origen natural) para tratar el cáncer. Generalmente se administra en la dosis máxima tolerada (DMT) usando vías oral, intravenosa, intracavitaria o intratumoral. El uso de quimioterapia a dosis máxima tolerada en la clínica está indicado en diferentes situaciones:

- Tumores hematológicos que son sensibles a la quimioterapia.
- Como terapia adyuvante para prevenir la recurrencia local o retrasar el desarrollo de metástasis (p.e sarcomas, -hemangiosarcoma, osteosarcomas-, carcinomas, mastocitomas).
- Como tratamiento previo a cirugía intentando disminuir el tamaño del tumor (quimioterapia neoadyuvante).
- Como tratamiento paliativo en enfermedad metastática o inoperable.

La dosis de los fármacos citotóxicos se realiza sobre una estimación del área de superficie corporal, que se supone que es proporcional al metabolismo del animal. Sin embargo, este sistema puede producir sobredosificaciones en animales pequeños. Para algunos fármacos, como la doxorrubicina, la dosis en animales pequeños (de menos de 15 kg) se realiza en base al peso y no a la superficie corporal.

En general, las combinaciones de fármacos han demostrado ser más efectivas que el uso de uno sólo (excepciones a esto son la vincristina en el TVT o la doxorrubicina o el carboplatino en el OSA).

Principios generales del tratamiento.

Aunque ya se han mencionado las indicaciones generales de la quimioterapia, hay que tener en cuenta una serie de factores a la hora de establecer un tratamiento con quimioterapia.

- Factores relacionados con el paciente. La evaluación general del paciente, edad, presencia o no de enfermedades sistémicas, alteraciones funcionales, etc, influyen directamente en la elección del tratamiento para un determinado tumor. P.e. el uso de doxorrubicina no está indicado en pacientes caninos con cardiomiopatía dilatada o en gatos con fallo renal.
- Factores relacionados con el propietario. La predisposición al tratamiento, la tolerancia a los efectos secundarios, la disponibilidad para las visitas necesarias a la clínica, o el coste de los tratamientos deberían ser discutidos con los propietarios.
- Factores relacionados con la enfermedad. El objetivo principal es curar la enfermedad, pero cuando usamos quimioterapia (sin cirugía) raramente se puede conseguir. Si las posibilidades de curarlo son bajas, el objetivo del tratamiento será mantener una buena calidad de vida e intentar conseguir la remisión del tumor. Diferentes estadios clínicos de un mismo tumor (p.e. presencia de enfermedad metastática o no) hacen necesarios enfoques distintos para el tratamiento, pero en general un diagnóstico (y tratamiento) precoz, aumentan las posibilidades de curación. Esperar a ver qué pasa no debería ser una opción ofrecida a los propietarios.

Complicaciones y efectos secundarios de la quimioterapia.

Como se ha comentado, la quimioterapia afecta a células en división, pero lo hace de forma no selectiva, por lo que los tejidos normales del cuerpo con una alta tasa de células en división se ven siempre afectados. De hecho, la dosificación de la quimioterapia viene limitada por la tolerancia de los tejidos normales afectados (digestivo y médula ósea). Es importante explicar al propietario qué efectos podemos observar y hasta dónde esperamos que se produzcan.

Se pueden observar alteraciones gastrointestinales durante los tratamientos con quimioterapia (anorexia, vómitos o diarreas) aunque generalmente son leves o moderados y responden al tratamiento sintomático. Podemos anticiparnos a su presentación conociendo los efectos de los fármacos que usamos. El uso de maropitant en protocolos de doxorrubicina ha demostrado disminuir la frecuencia e intensidad de los vómitos asociados a este fármaco, por lo que se recomienda su uso el mismo día y los cuatro posteriores a la administración de la doxorrubicina.

Otro tejido afectado por la quimioterapia es la médula ósea. El descenso al nivel más bajo de neutrófilos ocurre generalmente a los 7-10 días (dependiendo del fármco). Generalmente el número de neutrófilos es el

que nos limita la dosis de quimioterapia que podemos usar, siendo importante controlar también el de las plaquetas. Raramente vemos anemia secundaria a la quimioterapia que requiera cambios en las dosis de los fármacos.

Otros efectos secundarios, más específicos de cada fármaco, deben conocerse para intentar prevenirlos o, en su caso, tratarlos tan pronto como se produzcan (cistitis con ciclofosfamida, cardiotoxicidad con doxorrubicina, dermatitis o necrosis por extravasación de vincristina o doxorrubicina respectivamente). Además es importante conocer las toxicidades en distintas especies. Usando el caso de la doxorrubicina como ejemplo, produce nefrotoxicidad en el gato pero no en el perro, y los gatos no se ven afectados dela cardiotoxicidad que ocurre en la especie canina. Otros fármacos, como el cisplatino o el 5-Fluorouracilo son letales para el gato por vía intravenosa por lo que están contraindicados en esta especie aunque sí se pueden administrar así en perros.

Manejo seguro de fármacos citotóxicos.

La exposición continua a los fármacos citotóxicos puede suponer un riesgo para el personal que los manipula. En veterinaria además podemos tener contacto con saliva, heces u orina de los pacientes, que es donde se pueden encontrar residuos de la quimioterapia. Se recomienda el uso de sistemas cerrados de administración de quimioterapia y conocer y cumplir las normativas locales sobre manejo de fármacos y residuos citotóxicos.

Protección del paciente.

La prescripción del tratamiento, dosis y vías de administración, deben realizarse por alguien con experiencia. El margen terapéutico de estos fármacos es estrecho y el riesgo de toxicidad alto si hay errores en la dosificación. Se recomienda que todos los cálculos de dosis sean revisados por dos personas para minimizar el riesgo de equivocación. Además, algunos de los fármacos usados frecuentemente pueden producir lesiones graves si se administran accidentalmente fuera de la vena, por lo que es importante usar una buena técnica y si hay la más mínima duda, cambiar a otra vena o retrasar la administración del tratamiento. La administración por parte de personal entrenado y con experiencia minimiza el riesgo de extravasación.

PROTOCOLOS PARA LINFOMA CANINO

WISCONSIN-MADISON 19 SEMANAS

Anotar siempre extremidad en la que se administran VIN y DOXO.* Mientras tome corticoides, se puede añadir omeprazol
SID por la mañana, en ayunas.

** Ajustar a un número entero de grageas (no se pueden partir las grageas) y dividir la dosis total en dos días para reducir la
incidencia de Cistitis Hemorrágica Estéril. Si desarrolla Cistitis Hemorrágica Estéril, cambiar la ciclofosfamida por clorambucilo
(Leukeran 2 mg) a una dosis de 1.4 mg/kg PO.

*** Si el perro pesa menos de 15 kg, la dosis de doxorrubicina es 1 mg/kg IV.

Semana 1: Vincristina: 0,5-0,75 mg/m² IV,

Prednisona*: 2 mg/kg PO cada 24 horas.

Semana 2: Ciclofosfamida**: 200-250 mg/m² vía oral.

Prednisona*: 1,5 mg/kg PO cada 24 horas

Semana 3: Vincristina: 0,5-0,75 mg/m² IV,

Prednisona*: 1 mg/kg PO cada 24 horas.

Semana 4: Doxorrubicina***: 30 mg/m² IV (o 1 mg/kg si <10 kg) IV,

Prednisona*: 0,5 mg/kg PO cada 24 horas.

Semana 5: Sin tratamiento.

Semana 6: Vincristina: 0,5-0,75 mg/m² IV.

Semana 7: Ciclofosfamida**: 200-250 mg/m² vía oral

Semana 8: Vincristina: 0,5-0,75 mg/m² IV.

Semana 9: Doxorrubicina: 30 mg/m² IV (ó 1 mg/kg si <10 kg) IV.

Semana 10: Sin tratamiento.

Semana 11: Vincristina: 0,5-0,75 mg/m² IV.

Semana 12: Ciclofosfamida**: 200-250 mg/m² vía oral

Semana 13: Vincristina: 0,5-0,75 mg/m² IV.

Semana 14: Doxorrubicina: 30 mg/m² IV (ó 1 mg/kg si <10 kg) IV.

Semana 15: Sin tratamiento.

Semana 16: Vincristina: 0,5-0,75 mg/m² IV.

Semana 17: Ciclofosfamida**: 200-250 mg/m² vía oral.

Semana 18: Vincristina: 0,5-0,75 mg/m² IV.

Semana 19: Doxorrubicina: 30 mg/m² IV (ó 1 mg/kg si <10 kg) IV.

COP (Se repite durante seis-ocho semanas).

Ciclofosfamida: 50 mg/m² PO cada 48 horas en perros ó 300 mg/m² PO cada 3 semanas en perros y gatos.

Vincristina: 0,75 mg/m² IV un día por semana.

Prednisona: 40-50 mg/m² PO cada 24 horas durante una semana y después 20-25 mg/m² PO cada 48 horas.

Fase de mantenimiento: Tras coompletar 6-8 semanas de COP se hace mantenimiento

Clorambucilo: 20 mg/m² PO cada 2 semanas.

Metotrexato: 2,5 mg/m² PO dos o tres veces por semana.

Prednisona: 20 mg/m² PO cada 48 horas

PROTOCOLOS PARA SARCOMAS EN PERROS

VAC (Protocolo de 28 días)

Día 1: Doxorrubicina: 30 mg/m² (ó 1 mg/kg si <10 kg) IV

Día 8: Vincristina: 0,75 mg/m² IV

Día 15: Ciclofosfamida: 200-300 mg/m² PO

Día 21: Vincristina: 0,75 mg/m² IV

Sulfa-trimetropin: 15 mg/kg/12 horas PO

Día 29 se comienza un dminis ciclo. Se dministrant 3-4 ciclos si se tolera bien.

AC (Protocolo de 21 días)

Día 1: Doxorrubicina: 30 mg/m² (ó 1 mg/kg si <10 kg) IV

Día 10: Ciclofosfamida: 200-300 mg/m² PO

Día 21: se comienza un dminis ciclo. Se dministrant 3-4 ciclos.

PROTOCOLOS PARA CARCIOMAS CANINOS

Carboplatino 300 mg/m² cada tres semanas (puede ser necesario disminuir la dosis en perros de menos de 10 Kg a 10 mg/Kg)

Hacer un hemograma a los 7-10 días de la primera dosis para vigilar el número de neutrófilos y ajustar dosis siguientes si
fuera necesario.

TUMORES ORALES

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ABSTRACT BODY:

1. INTRODUCCIÓN

La cirugía de los tumores orales es apasionante, vivirla con entusiasmo ayuda a convencer a los propietarios de nuestras mascotas a seguir con los tratamientos quirúrgicos que les proponemos, la mejora en la calidad de vida y la curación del proceso son hechos que nos llevan a este entusiasmo. El aspecto estético poco importa y mejorar la supervivencia manteniendo calidad de vida justifica cualquier técnica quirúrgica por muy agresiva que nos parezca.

Los tumores orales son neoplasias que crecen desde la encía, mucosa bucal o labial, lengua, tonsilas o periodonto. Representan el 6% y el 7,3% de todas las neoplasias del perro y gato, respectivamente. La incidencia de los tumores orales y maxilofaciales es del 0,5% . Una gran variedad de lesiones podemos encontrar en la cavidad oral, según un estudio reciente, el 61% de las lesiones que aparecían en la cavidad oral del perro eran neoplásicas siendo el resto hiperplásicas e inflamatorias correspondiendo exclusivamente a un 27% en el caso de los gatos.[1,2]

Aunque en los últimos años la nomenclatura ha ido cambiando los tumores dependientes del entorno del ligamento periodontal siguen siendo los más frecuentes teniendo buen pronóstico, y han modificado su nombre de épulis por ameloblastomas, ya por orden de frecuencia sigue el **carcinoma de células escamosas** (CCE), el **melanoma maligno**(MM) el **fibrosarcoma** (FBS) y por último, el **osteosarcoma** (OS).

Existen factores predisponentes a sufrir tumores orales como son la *edad* (geriátricos), el *sexo* (mayor riesgo en machos), *raza* (Cocker Spaniel, Caniche, Husky, Pastor Alemán, Pointer, Golden Retriever, San Bernardo y Boxer) y *pigmentación de la mucosa oral* (mayor predisposición a padecer melanoma maligno). [3]

FIBROMAS PERIFÉRICOS ODONTOGÉNICOS

Los Épulis, así llamados anteriormente suponen el 30% de los tumores orales y se localizan frecuentemente en las encías de los dientes incisivos y actualmente se clasifican de manera diferente. El fibroma odontogénico periférico es un tumor benigno, los bóxers de edad avanzada (8-9 años) machos están bien representados y su exéresis es sencilla, mediante bisturí eléctrico. Eran los clásico épulis fibromatosos y osificantes. Hay proliferaciones epiteliales a partir del ligamento periodontal con aspecto similar a hiperplasias gingivales.[3]

El ameloblastoma acantomatoso, incluido antes como épuli acantomatoso aunque está clasificado como tumor benigno, es un tumor que requiere un tratamiento quirúrgico radical, por ser un tumor muy agresivo localmente ya que invade el tejido adyacente y el hueso produciendo lisis ósea.

La cirugía radical (mandibulectomía/maxilectomía) es casi siempre curativa, la tomografía ayuda a determinar los límites, en el bloque de escisión incluiremos una pieza dental sana por cada lado de la masa. Una opción es la radioterapia, pero no lo tenemos disponible y en algunos casos lo sustituimos por electroquimioterapia.

CARCINOMA DE CÉLULAS ESCAMOSAS

Es el segundo tumor oral más frecuente en perro y el más frecuente en el gato. Su conducta biológica es extremadamente dependiente de su localización, tamaño y edad. Suele aparecer en la encía (en un 80% de los casos), sobretodo en la porción rostral de la mandíbula y maxila y en animales mayores (de unos 9 años de edad). Hay un tipo histológico papilar que puede afectar a cachorros hasta 9 meses y nos puede confundir con los épulis.

Tienen mejor pronóstico aquéllos localizados en la maxila, rostralmente y en perros de menos de 6 años, es invasivo localmente pero tiene una ratio de metástasis a distancia muy baja.

De forma infrecuente puede afectar a la mucosa de la lengua (más agresivo que en la encía y mayor probabilidad de metastatizar) y labios.

El CCE tonsilar es el tumor más agresivo localizado en la cavidad oral y presenta una capacidad metastática mayor.

Una cirugía radical es la técnica de elección. No encontrar células tumorales en los ganglios mandibulares y retrofaríngeos todavía mejora más el pronóstico. Pueden combinarse con la cirugía la radioterapia + hipertermia y la electroquimioterapia si hay metástasis.

En el gato hay una clara correlación entre el carcinoma de células escamosas, el uso de collares insecticidas

y el humo del tabaco pudiendo incrementar hasta en 4 veces la incidencia de este tipo de tumores.

FIBROSARCOMA

Es el tercer tumor maligno más frecuente en el perro y el segundo del gato. Las zonas de mayor afectación son: el tejido gingival de los molares superiores y en el paladar blando adyacente, y en la mitad anterior de la mandíbula inferior. Actúa de forma agresiva pero raramente metastatiza.

Los machos y las razas grandes, retrieever en concreto presentan mayor predisposición a padecer esta neoplasia. Aunque la edad de presentación es avanzada, puede afectar a animales jóvenes.

El tratamiento de elección es la cirugía agresiva mediante maxilectomía o mandibulectomía para obtener unos márgenes libres de células tumorales.

Se recomienda la combinación de la cirugía radical + radioterapia y/o quimioterapia. No responde a la crioterapia.

MELANOMA MALIGNO

El melanoma maligno es el tumor de la cavidad oral más difícil de tratar y de peor pronóstico, aunque un diagnóstico agresivo y una técnica contundente nos pueden llevar a la curación. Sus lesiones crecen rápidamente, los ganglios linfáticos regionales ya suelen estar afectados y la metástasis pulmonar es común en el momento de su diagnóstico.

Las zonas de la cavidad oral que afectan con mayor frecuencia son: encías, paladar, superficie dorsal de la lengua y labios. Suele presentarse en animales viejos y sobretodo en razas pequeñas con pigmentación oral.

El tamaño y localización del tumor afecta el pronóstico de la enfermedad, así los localizados en la mandíbula rostral y maxila caudal tienen más tiempo de supervivencia.

El tratamiento de elección es la escisión quirúrgica radical, aún así, las recidivas son frecuentes. Técnicas coadyuvantes utilizadas son: la radioterapia, la quimioterapia, o inmunoterapia consiguiendo tiempos de supervivencia más prolongados.

1.3. SIGNOS CLÍNICOS

Como ya hemos visto, el éxito del tratamiento está en el diagnóstico precoz y la actuación rápida. El propietario acostumbra a darse cuenta tarde del proceso y más cuando son muy caudales, es por ello muy importante aprovechar revisiones anuales durante la vacunación para realizar un examen minucioso de la cavidad oral y encontrar posibles masas que nos puedan hacer sospechar estos procesos.[4]

Normalmente acuden a la consulta por la presencia de una masa en la cavidad oral, asimetría facial o por los síntomas que pueden dar estas masas: disfagia, dificultad en la masticación, halitosis, salivación sanguinolenta, desplazamiento o pérdidas de dientes, cara hinchada o deformación, exoftalmia, dolor al abrir la boca, engrosamiento óseo de la maxila y/o mandíbula.

Si existen metástasis, podríamos observar engrosamiento o hipertrofia de los ganglios linfáticos mandibulares, retrofaríngeos o cervicales y síntomas de otros órganos asociados a metástasis o propios del síndrome paraneoplásico.

1.4. DIAGNÓSTICO

Realizaremos citologías y/o biopsias de la masa, para determinar de qué tipo de neoplasia se trata, y de los nódulos linfáticos regionales para evaluar la presencia de metástasis.

Es imprescindible realizar pruebas complementarias para determinar la extensión de la masa y afectación ósea. Podemos realizar radiografías pero un TAC actualmente es lo más habitual con información superior a la RNM ya que delimita mejor los procesos óseos.

1.5. TRATAMIENTO

La exéresis quirúrgica rápida y agresiva del tumor es el tratamiento de elección para el control local del mismo y sigue siendo el tratamiento de elección.[5]

Otros tratamientos, normalmente complementarios, incluyen: la criocirugía, la electroquimioterapia, el cisplatino intralesional (braquiterapia) radioterapia, la hipertermia, terapia fotodinámica, quimioterapia e inmunoterapia.

La radioterapia es una técnica efectiva en el control local del tumor pero nosotros no la tenemos disponible, mientras que la quimioterapia, además de ser una ayuda en el tratamiento local para el melanoma maligno, puede ser efectiva en pacientes que presenten metástasis.

Después de diversos estudios no se ha encontrado una utilidad aceptable de la criocirugía en el tratamiento de tumores orales, únicamente sirve como una terapia citoreductora que se debe ir repitiendo si mejora la calidad de vida del paciente.

1.6. CONSIDERACIONES ANESTÉSICAS

La elección del protocolo anestésico dependerá de la evaluación preoperatoria del animal, teniendo en cuenta principalmente que los animales afectados son perros de edad avanzada.

Siempre administraremos analgésicos opioides y, en el caso de realizar mandibulectomía o maxilectomía, procedimientos altamente dolorosos, proporcionaremos una buena cobertura analgésica

mediante agonistas puros. Podemos administrar fentanilo en bolo o infusión durante el período perioperatorio y fentanilo en parche para el postoperatorio, ayudado de los AINES. En los últimos tiempos está ganando adictos los bloqueos de nervios mentonianos, mandibular y maxilar así como los splash postquirúrgicos con bupivacaina y ropivacaína.

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THE HIP - A BALL & SOCKET AND ITS CHALLENGES FROM BIRTH TO DEATH

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ABSTRACT BODY:

THE HIP - A BALL & SOCKET AND ITS CHALLENGES FROM BIRTH TO DEATH

Introduction

In the canine and feline population there are a series of disorders that affect the coxofemoral joints and cause differing degrees of pain, lameness, and dysfunction. As these disorders often present at specific times in the patient's life having an accurate signalment and history regarding severity and progression is important. The presence and severity of pelvic limb lameness along with definitive examination findings of hip joint pain and changes in range of motion is a strong indication for pelvic saddle imaging.

Examination:

Following gait exam and determination of the presence and severity of a pelvic limb lameness, a complete pelvic limb exam should be performed. During the initial exam phase, when situated behind the patient, it is helpful to attempt to determine if there is a differential in the amount of weight being born by the left and right pelvic limbs. It is also wise when commencing the exam to test for conscious proprioceptive response in both pelvic limbs to examine for a possible concurrent pelvic limb neuropathy.

It is very informative when assessing possible hip pathology to examine the hip joint's range of motion (ROM). Patients rarely have a loss of hip joint flexion and it is change in hip joint extension that indicate possible hip joint pathology. The normal reported values for hip flexion/extension in the Labrador Retriever are 50/162 degrees. Normal abduction should be at least 90 degrees and internal/external rotation should also be concurrently assessed. No crepitus should be noted on range of motion. The presence of patient discomfort and pain on hip extension combined with a loss of normal ROM primarily indicates coxofemoral joint abnormality. Patient's with caudal lumbar pathology or sciatic nerve nerve pain syndrome (NPS) will often also demonstrate discomfort at full extension and will resist efforts to fully extend the hip joint. Hip extension with internal rotation may indicate iliopsoas m. myopathy or tendinopathy. This pathology is uncommon and often over-diagnosed.

It is very difficult to assess joint effusion in the hip based on palpation unless there is severe muscle atrophy combined with massive effusion (as in a septic hip joint).

Determining the degree of hip joint laxity is an important part of examining juvenile patients and the performance of an Ortolani and Barlow maneuver can be very helpful in determining the presence and severity of inherent congenital laxity. These exams are best performed in a sedated patient in dorsal recumbency and although some information can be gleamed from an unsedated patient in lateral recumbency, the challenge of patient anxiety and pain will adversely affect the accuracy and reproducibility of results. The Ortolani maneuver is most commonly conducted in dogs one year or less in age.There are two components to this test: The Barlow and the Ortolani. The Barlow is defined by hip subluxation and the Ortolani maneuver by hip reduction. To conduct these tests, maintain the stifle and hip at about 90 degrees of flexion. Apply pressure axially along the shaft of the femur to subluxate the hip. (Barlow Positive). This should not occur if the hip is normal in dogs older than 6 months of age. If excessive hip joint laxity is present, the femoral head subluxates dorsally out of the acetabulum, providing a positive Barlow test.Then, keeping constant and slight axial pressure towards the acetabulum the limb is slowly abducted, and a 'pop' can be felt as the femoral head relocates into the acetabulum. (Ortolani positive).The angle of reduction can be measured at this point (Ortolani angle). The second portion of the test is to slowly return the femur to upright position until the femoral head disengages from the acetabulum and once again dorsally subluxates. The angle to vertical femur position is measured and this is termed the Barlow angle. The Barlow angle approximates the angle of the articular surface of the dorsal acetabular articular surface to horizontal and (dorsal acetabular angle). This angle is variable in normal dogs (usually < 8 deg) and in dysplastic dogs with acetabular malformation or dysplasia the angle increases as the severity of the dysplasia syndrome increases.

Pathologic Conditions:

Pathology of the coxofemoral joint has breed, age

and individual underlying predispositions that should be considered carefully when the results of clinical examination indicate hip area pathology. As a juvenile canine or feline, the capital physis of the femoral head is prone to Salter-Harris type fractures. The majority of these fractures are Type I with the 'slipped capital physis' being used as the common description. A combination of trauma, genetic and endocrinological causes prevail with young male cats and boisterous large breed dogs being over-represented. Diagnostic imaging includes well-positioned sedated pelvic radiographs with ventro-dorsal views in both the hip extended and 'frog leg' position being helpful. Treatment is surgical in the majority of cases with primary repair and femoral head and neck excision (FHNE) predominating. Fractures of the femoral neck are also commonly seen in pelvic trauma cases with similar treatment options as capital physeal fractures. The development of ischemic necrosis of the femoral neck in Legg-Calvé-Perthes (LCP) disease in small breed juvenile dogs is seen commonly and is characterized by progressive pelvic limb lameness, often with a skipping component with pain on direct examination of the hip joint. Treatment options include FHNE with follow-up physical rehabilitation and total hip replacement (THR). The development of the ventral approach to the hip joint for FHNE has improved the clinical recovery of patients in many cases and has allowed the dorsal capsule and musculature to not be surgically traumatized.

Hip Dysplasia

Hip dysplasia (HD) is a polygenic trait which results in periarticular soft tissue laxity and concurrent abnormal formation of the acetabulum. Hip laxity is a crucial component of hip dysplasia.It is important to assess young dogs for the presence of HD as early as possible to increase the availability of treatment options.Radiographically the PennHip examination is able to accurately assess the presence and predict the severity of hip laxity in patient from 16 weeks of age. This is very helpful when determining affected animals candidacy for Juvenile Pubic Symphysiodesis (JPS) which must be performed before 20 weeks of age to maximize its beneficial results. The combination of the PennHip Distraction Index (DI) and a measurement of acetabular angle via either CT scan or dorsal acetabular rim (DAR) view allows assessment of juvenile dogs who are moderately affected with HD as candidates for triple pelvic osteotomy (TPO). As dogs affected with HD age their hip joints will deteriorate as their osteoarthritis (OA) and degenerative joint disease progress. Cartilage loss, chronic fibrosing synovitis, persistent instability, and concurrent pelvic musculature atrophy results in a range of dysfunction and discomfort from mild to severe. Medical therapy for OA should be multi-modal in nature, be re-assessed by the clinician routinely and when it becomes ineffective, and patients are in persistent pain with a significant decrease in their mobility and quality of life salvage surgery should be considered. Use of the FHNE and total hip replacement may allow a significant improvement in both pain scores and function for affected patients. The choice of which procedure to recommend and when it is to be performed is a complex one that should be explored with the client carefully, with good attention to both the client's concerns and their ability to commit to the post-operative period in a detailed and consistent fashion.

Hip Luxation

The traumatic luxation of the femoral head, more commonly in a cranio-dorsal direction than ventrally, is a treatment challenge that should be addressed promptly and completely. The decision of closed versus open reduction is one that is affected by the time lag to initial treatment, ability to reduce, degree of instability post-closed reduction, presence of underlying hip dysplasia, access to experienced orthopedists and presence of significant trauma to the articular surfaces. Following stable closed reduction and Ehmer-style sling placement in patients with normal hip morphology there is an approximately 50% chance of reduction being maintained and the joint being functional. In unstable reduced hips or in hips that are unable to be reduced the surgical options include open reduction and stabilization, total hip replacement or FHNE. All these options have inherent challenges and treatment of individual patients should consider those challenges and discussion with owners should focus on the strengths and weaknesses of the different surgical options in relationship to the individual patient. Clinicians should be careful to not commit to personally perform a procedure they are not trained to perform, are not experienced in performing and are not comfortable going ahead with.

CÓMO IDENTIFICAR EL PROBLEMA EN LOS PACIENTE CON SANGRADO EXCESIVO

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ABSTRACT BODY:

La función del sistema hemostático es limitar el sangrado ante daño vascular formando un coágulo, normalizar el flujo sanguíneo y la perfusión tisular disolviendo el mismo alcanzada la reparación vascular definitiva y limitar localmente estos procesos.

Los desórdenes hemostáticos más comunes pueden ser diagnosticados en forma presuntiva considerando anamnesis, signos clínicos, evaluación del frotis sanguíneo y ciertas pruebas diagnósticas rápidas para evaluar la cascada de la coagulación. El diagnóstico definitivo puede requerir la realización de pruebas adicionales.

El tratamiento incluye el soporte vascular, el tratamiento de la enfermedad subyacente, y el remplazo o corrección de los factores hemostáticos deficientes.

La anamnesis es fundamental para descartar la presencia o no de un disturbio hemostático, su carácter congénito hereditario versus adquirido e identificar la causa primaria.

Debemos diferenciar la presencia de enfermedades locales (carcinoma nasal, cálculos urinarios, etc.) de los trastornos hemostáticos, y hemoglobinuria de hematuria mediante centrifugación de la orina.

En las razas predispuestas (Tabla 1), la referencia a mortalidad perinatal aumentada y episodios anteriores puede sugerir un problema congénito/hereditario. En aquellas recesivas ligadas al cromosoma X, los machos están sobrerrepresentados.

DEFECTOS CONGÉNITOS DE FACTORES DE LA COAGULACIÓN		
Factor	**Defecto o Enfermedad**	**Razas**
Factor I	Hipofibrinoginemia Disfibrinoginemia	San Bernardo, Borzoi
Factor II	Hipoprotrombinemia	Boxer
Factor VII	Hipoconvertinemia	Beagle, Malamute
Factor VIII	Hemofilia A	Diversas razas
Factor IX	Hemofilia B	Diversas razas caninas, gatos doméstico de pelo corto y británico de pelo corto
Factor X	Rasgo de Stuart-Prower	Cocker spaniel
Factor XI	Hemofilia C	Springer spaniel, Terrier azul de Kerry, Gran pirineo
Factor XII	Factor de Hageman	Diversas razas caninas y felinas

Las vacunas a virus vivo y algunas drogas pueden ocasionar trombocitopenia, trombocitopatías e inactivación de factores de la coagulación.

La uremia, insuficiencia hepática, enfermedad GI inflamatoria crónica, AHI, neoplasias, infecciones sistémicas, hemoparásitos pueden ocasionar desórdenes hemostáticos secundarios.

Los desórdenes hemostáticos se presentan como sangrado en ausencia de injuria externa, o como prolongación del sangrado esperado luego de injuria vascular.

Los defectos plaquetarios limitan la formación del tapón primario, pero se activa la cascada de la coagulación, resultando en sangrados pequeños o limitados (petequias, equimosis) y sangrado mucoso (epistaxis, hematuria, hematoquecia y melena).

La disfunción de la cascada de la coagulación impide la estabilización del tapón plaquetario primario por la red de fibrina. Su rápida desestabilización y disolución permite la recurrencia del sangrado. El sangrado es detenido finalmente por la formación demorada de un coágulo de fibrina o por la presión ejercida por los tejidos circundantes. Observándose sangrados profundos más profusos, hematomas, hemartrosis, hemorragia muscular y sangrado cavitario. Pueden asociarse también a hemorragias pulmonares, mediastinales y retrobulbares.

La fibrinolisis patológica resulta del exceso de activadores o disminución de inhibidores de plasmina e hiperplasminemia. La digestión del complejo fibrinógeno-trombina y de la fibrina disuelve prematuramente el coágulo. La disminución

consecuente de protrombina, factores V, VIII y XII, y el aumento de productos de degradación de fibrinógeno FDP aumentan la tendencia al sangrado. Son más frecuentes los procesos fibrinolíticos secundarios a coagulación intravascular diseminada que los primarios.

El **perfil hemostático** completo incluye: frotis sanguíneo, tiempo de sangrado mucoso (TSM), conteo plaquetario, tiempo de protrombina (TP), tiempo de tromboplastina activado (TTPA), tiempo de coagulación activado (TCA) y determinación de factores de degradación del fibrinógeno. Algunas situaciones requieren pruebas específicas, como el antígeno relacionado al FVIII (enf. von Willebrand), o el PIVKA (protein induced by vitamin K antagonism) en pacientes intoxicados con antagonistas de la vitamina K.

Se puede obtener un diagnóstico presuntivo que posibilite un manejo inicial en base a un hematócrito (Hto.), proteínas plasmáticas totales (PPT), estimativo plaquetario, TSM y TCA.

El Hto. y las PPT estiman el grado de hemorragia y la necesidad o no de proveer glóbulos rojos. El estimativo plaquetario y el TSM posibilitan el diagnóstico de desórdenes hemostáticos primarios; y el TCA evalúa en forma grosera la presencia de defectos hemostáticos secundarios.

Los defectos hemostáticos pueden ser secundarios a otras enfermedades, y/o ocasionar disfunción orgánica secundaria; el examen completo debería incluir hemograma completo, perfil bioquímico, urianálisis, serología, diagnóstico por imágenes y/o biopsias.

El **frotis sanguíneo** evalúa un estimativo y la morfología plaquetaria. La presencia de acúmulos plaquetarios en los bordes del frotis revela activación plaquetaria, induciendo a estimativos y conteos erróneos, indicando la necesidad de una nueva muestra.

El estimativo plaquetario es el promedio de plaquetas por campo de inmersión (normal 10-15). Cada plaqueta representa entre 12000-15000 plaquetas/ul. Si la función plaquetaria es normal, recuentos >30000/ul raramente inducen sangrado espontáneo, estimativos >3-5 descartan trombocitopenia como etiología.

Gran número de plaquetas grandes sugiere trombopoiesis activa. Las plaquetas grandes presentan mayor funcionalidad. Los hemocitómetros brindan información objetiva del volumen plaquetario medio (VPM). La masa plaquetaria, o el producto del número de plaquetas por su VPM, es una medida más funcional.

Un hemocitómetro permite un conteo manual. Los contadores automáticos resultan más precisos. Sin embargo, los analizadores que estudian el grosor del buffy coat, brindan resultados erróneos en presencia de plaquetas grandes.

El **tiempo de sangrado mucoso** evalúa la función plaquetaria. Se realiza un corte estandarizado en la mucosa bucal y se mide el tiempo al cese del sangrado. Evitar el contacto con el corte porque interfiere la formación del tapón hemostático primario. Es normal 2 a 3min. en caninos y 1,5 a 2,5min. en felinos.
Se prolonga en trombocitopenia, trombocitopatías y enfermedad de von Willebrand. En defectos hemostáticos secundarios el TSM es normal, pero dada la ausencia de estabilización secundaria del tapón plaquetario por la fibrina el paciente vuelve a sangrar desde el sitio de punción.

DIAGNÓSTICO DE PROBLEMAS HEMOSTÁTICOS				
	Plaquetas	**Factores de la coagulación**	**Sistema Fibrinolítico**	**Vasos sanguíneos**
Función normal	Tapón plaquetario	Coágulo de fibrina	Disolución del coágulo	Retener la sangre
Formas de evaluación	***Trombocitopenia*** - Conteo plaquetario - Estimativo plaquetario ***Trombocitopatía*** - Tiempo de sangrado mucoso	***Coagulopatías*** - Tiempo de Coagulación Activado (TCA) - vía intrínseca y común - - Tiempo Parcial de Tromboplastina Activado (TPTA) - vía intrínseca y común - - Tiempo de Protrombina (TP) - vía extrínseca -	***Coagulopatías Consuntivas*** - Productos de Degradación del Fibrinógeno (FDP)	***Vasculopatías*** - Histopatología

Tabla 2

El **Tiempo de coagulación activado TCA** aunque poco sensible, permite descartar la presencia de coagulopatías asociadas a factores presentes en la vía intrínseca (factores XII, XI, IX, y VIII) y común

(factores X, V, II y I). Colocar 2ml de sangre en un tubo con tierra de diatomea como activante (tubo tapa gris), e incubar a 37º C durante 60seg., el tubo es invertido luego cada 5seg. hasta la formación de coágulos. El TCA normal es 60 a 110seg. caninos y 50 a 75seg. felinos.

Dado que las plaquetas proporcionan el fosfolípido (FP3), las trombocitopatías severas pueden prolongar el TCA en presencia de niveles normales de factores de la coagulación.

Tiempo parcial de tromboplastina activado

TPTA es más sensible y específico que el TCA, en la evaluación de la vía intrínseca y común, detectando defectos que no pueden ser determinados por el TCA (portadores de hemofilia A o B). El TPTA no se afecta por trombocitopenia ya que el reactivo utilizado como activante provee el fosfolípido necesario.

Tiempo de protrombina TP evalúa la vía extrínseca (FVII) y la común. En el antagonismo de vitamina K (FII, VII, IX, y X), el factor VII es de vida media más corta; y se afecta antes. El TP en conjunción con el TPTA, delimita un problema a una vía particular. Un defecto en la vía extrínseca prolongará el TP, pero no al TPTA, y un defecto en la vía intrínseca aumentará el TPTA, con TP normal. La deficiencia de un factor perteneciente a la vía común afectará ambas pruebas.

Existen **Tests específicos** para identificar la deficiencia de un factor particular como en la hemofilia y la enfermedad de von Willebrand.

RESULTADOS ESPERADOS DEL PERFIL HEMOSTÁTICO EN DIFERENTES ENFERMEDADES

	Perfil Hemostático					
Enfermedad	**Tiempo de sangrado**	**Conteo plaquetario**	**TCA**	**TTPA**	**TP**	**FDP**
Trombocitopenia	A	D	N	N	N	N
Trombocitopatía	A	N	N	N	N	N
Defectos de la vía intrínseca (Hemofilia A o B)	N*	N	I	I	N	N
Deficiencia de FVII	N	N	N	N	A	N
Defectos múltiples (antagonismo de la vit. K)	N*	N	A	A	A	N
CID	A	D	A	A	A	A
Enfermedad de von Willebrand	A	N a D	N a A	N a A	N	N

A: aumentado N: normal D: disminuido * Inicialmente se detiene pero puede volver a sangrar

Tabla 3

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ENVIRONMENTAL CHANGES AND THE IMPACT ON SMALL COMPANION ANIMALS

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ABSTRACT BODY:

ENVIRONMENTAL CHANGES AND THE IMPACT ON SMALL COMPANION ANIMALS

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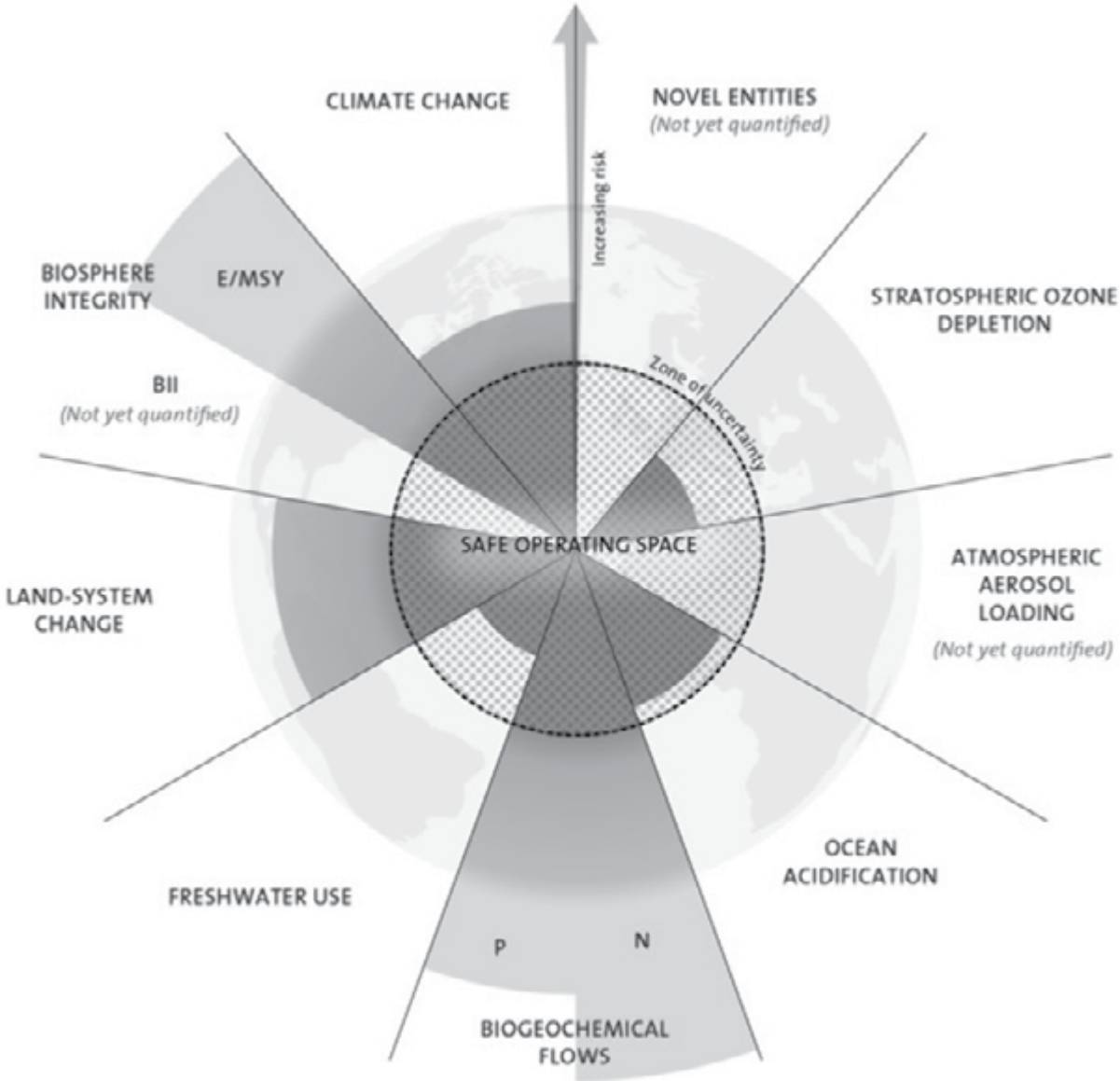
The world continues to be marred by numerous problems due to bad environmental stewardship. These include issues such as climate change, the depletion of biodiversity, pollution, deforestation, and overexploitation of natural resources.

The planetary boundaries, nine processes that regulate the stability and resilience of the Earth system, were identified by a group of 28 internationally renowned scientists, which suggests that our developmental trend is not sustainable and therefore change is needed



(Fig.1). Climate change which results in global warming is one [1].

The planetary boundaries [1].

Global warming is certainly the main environmental challenge that need to be tackled by the world. This phenomenon leads to ocean acidification, melting ice caps, sea level rise and unprecedented natural disasters. These natural disasters include tropical storms, hurricanes, typhoons, heat waves, forest fires and flooding which are more intense and frequent than we are accustomed to. This is because of the increased emissions of greenhouse gasses mainly due to the use of fossil fuels, manufacturing activities and deforestation.

These effects of global warming affect companion animals as much as they do humans. This affectation is harm indirectly caused by disturbing ecological systems and the processes of nature [2]. In many respects, animals are more vulnerable than humans since they tend to be more in contact with the earth, do not wear protective clothing, and are very dependent on pet owners for protection. This vulnerability is partly why companion animals are useful indicators in identifying sources of environmental pollution, food contaminants, infectious disease transmission and other harmful exposures [3].

While it is essential that we consider the effects of environmental changes on companion animals, it is important to be cognizant of the fact that many factors associated with the keeping of companion animals contribute to the exacerbation of those very environmental changes. This is why a harmonious relation with the environment is needed, as is adequately represented in the Japanese concept of Satoyama-Satoumi [4].

The World Small Animal Veterinary Association (WSAVA) One Health Committee outlined three key areas of One Health regarding companion animals: (1) The human–companion animal bond, (2) comparative and translational medicine, and (3) zoonotic infectious disease [5]. From a One Health perspective, we must broaden our scope to fully consider issues such as Climate Change. To this end, clinicians must redefine what a healthy companion animal is. It can no longer be considered that an animal is healthy simply because it is in a biological state of absence of disease. Rather, within the context of climate change a healthy animal must be capable of responding adequately to the stressors of climate change such as heat stress [6].

The effects of Climate Change on small companion animals are generally centered around increased ambient temperatures which lead to heat-related illness. This is particularly compounded when there is inadequate shade, water, and ventilation. Pet owners need to be aware of the level of risk their animal possess based on factors such as age, body condition, health, reproductive status and breed. Heat stroke manifests itself in different ways and include abnormal breathing, lethargy, collapsing, and diarrhea. Pet owners will need the right guidance to keep their animals safe and clinicians must be prepared to provide advice accordingly. This is critical since all animals are

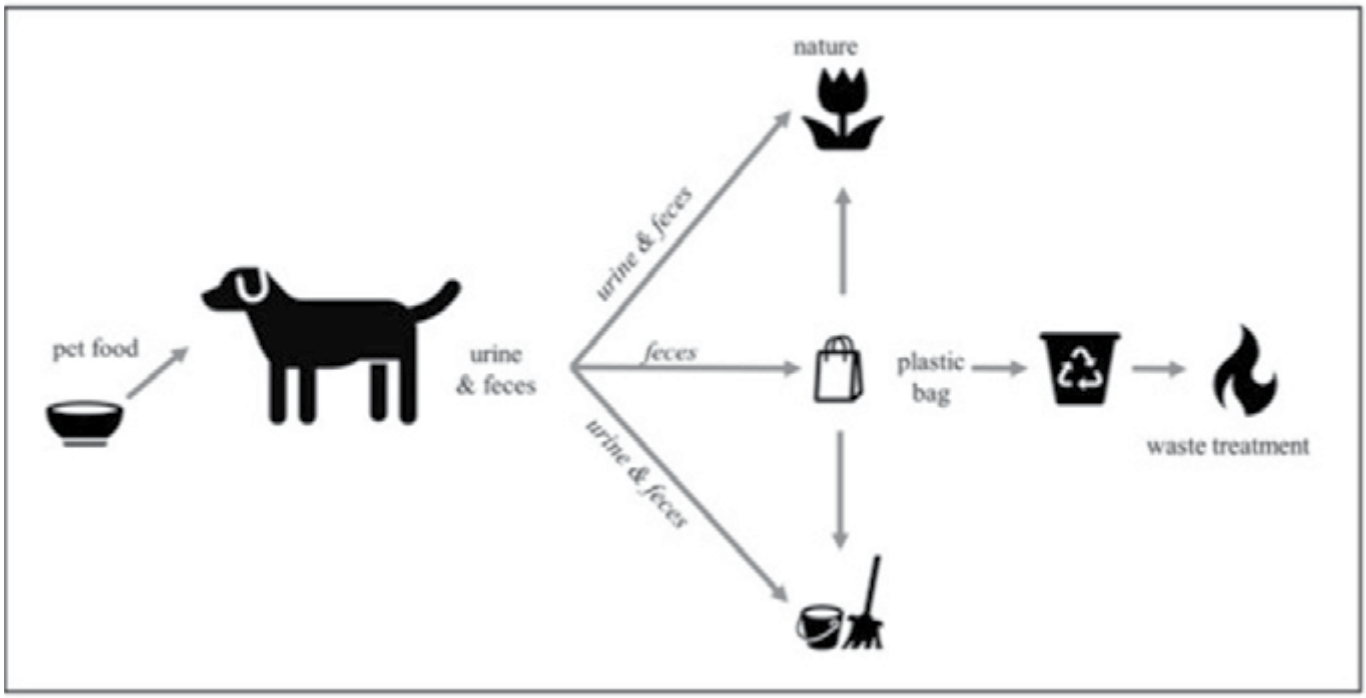


Fig. 2 Life Cycle Assessment of a Dog [10].

susceptible to climate change in one way or the other and temperature is likely to continue increasing in the future [7].

Climate Change is cyclical in nature, how do the keeping of small companion animals contribute to this phenomenon? Pet food is the main aspect related to the keeping of companion animals that has a direct link to Climate Change. Therefore, there is a need to reduce carbon emissions along the pet food value chain which starts from the sourcing of raw materials, manufacturing and transportation and concludes with waste disposal (Fig. 2) [8]. A reduction in the rate of dog and cat ownership in favor of other types of companion animals would contribute as well [9].

Additionally, the COVID19 pandemic which likely arose from a breakdown in a harmonious relationship with the environment resulted in an increase of take away services, home deliveries, sanitation protocols and individualized packaging which caused an increase in plastic use for wrapping and packages, gloves, and face shields. These oftentimes ended up in areas easily accessible to small companion animals and strays who were susceptible to the virus. Some found their way into the water ways exacerbating the marine plastics problem. Lessons learned from the pandemic will be essential to help us live and keep our companion animals harmoniously with the environment. The way we deal with climate change will define the adjustments and compromises we are willing to make for the greater good of the planet and how we bequeath it to future generations.

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APPROACH TO THE VOMITING CAT

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ABSTRACT BODY:

Vomiting is a common problem in cats. Comparatively, it seems like cats les frequently experience acute vomiting than dogs do, probably because of their more discriminating eating behavior. However, chronic vomiting is a common problem in cats. Occasional episodes of vomiting in an otherwise healthy-looking cat generally do not warrant a thorough work-up. However, if vomiting occurs more frequently or if other clinical signs are present, a more in-depth exam is recommended. While there is no uniformly agreed upon limit between “acceptable” and “pathologic”, many clinicians agree a diagnostic workup is recommended in cats vomiting more than once every 1-2 weeks. While hairballs are commonly observed as a cause of vomiting in long-haired cats and are not particularly concerning, frequent vomiting of hairballs in short-haired cats may also be an early sign of chronic intestinal disease.

In cats, vomiting can be associated with a wide variety of diseases, probably because of a high number of abdominal peripheral receptors that can stimulate the vomiting center in the brain stem. Moreover, the close association between the vomiting center and the chemoreceptor trigger zone located on the floor of the 4th ventricle plays an important role when cats experience metabolic disorders or intoxications.

History and physical exam

In cases requiring veterinary attention, a good history can be very helpful in discovering hints: how often does the cat vomit? what is the timing of the vomiting events in relationship to meals? how does the vomit look like? is the cat’s general health affected (e.g., hyporexia, anorexia)? These are all pertinent questions to better characterize the scope and severity of the problem and its syetmic repercussions. A detailed physical exam is performed to identify diseases responsible for vomiting, and assess the consequences of vomiting (dehydration, weight loss) and their severity. A thorough oral exam should always be performed, looking for a piece of string looping around the base of the tongue in cats that play with potential linear foreign bodies. Thorough palpation of the ventral neck on both sides of the trachea can help detect enlarged thyroid glands. Additionally, cardiac auscultation may reveal a gallop rhythm or other findings associated with thyrotoxicosis. Although exceptions may occur, most hyperthyroid cats are 5-7 years of age and older. However, absence of palpable masses or cardiac changes is not sufficient to rule out hyperthyroidism. Finally, the abdomen must be thoroughly palpated to detect possible pain, space occupying lesions, intussusception, abnormal intestinal loops, kidneys of abnormal size and shape, etc.

Differential diagnoses

Generally, it is useful to differentiate causes of vomiting located in the GI tract (e.g. obstruction, digestive diseases) from those occurring due to liver (e.g. cholangitis) or pancreatic disease (e.g., pancreatitis) or those due to systemic diseases (e.g. chronic kidney disease, endocrinopathies, etc.). Table 1 shows a list of differential diagnoses that should be considered for vomiting cats.

Diagnostic tests

Radiographs are always indicated in vomiting animals, as they are quite sensitive and specific for the diagnosis of intestinal obstructions. Radiologic signs of mechanical ileus include dilated small intestinal loops >12 mm in width (obstructive ileus) or accordion-like, clumped pattern of the small intestine (linear foreign body). If survey radiographs are not diagnostic in a cat suspected to have an obstructive lesion, radiographic contrast may be administered to allow better visualization. However, the risk of aspiration of the contrast agent should be carefully weighed against the possible benefits. Additionally, abdominal ultrasound can be very useful as a complement to radiographs: foreign bodies, intestinal masses, intussusceptions, abnormalities in wall layering and many more lesions can easily be detected by an experienced ultrasonographer.

In cats with access to the outdoors and concurrent diarrhea, a fecal parasitological exam should be done to rule out GI parasites, or alternately treatment with a broad-spectrum anthelminthic such as fenbendazole (50 mg/kg q24 for 3 days, the liquid formulation is easier to administer) can be started. Cats with chronic vomiting, hyporexia and weight loss may suffer from idiopathic inflammatory bowel disease (IBD). In mild cases, switching to an elimination (novel protein) diet or to a hydrolyzed peptide diet may result in resolution of the clinical signs. Low-grade intestinal T-cell intestinal lymphoma (LGITL) can be difficult

to differentiate from IBD without special staining of intestinal biopsies.

For cats with diseases likely originating outside the alimentary tract, a broad search must be initiated. Practically, a stepwise approach is meaningful. The first step consists of a minimal data base including CBC, chemistry panel and urinalysis, as well as retrovirus check and, if the cat is older than 5 years of age and the signs fit, a serum total thyroxin concentration. This initial screening may be followed by a more targeted approach focused on the abnormalities detected in the lab work. An abdominal ultrasound exam can be very helpful to confirm or rule out diseases affecting the liver, the kidneys, possibly the pancreas or other organs.

In some instances, results of all exams listed above are either normal or inconclusive. If diseases originating outside the GI tract can be excluded, and the GI tract is the most likely origin of the problem, gastro-duodeno-ileoscopy may be indicated to visualize the gastric, duodenal and ileal mucosa, and collect mucosal biopsies for histological analysis. Alternatively, exploratory abdominal surgery or laparoscopy could be useful to explore the stomach and the whole bowel as well as the abdominal cavity, and to sample full thickness biopsies from abnormal looking gastric or intestinal segments or from other organs (e.g., liver, pancreas).

Table 1: Differential diagnoses to consider in vomiting cats

Gastrointestinal (GI)	**Outside GI**
Stomach	**Abdominal**
- infectious (bacterial e.g. *Helicobacter*)	- peritonitis (e.g. FIP)
- obstructive (including bezoars)	- diseases of liver, gallbladder and bile ducts
- adverse reaction to food (food intolerance or food allergy)	- pancreatitis
- chronic gastritis (possibly with IBD)	- diaphragmatic hernia
- gastric (*Ollulanus*) or intestinal nematodes	- neoplasia
- gastric neoplasia	
- motility disorders	**Metabolic**
	- uremic syndrome (kidney diseases)
Small bowel	- endocrine disease (hyperthyroidism, DKA)
- infectious (viral, bacterial, parasitic)	- drugs (antibiotics, NSAIDs)
- obstructive (including linear foreign body, intussusception, eosinophilic sclerosing fibroplasia)	- toxins (toxic plants, etc.)
- adverse reaction to food (food intolerance or food allergy)	- sepsis
- IBD or feline low-grade intestinal T-cell lymphoma	- systemic mastocytosis
- other intestinal neoplasia	
- adynamic ileus (of different causes)	**Neurological**
	- motion sickness
Colon	- vestibular disorders
- severe constipation	- encephalitis
	- dysautonomia
	- increased intracranial pressure
	- psychogenic (excitation, fear, pain)
	Other
	dirofilariasis

Basic treatment guidelines

If an underlying disease can be identified, treatment specifically addressing this condition should be initiated. For instance, the International Renal Interest Society (IRIS) has established guidelines for the approach and management of cats with chronic kidney disease (www.iris-kidney.com). The treatment of IBD and LGITL often focuses on dietary changes, immune-suppressive doses of prednisolone with or without the addition of chlorambucil. Cholangitis may benefit from antibiotic treatment against enteric bacteria. In addition, anti-inflammatory or immune-suppressive doses of prednisolone or occasionally cyclosporine were

recommended to treat cats with chronic pancreatitis.

However, symptomatic and supportive treatments are often necessary in the sick vomiting cat. *Supportive treatment* consists essentially of IV fluid therapy, often with electrolyte replacement (e.g., potassium). *Symptomatic treatment* includes antiemetics (see table 2). If gastritis is suspected, gastric mucosal protection with omeprazole, a proton pump inhibitor that appears to be most efficacious at decreasing gastric acid production in cats (1 mg/kg PO q12h). H2-receptor antagonists such as famotidine are a second choice (1 mg/kg PO or SC q12h). Consider adding sucralfate 0.5 g per cat TID to QID if erosive or ulcerated gastritis is suspected (hematemesis). When the appetite returns, a bland or a hypoallergenic diet can be fed.

Table 2: List of antiemetic drugs that can be used in cats

Antiemetic drug	Target/antagonism	Dose	Other effects
Maropitant	NK1-receptors: substance P antagonist	1 mg/kg/day SC or slow IV	
Ondansetron	5-HT_3 receptors in the CRTZ and in the abdomen	0.5-1 mg/kg q12h IV, PO	
Dolasetron		0.6-1 mg/kg q12h IV, PO	
Chlorpromazine	α_2 receptors, dopamine-2 receptors, histamine-1 receptors, muscarinic-1 receptors	0.2-0.4 mg/kg q8h SC, IV	Hypotensive, sedative
Prochlorperazine		0.1-0.5 mg/kg q6-8h SC, IM	Hypotensive. sedative

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OTITIS ¿SON REALMENTE TAN DIFÍCILES?

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ABSTRACT BODY:

Otitis Externa

Es la inflamación del conducto auditivo externo, desde el meato al tímpano pero también envuelve el pabellón. Son el 20 %(1) de los casos caninos que se remiten a los hospitales, y el 6.6 % de los casos felinos.

Las secreciones combinadas de estos dos tipos de glándulas son llamadas cerumen o cera. El epitelio puede también contener folículos pilosos.

Factores Predisponentes

A. Aumento en la temperatura y humedad ambiental.
B. Pobre ventilación, relacionado a su forma anatómica (Conformación).
C. Los componentes de la flora normal del oído (*Staphylococcuss pseudintermedius, Streptococcus sp. y Malassezia sp.)* son potencialmente patógenos.
D. Enfermedades de piel subyacentes: Hipotiroidismo, enfermedades de la piel que produzcan prurito, Etc.
E. Otitis media, generalmente en sentido contrario.
F. Seborrea sea primaria o secundaria.
G. Oreja de nadador, sobre todo luego de nadar o de un baño.

Causas de Otitis Externa

- Cuerpos extraños, generalmente unilaterales y agudos.
- Puede ser una causa secundaria que contribuye a la severidad y persistencia de la enfermedad una vez que el canal externo se altera (Recuerden las bacterias que son habitantes normales del oído).
- Levaduras: *Malassezia pachidermatis* muy común y *Candida albicans* muy rara.
- Hongos: Saprofíticos oportunistas (*Aspergillus),* también *Rhizopus* y ocasionalmente *Mucor* y *Penicillium.* Los dermatofitos y las micosis sistémicas envuelven primariamente el pabellón.

E. Parásitos:

Ácaros

1. *Otodectes cynotis*: Tratar por 4 semanas (Ciclo de vida).
2. *Demodex canis.*
3. *Notoedres cati.*

Garrapatas

1. *Otobius* y *Dermacentor.*

F. Alérgicas:

1. Atópica. La causa más común de otitis alérgicas.
2. Alergia a alimentos.
3. Contacto, esencialmente a preparados tópicos con neomicina.
4. D. A. P.

G. Endocrinopatías, relacionados a cambios y/o defectos en la queratinización y secreción glandular.
H. Complejo seborreico.
I. Neoplasia: Adenomas de las glándulas ceruminosas y adenocarcinomas
J. Autoinmunes
   1. Penfigoide ampular.
   2. Pénfigo foliáceo, pénfigo vulgar.
   3. L. S. E.

Clasificación de la Otitis Externa

A. Localización: Unilateral o Bilateral.
B. Duración: Aguda, crónica o recurrente.
C. Causa: Primaria o secundaria.
D. Patrón de reacción: Eritematoso, proliferativo, osificado o ulcerativo.
E. Exudado Producido: Purulento o ceruminoso.

Diagnóstico de la Otitis Externa

A. Historia
1. Aparición.
2. Unilateral o bilateral.
3. Edad del animal.
4. Raza.
5. Tipo de pabellón.
6. Historia previa.
7. Ambiente.

B. Signos clínicos
1. Sacude la cabeza.
2. Dolor.
3. Inclinación de la cabeza.
4. Rascado.
5. Excoriaciones peri-auriculares.

6. Mal olor.

7. Descargas.

8. Otohematoma.

9. Cambios de conducta.

C. Examen del paciente

Examen físico general, otras condiciones que se manifiestan como una otitis.

2. Examen ótico, primero la menos afectada.

Pabellón: Excoriaciones, alopecia, excoriación, eritema.

Orificio auditivo externo: Exceso de pelo, cuerpos extraños.

Palpación de los cartílagos auriculares: Calcificados, pueden estar muy sensibles al dolor.

Examen otoscópico: Bajo anestesia general y se debe evaluar:

Condición y ancho del canal

Apariencia del detritus o descarga.

Olor del oído.

Condición del tímpano: Color, transparencia, bultos, perforaciones.

D. Citologías directas.

Apariencia visual de la descarga ótica.

Citología: Preparación y coloración de las laminillas, buscar bacterias o levaduras (Pequeñas cantidad es normal). Células neoplásicas.

Parásitos

E. Cultivo y Antibiograma.

De valor cuestionable.

1. Los discos contiene drogas a concentraciones séricas mas no a concentración tópica.

2. Generalmente no prueban antisépticos.

3. Caros?.

F. Biopsia: Si se sospecha de neoplasia.

G. Radiología: Si hay osificación o el oído interno está afectado.

Limpieza del oído:

Tranquilización o anestesia general.

Cortar el pelo del pabellón.

Arrancar el pelo del canal.

Remover cuerpos extraños.

Enjuagar el canal.

Limpiar el pabellón.

Secar el canal, usar un tubo de succión pequeño (Sondas) o hisopos (No es recomendable).

Curetaje del detritus remanente.

Repetir el enjuague y secado.

Re-examinar.

Medicación tópica adecuada según lo que se haya encontrado.

Deben Recordar:

Los perros con DA desarrollan Otitis Externa Alérgica y esta lleva a Otitis Media (2–5)

El prurito de los partes distales de las patas fue notado en un 76% y la Otitis externa en un 60% de los perros(2).

En 43% de los perros alérgicos con otitis crónica, los signos de otitis fueron lo primero que notaron los dueños antes que los otros signos de alergia(5).

Se pueden emplear GC tópicos para Tto sintomático. Incluso pueden ser de beneficio como prevención luego de la resolución de la infección y la inflamación aguda(6).

El tratamiento para la otitis bacteriana o por levaduras sigue los mismos principios que las infecciones de piel: Tópico para la Externa y Sistémica para la Media.

Identificación del origen/perpetuador

Control de la(s) infecciones

Restablecer la normalidad de la piel ótica

Evitar las recurrencias

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HOW I TREAT ANTIBIOTIC-RESISTANT BACTERIAL INFECTIONS

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ABSTRACT BODY:

Multidrug resistant bacterial infections continue to rise in prevalence in veterinary practice and result in increased costs of treatment, emotional distress for pet owners, and poor outcomes such as limb amputation, organ failure or death or euthanasia. Before treating a multidrug resistant bacterial infection, I ask the following questions:

Is this organism that was cultured likely to be the cause of an infection? (i.e., should I even treat this organism?)

Once a positive culture has been obtained, the veterinarian must consider the significance of the positive test result. Bacterial organisms detected within a specimen are often not the cause of an animal's clinical signs, and in many situations, multidrug resistant bacterial isolates do not require antibiotic treatment at all. Contamination is the most common cause of false positive cultures. Isolation of only one or two colonies of coagulase-negative staphylococci, *Bacillus* spp., *Corynebacterium* spp., and propionibacteria commonly suggest contamination. Isolation of large numbers of a single type of bacteria from a normally sterile site is generally clinically significant, especially when supported by cytologic examination of a stained smear that demonstrates the presence of bacteria within leukocytes. Open wounds are often contaminated with bacteria, but rarely do these go on to cause infection. Positive cultures from open wounds should only be considered significant if there is clinical evidence of infection (pus, marked erythema, fever, foul odor). In general, bacteria isolated from the urine of an animal without signs of urinary tract disease are not significant clinically and should not be treated. This is discussed in more details in the ISCAID guidelines for treatment of urinary tract diseases in dogs and cats (www.iscaid.org).

Are any of the drugs shown as "susceptible" the appropriate drugs for treatment of the bacterial species cultured?

Laboratories often (but not always) report results for specific antimicrobials on the basis of the organism being tested (e.g., cephalosporins are typically not reported for enterococci because of intrinsic resistance). Certain antimicrobials should generally be reserved for treatment of multiple-drug resistant organisms that cause life-threatening infections (e.g., vancomycin, linezolid, meropenem).

Assuming the drugs are active against the bacterial species isolated, are the drugs the right drugs for the patient in question?

Will they achieve adequate concentrations at the site of infection?

What route of administration is necessary and can the antimicrobials be administered by the route that is most appropriate for my patient?

Could adverse drug reactions occur in this patient with these antimicrobials?

Could drug interactions occur in this patient with these antimicrobials?

For infections in sites such as the CNS, the clinician needs to consider whether or not an antimicrobial to which the organism is reported as susceptible will penetrate that site. The clinician should also consider other factors, such as immunosuppression, pregnancy and other concurrent illness or drug therapy, when treating infections on the basis of antimicrobial susceptibility test results.

Is the antimicrobial drug currently being administered the most appropriate for the infection I am trying to treat?

Because antimicrobial susceptibility testing results are generally not available until 2-3 days after submission of a specimen for culture, in animals that are critically ill, antimicrobial therapy may already have been initiated by the time those results are available. The susceptibility results may show that the organism is resistant to a drug being used, in which case (if clinical improvement has not occurred) the drug might need to be changed to one that the organism is susceptible to. The susceptibility pattern can also aid in choosing an alternate drug when the patient does not tolerate the initial drug prescribed. Susceptibility testing may indicate that the organism is susceptible to a more narrow-spectrum (and generally less expensive) antimicrobial drug than the drug initially prescribed, in which case the treatment should be changed to minimize the development of antimicrobial resistance and adverse gastrointestinal effects.

Can I shorten the duration of therapy?

Reducing the duration of treatment to the minimum required for effective treatment is recommended to

minimize selection pressure on bacterial populations. This differs from previous recommendations to 'finish a course' of antimicrobial drugs. In general, surgical prophylaxis should involve administration of antimicrobials only in the immediate peri-operative period (immediately before and during surgery).

What is the underlying problem predisposing to infection?

Bacterial infections are rarely primary problems and usually there is an underlying predisposing disorder, such as chronic bronchitis (pneumonia), allergic skin disease (pyoderma), a foreign body, or a recessed vulva. Identification and management of the underlying problem to the best extent possible is critical to minimize recurrence of infection following antimicrobial drug therapy. In some cases, the underlying disorder cannot be effectively treated, and the clinician needs to decide to what extent treatment of secondary bacterial infections is necessary.

7. Are there non-antimicrobial alternatives to treating the problem?

A variety of non-antimicrobial alternatives exist for treatment of resistant bacterial infections. These include frequent bathing for canine superficial bacterial folliculitis, and live biotherapeutic products for urinary tract infections. Topical antimicrobial drugs overwhelm bacterial resistance mechanisms and may be useful for focal pyoderma or bacterial otitis externa.

HOW I TREAT CHRONIC FELINE UPPER RESPIRATORY INFECTIONS

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ABSTRACT BODY:

In this proceedings, updated information concerning management of chronic bacterial and viral pathogens is presented.

Bacterial causes. Almost all cats with chronic mucopurulent or purulent nasal discharge have a bacterial component to their disease. Diagnosis and treatment was reviewed by the International Society for Companion Animal Infectious Diseases. Primary bacterial disease is rare but may be associated with *Bordetella bronchiseptica*, *Mycoplasma* spp., *Chlamydia felis*, and some *Pasteurella* spp.. Recently it was shown that *Bartonella* spp. are not a causes of chronic rhinitis in cats. Most cases of chronic or recurrent bacterial rhinitis are secondary to other diseases including trauma, neoplasia, inflammation induced by viral infection, foreign bodies, inflammatory polyps, and tooth root abscess. Thus, if routine antibiotic therapy fails with doxycycline or amoxicillin, a diagnostic workup should be performed. If the diagnostic workup fails to find a primary disease and neutrophilic or mixed inflammation is noted, other antibiotics could be considered.Pradofloxacin has been evaluated as a treatment of feline rhinitis and conjunctivitis in several studies and can be considered as a rescue drug for cats with suspected bacterial disease.This fluoroquinolone is known to be safe for the use in cats.

Since bacterial rhinitis leads to chondritis and osteomyelitis, antibiotic therapy may need to be continued for weeks in cats with chronic disease. Drugs with an anaerobic spectrum that also penetrate bone and cartilage well are often effective. Clindamycin or amoxicillin-clavulanate are frequently used. Amoxicillin-clavulanate has the advantage of killing most *B. bronchiseptica* isolates. Clindamycin has the advantage of effective against *Mycoplasma* spp. and is effective against many anaerobes. After being administered twice daily on the first day, azithromycin can be administered every third day.

Cefovecin can be used in cats that are difficult to treat orally, but since it is a beta-lactam, there is no effect against *Mycoplasma* spp. Topical administration of antibiotics by drops or nebulization may be beneficial for some cats but controlled studies are generally lacking. Lessening stress and immune stimulants as discussed for viral disease may be of benefit.

Viral diseases. Herpesvirus 1 (rhinotracheitis; FHV-1) and calicivirus (FCV) are the most common viral causes of sneezing and nasal discharge in the cat. If oral ulcers are present, calicivirus is most likely. If corneal ulcers are present, herpesvirus 1 is most likely. FHV-1 has now also been associated with chronic stomatitis, facial dermatitis, and endogenous uveitis. Viral rhinitis with or without secondary bacterial infection can be recurrent. FHV-1 can be documented by direct fluorescent staining of conjunctival scrapings, virus isolation, or polymerase chain reaction. Since FHV-1 DNA can be amplified in conjunctival cells of approximately 25% of healthy cats, the positive predictive value of these tests in diseased cats is low. Quantitative PCR may ultimately prove to correlate to the presence or absence of disease but some cats with chronic FHV-1 infections do not have high values. Currently used PCR assays also detect vaccine strains of FHV-1. RT-PCR assays can be used to amplify the RNA of FCV. However, these assays have the same problems with predictive value as those to detect DNA of FHV-1.

Feline viral rhinitis with or without secondary bacterial infection can be recurrent. There are no consistently effective primary therapies. For FHV-1, lysine at 250-500 mg, PO, once or twice may be helpful in some cats lessening recurrent disease and has been shown to be safe but should be given as a dose, not fed with food and is not a treatment for active disease. Lysine has been shown to be ineffective for prevention of upper respiratory tract infections in shelter studies and so should not be used for this purpose.

Administration of human alpha 2b interferon at 50 U, PO, daily may help some cats with suspected chronic calicivirus or FHV-1 infection. This can now be formulated for practitioners by prescription at some pharmacies. However, it appears this product may be taken off the market in the USA. In Europe, feline interferon may been beneficial in the management of some cats. Intranasal administration of modified live, intranasal FHV-1 and FCV vaccines may lessen disease in some chronically infected cats. If there is a positive response to intranasal vaccination in a cat with chronic disease, I will use this form of immunotherapy up to 3 times per year.

Famciclovir is currently the orally administered drug of

choice for management of acute (and possibly chronic) FHV-1 infections in cats. The drug has been prescribed mostly at 40 or 90 mg/kg and is safe at up to 90 mg/kg, PO, q8hrs and so the dose should be increased if lower doses were used and the initial response is suboptimal and FHV-1 is still suspected. Administration of one dose of famciclovir (125 or 500 mg) on admission to an animal shelter was ineffective in lessening clinical signs of disease.

Topical cidofovir (product for humans) can be used for the treatment of FHV-1 conjunctivitis twice daily and was effective in a controlled research project. The drug is easier to administer (twice daily) than idoxuridine or other anti-FHV-1 ocular therapies and does not cause as much irritation. This drug is available in some compounding pharmacies (www.rxfixer.com). In a recent research study, raltegravir was effective for the management of FHV-1 associated clinical signs in a model.

Immune modulation with a the probiotic *Enterococcus faecium* strain SF68 (FortiFlora®, Purina Pet Care) was effective in lessening stress reactivated FHV-1 signs in a model. Field studies with this probiotic are in progress. Recently, the use of an intranasal product containing 2 Toll-like receptor agonists was beneficial in lessening signs and shedding of FHV-1 in a model. Field studies with this compound are in progress.

Many of the cats with chronic recurrent signs of upper respiratory disease are likely to be infected by FHV-1 or FCV. Stress reactivation of feline viral infections is thought to be common, in particular for FHV-1. All the principles of stress relief for management of feline interstitial cystitis also apply to cats with recurrent signs of URI. In a recent study, use of a facial pheromone diffusor could lessen recurrent signs of FHV-1 in a mild stress model in experimentally inoculated cats. We have also recently shown that *Bifidobacterium longum* 999 (Calming Care; Purina) lessens stress and signs of FHV-1 infection in cats.

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USING THE WSAVA DENTAL GUIDELINES TO IMPROVE PATIENT CARE

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ABSTRACT BODY:

Why is marketing the dental department important?

1. Oral disease is by far the most common problem in veterinary medicine and there are generally only subtle to no clinical signs. However, patients afflicted with dental disease are quite often painful despite the lack of clinical signs. In addition, these disease processes cause significant localized and systemic medical problems. Ignorance abounds regarding dentistry both in the general public as well as in the veterinary field. This results in most patients being under treated. Therefore proper dental therapy is financially rewarding and good medicine.

2. Over the last decade or so, there has occurred a significant loss of traditional revenue streams due to many factors. Vaccine revenue has been markedly reduced by new studies. In addition, flea and heartworm prevention as well as other prescription revenue has been lost due to online prescriptions. Finally, increased reliance on the internet or other information decreases the client trips to the clinic

How to Increase Dental Revenue

Dental revenue can be improved in four distinct ways. However, they do not stand alone; all of them should be included in the marketing plan. In fact they are synergistic, by increasing more than one, they positively affect each other, further improving gains.

- The first and most cost effective way to attain this goal is to **increase the number of dental prophylaxis** procedures performed.
- **Client education:** This is best performed by enlightening the population about dental disease. This should come not only from the veterinarian, but the entire staff. This includes technicians and possibly most importantly, receptionists. By educating the veterinary staff, you educate the clients and provide more dentistry. This ideally is in person, but if time is an issue, handouts or qualified websites can be effective as well. There are can be in person, or via handouts and/or websites. Client educational videos are available at www.dogbeachdentistry.com
- **Superior, new equipment:** Once the marketing plan is underway and the days are full, superior equipment will speed procedures. A new drill, ultrasonic scaler, elevator, or curette can markedly cut down on surgical time and increase the number of procedures performed a day. If a practice can do one more procedure a day 5 days a week at an average of say $400 it will pay off $8,000 worth of equipment in a month. Moreover, this will result in shorted anesthetics, which is better for the patient.
- **Continuing education/training:** By learning better techniques veterinarians and technicians can speed the dental procedures benefiting the practice and the staff. The staff can be more efficient, which will also allow for the possibility of additional procedures. Furthermore, this efficiency will decrease operator stain and stress. Finally, proper performance of dental procedures should result in less surgical trauma and superior patient care. Ask your AHI rep if a lab is scheduled in your area, or visit www.vetdentaltraining for a San Diego class schedule.
- The next way to increase income is by **increasing the per dental procedure charge.** Increase the number of treatment options for the clients. This does not mean doing things like root canals, jaw fracture repair and major oral surgery since what most DVM's charge for these it is not efficient time usage. By spending that time doing office calls the practitioner will increase income with less stress. A more efficient way to do this is by offering superior "basic" care. This should include: dental radiology, root planing/doxirobe/clindoral, oravet, nerve blocks, proper pain management, bonded sealants, and fluoride therapy. All of these will greatly increase income without a significant investment of time or money. Practitioners, who have mastered the basics, can consider proceeding to composite restorations and periodontal flap surgeries, which are taught at the level 2 course in San Diego (See above).
- Clinics can markedly improve their dental and income by **improving their pre-operative testing protocol**. Furthermore, perform the pre-operative testing the day the cleaning is recommended, this will help lock clients into the procedure.

A. Complete blood panel (renal, hepatic, CBC, T4)

B. Urinalysis

C. Chest radiographs

HCM is often not ausculted

Over 50% of patients over 6 have significant findings on chest films

- Provide superior (and necessary!) **post-operative treatment**
- Pain management: Opiates, NSAIDS, Local Anesthetics (nerve blocks)
- Maxiguard, Oravet, homecare kits
- Rechecks

UVEITIS IN DOGS AND IN CATS

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UVEITIS

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Definition

Uveitis is the inflammation of any uveal component that occurs because of a breakdown of the blood aqueous barrier. It is a common process as the uvea is a very vascularised part of the globe, because of its immunosensitivity and its proximity to other structures. It is important to try to ascertain the cause of the uveitis as severe sequelae and blindness can occur.

There are different types depending on the affected parts:

- Anterior: inflammation of the iris and the ciliary body
- Posterior: inflammation of the choroid
- Panuveitis: inflammation of all the above
- Panophthalmitis: inflammation of all the globe tunics
- Endophthalmitis: inflammation of all ocular and adjacent structures

And two types depending on the duration:

- Acute: less than 4 weeks duration. In the acute form we see more elevated protein values, polymorphonuclear cells predominate and it is more painful than the chronic one
- Chronic: more than 4 weeks duration and mononuclear cells predominate

The clinical signs are photophobia, blepharospasm, conjunctival hyperaemia, corneal oedema, aqueous flare, hypotension, fibrin, hypopyon, hyphaema, change in iris colouration, sinequia formation and vision loss. Other clinical signs are here explained in more detail:

- Ciliary flush: deep blood vessels that can be seen into clear cornea but do not cross the limbus, do not blanch with phenylephrine, and do not move to the contrary of superficial conjunctival blood vessels.
- Corneal oedema: loss of corneal transparency by imbibition of fluid by the corneal epithelium or stroma caused by an increase of the endothelial permeability and reduction of the Na/K ATPase pump site density. It is caused by the presence of toxins, inflammatory mediators and cellular precipitates that damage the endothelium. Severe oedema may result in bulla formation.
- Miosis caused by the action of the prostaglandins causing ciliary body spasm. It is recommended to examine in a dark room with distant direct ophthalmoscopy. Remember that the mydriasis post tropicamide is slow in eyes with uveitis.
- Ocular hypotension is one of the first and subtle changes. An intraocular pressure less than 10 mmHg (combined with other ocular signs) is suggestive of uveitis. A difference of more than 5 mmHg in between eyes should also be taken into consideration.
- Aqueous flare occurs by disruption of the blood aqueous barrier permitting the entrance of proteins and cells. It is pathognomonic of uveitis. The degree of aqueous flare is classified from 1+ (mild) to 4+ (intense).
- Keratic precipitates are accumulations of inflammatory cells, fibrin and/or iris pigment that can be found on the corneal endothelium. They are usually found on the ventromedial cornea; try to move the head of the animal down. They also are pathognomonic of uveitis.
- Vitreal opacity can cause vision loss

Causes

It can occur independently of disease in other ocular structures or be caused by primary ocular disease such as lens, cornea, or sclera. It can also be secondary to neoplastic, infectious, or immune-mediated disease.

Making it simpler:

- Endogenous cause: it is originated in the eye, or it gets to the eye haematogenously or by extension from other structures: infectious, neoplastic, toxic, metabolic, autoinmmune
- Exogenous cause: trauma, radiation, chemical.

A list of causes will be presented at the lecture.

Idiopathic uveitis continues to be the more common cause and it is a diagnosis of exclusion.

Diagnosis

We have to start performing a full physical and ophthalmological examination, bloods for full biochemistry and haematology, urianalysis and

serology of infectious diseases, chest radiographs (i.e. for evidence of metastasis or fungal disease), abdominal ultrasound +/- fine neede aspirates, aqueocentesis and/or vitreocentesis.

Aqueocentesis is valid in the diagnosis of lymphoma in dogs and cats.

Treatment

We need to be aware of the potential severity of the sequelae of uveitis therefore treatment needs to be started as soon as possible. If the uveitis is severe or the cause is systemic, systemic therapy will be needed. Our first choice is corticosteroids.

Topical treatment

- Corticosteroid:
  - drug of choice is acetate of prednisolone 1%. It is more potent than dexamethasone phosphate in stabilising the blood aqueous barrier.
  - Initially start at 4-6 times daily
  - Topical non-steroidal antinflammatory (NSAID) and corticosteroid can be given together
  - It can be used in diabetic animals but beware of potential systemic absorption.
  - Gradual reduction of the frequency of treatment is advised
  - Be aware in cats of a potential recrudescence of FHV-1
- NSAID: ketorolac, bromfenac, nepafenac
  - Advised to use when there is presence of a corneal ulcer
  - Start 4 times daily
  - Increasing its frequency of administration will not increase the anti-inflammatory action
  - Recommend decreasing frequency gradually
  - Systemic absorption has been documented
- Cyclopegic: atropine (parasympatholytic). Achieves mydriasis diminishing contact of iris and lens and therefore preventing posterior synechia. It also diminishes pupil occlusion that could cause decreased vision.

What cyclopegia achieves is paralysation of the iris and ciliary body musculature. Atropine also stabilises the blood aqueous barrier blocking the acetylcholine effect. It is contraindicated when the intraocular pressure is elevated except for iris bombe in which case, we can also combine it with phenylephrine.

It can be given once or twice daily but its frequency can be increased to 4 times daily and reduce it according to response.

Note that atropine has a sour taste, and most animals will drool profusely after its application. The effect and duration of atropine will last less in inflamed eyes.

Note as well that atropine can cause a decrease in tear production, can cause tachycardia, decrease in intestinal motility, and can precipitate acute glaucoma.

The alternative to atropine is cyclopentolate, it is not sour, and it is tolerated better by cats. Remember that tropicamide has hardly got any cyclopegic effect, but it is a mydriatic. This points out that fact that mydriasis does not mean cyclopegia.

Systemic treatment

- Corticosteroid: we should wait to start systemic treatment with corticoisteroids until all tests have been completed. Treatment with prednisolone when it is recommended is started on 1-2 mg/kg/day in divided doses. We can use them systemically when there is a corneal ulcer, and it can be combined with topical NSAID.
- NSAIDS: in less severe cases we can give systemic NSAIDS but never together with corticosteroids. We should try to avoid its use when there is hyphaema and bleeding.
- Antibiotic: only used when it is directed towards a specific systemic disease however we also give them prophylactically in corneal perforation and intraocular surgery (surgeon dependent). Its ocular penetration improves with the breakdown of the blood aqueous barrier.

Sequelae

- Sinequia formation: it can be anterior or posterior
  - Anterior: adhesions to cornea consequence of the narrowing of the anterior chamber owing to iris thickening and/or inflammatory exudates within the iridocorneal angle.
  - Posterior: adhesions to the lens anterior capsule because of the lens shape. When there is miosis the iris is more in contact with the anterior lens capsule and adhesions can form. It can cause pupillary occlusion and iris bombe with the consequent glaucoma. We can sometimes detect pigment on the anterior lens capsule from this contact and this indicates a previous episode of inflammation. It is important to differentiate this from persistent pupillary membranes.
- Secondary glaucoma is a common complication. The causes are obstruction of the iridocorneal angle by inflammatory debri, iris bombe (it is less common in cats), extense anterior sinequia and preiridal fibrovascular membrane formation.
- Phthisis bulbi is an end stage eye consequence of chronic uveitis, in which fibrosis or atrophy of

the ciliary body (or both) occur and contributes to decrease the secreting function. When there is marked atrophy phthisis bulbi occurs.

- Cataract formation is a common sequela. It is thought to originate from inflammatory mediators within the aqueous humour. The formation of posterior synechia can also result in cataract formation.
- Lens luxation is common in cats, and it is normally associated with the presence of cataracts. These luxated lenses can be attempted to be surgically removed.

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NUTRICIÓN DEL PACIENTE CON CÁNCER:DEBO CAMBIAR LA DIETA?

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ABSTRACT BODY:

Evaluación nutricional

La evaluación nutricional (1) es clave para dar las mejores recomendaciones dietéticas a nuestros pacientes con cáncer; estas recomendaciones incluyen dieta (+/- suplementos y premios), ración diaria, y pauta de alimentación. En el examen físico es importante notar la puntuación de condición corporal (PCC) y muscular (PCM). Se ha sugerido que la obesidad puede predisponer a cáncer de mama en perras (2) y también que ciertos tipos de pacientes oncológicos están predispuestos a la delgadez (3). La PCM se puede ver reducida por sarcopenia (proceso fisiológico asociado al envejecimiento) y caquexia (proceso patológico mediado por inflamación, común en ciertas enfermedades crónicas).

La evaluación debe incluir una historia dietética detallada, para averiguar el/los alimento/s que recibe el paciente, incluyendo suplementos. Tras un diagnostico de cáncer, se ha descrito que cierto porcentaje de los propietarios pueden cambiar el enfoque nutricional para su perro (4). Mas aun, una encuesta identifico que los propietarios de perros con cáncer daban alimentos no convencionales (como alimentos caseros o en base a carne cruda) y suplementos con mas frecuencia que los de perros saludables(5). Ambas practicas (alimentación poco convencional y suplementación) se consideran un factor de riesgo nutricional.

Papel de la dieta en la terapia del paciente oncológico

Pese a la multitud de consejos que se pueden encontrar al respecto, hay muy poca evidencia que ciertas dietas +/- la adición de suplementos, de forma general, reduzcan la progresión del cáncer o la posibilidad de recaídas.

Energía

En pacientes con sobrepeso, se debe considerar si un plan de perdida de peso es deseable, lo que dependerá del pronóstico del paciente y de como el sobrepeso está afectando a su calidad de vida. Como mínimo, el manejo nutricional debe asegurar que el peso no sigue aumentando.

En pacientes delgados, ciertas neoplasias se asocian a pérdida de peso (3), lo que puede indicar un balance energético negativo +/- caquexia. En estos casos, el uso de alimentos de alta densidad calórica es recomendable, además de un seguimiento frecuente del peso para ajustar la ración.

Proteína

Es importante notar que la caquexia (mediada por la enfermedad) puede suceder con dietas adecuadas o altas en proteína. En todos los casos, es importante que el alimento aporte proteína de alta calidad (digestible, con un buen perfil de aminoácidos) y que esta no se reduzca en la dieta salvo que sea necesario (por ejemplo, enfermedad renal crónica concomitante) para asegurar un aporte adecuado y mantener la palatabilidad de la dieta.

Grasa y ácidos grasos

Dietas altas en grasa, y altas en calorías, son útiles en pacientes delgados y de apetito caprichoso. Sin embargo, existen ciertas contraindicaciones, como la presencia de hiperlipidemia o pancreatitis (en perros).

Los ácidos grasos omega 3, específicamente EPA y DHA, son de interés. En perros con enfermedad cardiaca, se ha visto que su suplementación (115 mg EPA + DHA/kg$^{0.75}$, (6)) puede ayudar a reducir la perdida de masa muscular y reduce la concentración sanguínea de citoquinas proinflamatorias (7). Es posible que este efecto beneficioso también se vea en caquexia asociada con cáncer, aunque faltan estudios. Un estudio noto un aumento de la supervivencia en perros con linfoma alimentados con una dieta rica en EPA y DHA y arginina vs control, aunque el estudio tenia un numero de pacientes muy bajo y no se vieron efectos sobre la calidad de vida o gasto energético (8).

Carbohidratos

Existe la teoría de que el fuel preferido de las células cancerígenas es glucosa, la cual se fermenta a lactato. De ahí la idea de que alimentar dietas bajas en carbohidratos puede ayudar a enlentecer el crecimiento de las células cancerígenas. Sin embargo, la glucosa en sangre es un parámetro altamente controlado y alimentar dietas bajas en glucosa no tiene por qué afectar a la glucemia. No existe ningún estudio que demuestre un efecto positivo de dar un alimento bajo en carbohidratos prolongue la supervivencia o mejore la calidad de vida de perros o gatos con diferentes tipos de neoplasia. El uso de dietas bajas en carbohidratos tiene sus riesgos, al ser estas dietas suelen ser muy altas en grasa y en ciertos pacientes pueden estar

contraindicadas.

Antioxidantes

El tema de suplementar la dieta con antioxidantes en pacientes con neoplasia es controvertido. En parte se debe a que existen muchos tipos de antioxidantes y muchas dosis posibles, y no existe acuerdo. Además, su efecto puede ser distinto en diferentes tipos de cáncer. Respecto a la prevención, existe un estudio en veterinaria, retrospectivo (9), que sugiere que el riesgo de carcinoma de células transicionales en Yorkshire terrier se reduce si se añaden verduras a su dieta, y la teoría es que los micronutrientes y antioxidantes de estas verduras podrían ayudar a la prevención de neoplasia. El estudio tiene muchas limitaciones (incluyendo el bajo número de casos, la falta de control nutricional, el uso de preguntas a propietarios para recordar la dieta administrada hace años y la falta de control de otros parámetros) que dificulta sacar conclusiones.

Respecto al tratamiento, su uso es aún más controvertido. Aunque pueden tener efectos positivos, ciertos estudios en humana han mostrado que mega dosis pueden ser contraindicadas y en ciertos tratamientos (como radioterapia) ciertos centros piden que los pacientes no reciban suplementos con antioxidantes, con la idea de que pueden proteger a las células cancerígenas del tratamiento.

De momento no existen recomendaciones para pacientes oncológicos veterinarios, con lo que la recomendación de dar una dieta completa que ya incluya antioxidantes (sin suplementarlos adicionalmente) es una recomendación razonablemente prudente en este momento.

Cambio de dieta: ¿si o no?

Si la evaluación nutricional no identifica factores de riesgo, no es necesario cambiar de dieta. Esto esta indicado si se identifican enfermedades concomitantes que lo requieran, o para apoyar el/los órganos afectados (por ejemplo, dieta altamente digestible en cáncer intestinal) o para manejar los efectos secundarios de la terapia. En pacientes con reducción del apetito, donde el uso de otras terapias como estimulantes del apetito ha fallado, se debe considerar una sonda de alimentación hasta que se haya identificado y tratado la causa de la inapetencia.

En caso de que se desee una dieta casera, es recomendable contactar con un especialista en nutrición veterinaria para que sea formulada de forma adecuada. El uso de recetas de libros o internet no es recomendable (10). El uso de alimentos en base a carne cruda tiene riesgos asociados, incluyendo riesgos microbiológicos, y la autora no los recomienda.

Los suplementos se deben considerar de forma individualizada y su uso solo se debe instaurar si el análisis riesgo-beneficio lo indica (por ejemplo, suplementos de EPA y DHA).

Lo más importante es aportar una dieta completa y equilibrada (comercial o casera) según las especie y etapa de vida, palatable para el paciente, que incluya las modificaciones necesarias según el área afectada y la presencia de enfermedades concomitantes. Mas aun, este/os alimento/s deben aportarse en cantidades suficientes para mantener un peso estable, con una PCC ideal.

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NUTRICIÓN DEL PACIENTE CON CÁNCER:DEBO CAMBIAR LA DIETA?

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ABSTRACT BODY:

Un preocupación frecuente tras el diagnóstico de cáncer en animales de compañía es si la dieta ha podido estar relacionada con el desarrollo de la enfermedad o si un cambio en la misma puede ayudar a controlar mejor el cáncer. En nuestra clínica, una gran parte de los propietarios nos pregunta por temas relacionados con la dieta, y se han publicado resultados similares en estudios (1) que muestran que la mitad de los propietarios muestra preocupación por las dietas comerciales y una cuarta parte de los propietarios cambia la dieta de sus animales tras el diagnóstico de cáncer, en general hacia una dieta cocinada en casa.

Resultados similares de un estudio con mayor número de casos (2) mostró que los propietarios de perros con cáncer dedican más tiempo a buscar información sobre dietas o suplementos, y son más propensos a cambiar a una dieta cocinada o cruda, y al uso de suplementos que incluyen aceite de cannabis, hongos o cúrcuma.

Sin embargo, un análisis (3) de las dietas comúnmente usadas para alimentar a los perros con cáncer mostró que son inadecuadas de acuerdo a los estándares de nutrición recomendados. En ambos estudios se vio que los propietarios generalmente buscan información en internet pero solo una minoría consulta con el veterinario sobre este tema por lo que es importante que como clínicos ofrezcamos información de forma proactiva o guiemos a los propietarios a fuentes de información fiables.

En los últimos años parece haber un mayor interés por el uso de dietas de comida cruda para alimentar a los perros, tanto sanos como con cáncer. Estudios nutricionales (4) sobre estas dietas han demostrado que son deficientes y/o no equilibradas para elementos como calcio, fósforo algunas vitaminas o taurina y que además tienen mayor riesgo de contaminación bacteriana. Por otra parte, se ha encontrado consistentemente infecciones por *Salmonella*, enterobacterias, *Listeria, E. coli, Brucella* o *Toxoplasma.* Un estudio reciente (5) demostró que las bacterias detectadas en la comida cruda podían identificarse después en las heces de los animales alimentados con estas dietas, lo que supone también un riesgo de diseminación de las infecciones. Algunas de estas bacterias eran resistentes a múltiples antibióticos. La presencia de infección en la comida cruda puede ser peligrosa no solo para el perro que la come sino que algunas de las infecciones se consideran zoonosis, con el potencial riesgo para sus propietarios (6). Incluso cuando la carne cruda no es la dieta principal sin que son premios ocasionales que se le dan al perro, hay riesgo de que produzcan enfermedades severas, como se demostró con la relación entre los premios de carne cruda de pollo, infección por *Campylobacter* y desarrollo de poliradiculoneuritis (7).

La mayoría de las dietas caseras son ricas en grasa y bajas en carbohidratos. La idea se basa en el “efecto Warburg”, que describe que las células tumorales necesitan una mayor cantidad de glucosa debido a que la aprovechan de forma deficiente (obteniendo solo 2 moléculas de ATP por cada molécula de glucosa). Este efecto se ha usado en diagnóstico por imagen con el uso de la tomografía de emisión de positrones (PET por sus siglas en inglés) que usa un radiofármaco unido a la glucosa. Esta glucosa se acumula en las zonas de tumor y el radiofármaco permite visualizar estas zonas en la tomografía.

Tras el conocimiento de esta particularidad metabólica de los tumores, enseguida se postuló que reducir la glucosa (principalmente mediante la reducción de carbohidratos y dietas donde la grasa supone el 90% de la energía ingerida) podría ayudar a controlar el tumor. En personas, diferentes ensayos han mostrado que el uso de estas dietas es seguro y que puede hacerse de forma adyuvante a tratamientos médicos pero no hay evidencia clínica de que puedan curar el cáncer (8).

Más allá del cuestionable efecto de la dieta en el tratamiento del cáncer, no hay duda de la importancia de la nutrición en el paciente oncológico, como una herramienta más para mantener una buena calidad de vida durante el tratamiento. Los diferentes tumores, especialmente aquellos que afectan al tracto digestivo o a la cavidad oral, pueden afectar a la capacidad para comer o digerir la comida y llevar a un estado de malnutrición.

Se han publicado recomendaciones para la alimentación de pacientes oncológicos caninos y felinos (9). Las recomendaciones se tienen que adaptar a cada paciente en particular: en animales bien nutridos

con condición corporal óptima no necesitan cambios y deben evitarse comidas peligrosas (como las dietas crudas). No hay datos que demuestren que las dietas comerciales supongan un mayor riesgo de desarrollar cáncer en animales de compañía. De hecho, algún estudio mostraba que dietas caseras se asociaban al desarrollo de tumores mamarios en perras (10).

Más allá de cambios en la dieta, los propietarios de perros y gatos con cáncer con frecuencia usan suplementos alimenticios (2) como aceite de CBD, cúrcuma o derivados de hongos y otros muchos. La información sobre la eficacia o seguridad de estos productos es muy escasa y no hay datos de si pueden tener alguna interacción con las terapias antitumorales convencionales.

Es importante integrar en la consulta oncológica un asesoramiento nutricional que incluya las necesidades diarias de energía, el control del peso y condición corporal y es recomendable, en aquellos pacientes que decidan cambiar la dieta, que recurran a especialistas certificados en nutrición animal y no a fuentes poco fiables encontradas en internet.

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SHUNT PORTOSISTÉMICO

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ABSTRACT BODY:

SHUNTS PORTOSISTÉMICOS

Los Shunts portosistémicos SPS, consisten en comunicar venas subsidiarias del sistema digestivo, estómago, intestino, páncreas o bazo directamente a la cava sin pasar por el hígado y no llegar al sistema porta.

La corrección de esta anomalía aportaba un gran caudal venoso al sistema porta, llegando a un hígado no habituado a tal cantidad de sangre que derivaba en una hipertensión portal y en un desenlace fatal.

La aparición de sistemas graduales de cierre del shunt como el ameroide o el celofán permitieron ya en este siglo XXI abordar quirúrgicamente con éxito estos procesos, apoyados por la exactitud de los métodos de diagnóstico.

La sintomatología del shunt dependerá del % que se salte los sistemas de purificación hepáticos encontrando en la circulación general amonio, ácidos grasos de cadena corta, aminoácidos aromáticos , ácido gamma amino butírico, GABA, benzodiacepinas endógenas, glutamato, falsos neurotransmisores, produciendo encefalopatía hepática con cambios de comportamiento, ceguera, ataxia y convulsiones.[1]

Los shunts pueden ser extrahepáticos (cuando se sitúan fuera del parénquima hepático) o intrahepáticos (cuando están localizados dentro del hígado) así como congénitos o adquiridos.

Los shunts congénitos más habituales son los extrahepáticos, siendo los vasos más habituales que terminan en la cava, la gástrica izquierda y la gastroesplénica, aunque cualquier rama subsidiaria de la porta puede acabar en la circulación general, siendo el shunt de la frénica un desafío en su tratamiento quirúrgico.

Hay una variedad de shunt extrahepáticos que no abocan en la cava, sino que acaban en la ácigos también de naturaleza congénita.

Shunts intrahepáticos son los que comunican las ramas de la porta (árbol derecho o izquierdo con la cava ya dentro del parénquima hepático, también son de naturaleza congénita y el tratamiento actual es gracias a la radiología intervencionista y no trataremos en esta charla.

Por último nos quedan los shunts adquiridos, cuando se produce una dificultad de paso de la sangre por el hígado, por una fibrosis hepatoportal o a displasia microvascular hepática, una cirrosis u otro proceso hepático, el organismo crea conexiones que alivien la hipertensión portal, habitualmente entre los vasos renales y la circulación general, incluso el cierre correcto de shunts congénitos pueden inducir a esta creación de shunts adquiridos que únicamente podrán ser tratados de manera medicamentosa.

Los shunts congénitos extrahepáticos suelen afectar a perros de raza pequeña como el Yorkshire Terrier, Schnauzer miniatura, Maltés y Carlino, dejando los intrahepáticos para razas grandes como Labrador Retriever y del Irish Wolfhound. [2]

SINTOMATOLOGÍA

Por un lado, derivada de sustancias que han pasado sin depurarse en el hígado y que alteran el sistema nervioso central, por otro lado el hígado también necesita desarrollarse con las sustancias que se escapan a la circulación, al no recibirse, el hígado no se desarrolla y no cumple sus funciones (disminución de la producción de proteínas, factores de coagulación, detoxificación,…)

El hígado recibe el 70% de su vascularización a través del sistema porta, cuando hay un shunt, es más fácil que circule la sangre por el shunt ya que atravesar el hígado frenará la circulación, dependiendo del % que se escapa a través del shunt, este es el motivo por el que en animales de edad avanzada puede tardarse en diagnosticar el shunt por escasos síntomas, el Schanuzer miniatura de más de siete años está sobrerepresentado.[3]

Los principales signos clínicos que veremos en un animal con shunt portosistémico serán:

- Neurológicos: letargia, depresión, alteraciones del comportamiento, desorientación, encefalopatía hepática evidente (ceguera, head pressing, convulsiones y coma).

- Gastrointestinales: anorexia, vómito, polifagia, ascitis, diarrea

- Urinarios: hematuria, disuria, estranguria y obstrucción uretral debidas a la aparición de cálculos de urato amónico (patognomónico de shunt portosistémico excepto en Dálmatas), poliuria y polidipsia.

Coloración cobriza del iris en gatos, pobre tasa de crecimiento, pérdida de peso, pérdida de pelo, intolerancia a fármacos (especialmente sedantes y anestésicos).

Generalmente veremos afectados animales jóvenes (< de un año de edad), aunque se han diagnosticado animales de edades más avanzadas.

Ácidos biliares pre y postpandriales aumentados es una prueba patognomónica

DIAGNÓSTICO

El diagnóstico se realizará per la sintomatología clínica, las pruebas de laboratorio y el diagnóstico por imagen. La ecografía es una buena técnica de diagnóstico nos determinará la microhepatopatía y permite visualizar tanto los intra como los extrahepáticos pero es necesario experiencia.

Preferimos el TAC, nos localizará mejor el shunt y dará más datos del desarrollo del árbol portal, aunque al tratarse de un procedimiento intermitente puede darnos algún fallo en ese diagnóstico.

La portografía tiene diferentes utilidades, al cerrar el shunt nos determinará el verdadero desarrollo del árbol portal, nos ayuda en la localización si no disponemos de TAC, y en shunts difíciles de localizar nos asegura su perfecta localización.

La gammagrafía portal es otra posibilidad

TRATAMIENTO

El tratamiento del shunt portosistémico puede ser médico o quirúrgico, pero está demostrado que hay más supervivencia en animales en los que se realiza cirugía que en los que exclusivamente se realiza tratamiento médico. [4]

TRATAMIENTO MÉDICO

Será para estabilizar al paciente o en animales que el cierre provoque hipertensión portal y consistirá en dieta altamente digestible, antibióticos, lactulosa y glucosa oral.

TRATAMIENTO QUIRÚRGICO

La oclusión parcial del shunt ha reducido las complicaciones perioperatorias y nos ha permitido realizar la cirugía en clínicas con mínima infraestructura.

A pesar de ser la técnica a elegir pudiendo utilizar el celofán o el ameroide, se puede optar al cierre completo en la misma cirugía en algunos casos, si tras el cierre no se experimenta un aumento de la presión venosa portal de <10 mmHg (13,6 cm H2O) y una presión venosa portal total postoclusión de <16 mm Hg (21,7 cm H2O). [5] Otros signos como congestión del intestino o cambio en el color del páncreas también nos pueden indicar que no debemos hacer el cierre total.

Lo más habitual es buscar un cierre progresivo del shunt con ameroides o bandas de celofán aunque también pueden producir el cierre incompleto de dicho shunt por lo que la remisión de la sintomatología puede no ser completa (especialmente con las bandas de celofán).[6]Aunque la calidad de vida de los animales sea similar a pesar del método empleado.[7]

Gracias al TAC tenemos la perfecta localización del shunt, un gran porcentaje son extrahepáticos y entran a la cava por la izquierda.

Tras la laparotomía media craneal retiramos el duodeno descendente de la derecha a la izquierda y examinar la vena cava caudal cuidadosamente, los únicos vasos que deben entrar en la vena cava caudal entre las venas hepáticas y las renales son las pequeñas venas frénico-abdominales.

Cualquier vaso en esa localización será un shunt, es probable que debamos desplazarnos muy craneal hasta el foramen epiplóico, una vez localizado procederemos a una mínima disección y a la colocación del ameroide o del celofán

El ameroide absorbe agua, efecto higroscópico, a lo que se añade interacciones interproteicas mediadas por calcio [5] y la fibrosis que se desarrolla alrededor del vaso, normalmente colocamos un ameroide de 3,5 ó 5 mm ó 7,5 mm.

La colocación de un ameroide demasiado grande o la realización de una disección demasiado amplia pueden inducir colapsos del vaso, ya sea por peso o movimiento respectivamente. Esto provocará un cierre prematuro induciendo un aumento agudo de la presión portal. Se recomienda que la reducción inicial que provoca el ameroide no sea de más de un 25% del diámetro del vaso. Evaluar los intestinos en busca de congestión o medir la presión portal o venosa central para asegurar la correcta colocación del shunt

La alternativa más común al ameroide son las bandas de celofán, personalmente lo prefiero cuando el lugar de colocación permite movimiento. Hay un estudio que da un % mayor de fracasos en el cierre con el celofán y una de las causas es la inconsistencia del material[6], es por ello que debemos buscar uno rico en celulosa sin componentes plásticos, utilizaremos una banda de 10 cm de largo y 16 mm de ancho que doblaremos cuatro veces.

TRATAMIENTO POSTOPERATORIO

La principal complicación es la hipertensión portal con dolor abdominal, diarrea hemorrágica, shock endotóxico y muerte.

Tras dar el alta, se recomienda retirar de forma gradual el tratamiento médico., si evoluciona correctamente se retirarán los antibióticos en dos semanas y si en dos semanas más no se ha observado empeoramiento la lactulosa. A partir de aquí el retorno a una dieta normal (más proteica) será gradual.

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STIFLE INSTABILITY ~ THE CURRENT UNDERSTANDING

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ABSTRACT BODY:

STIFLE INSTABILITY ~ THE CURRENT UNDERSTANDING

The stifle joint is the most common site of orthopedic abnormality in small animals and issues with both the patellofemoral and tibiofemoral articulations having significant and severe issues that lead to pain, lameness, and structural dysfunction. The presence of widespread patellar instability and cranial cruciate ligament (CrCL) deficiency in the pet population demands that the small animal orthopedist has a deep and current understanding of the underlying pathological process and the treatment options available to address the structural abnormalities present.

Patellofemoral Disorders

Instability and subluxation of the patella in relationship to the femur is a common and challenging cause of lameness. The presence of malalignment of the femoro-tibial axis in combination with patellofemoral soft tissue laxity and dysplasia of the femoral trochlea induces patellar instability.The direction (medial or lateral), Grade of luxation (I – IV), the presence of increased distal femoral varus (or valgus), internal (or external) proximal tibial torsion and medial (or lateral) displacement of the tibial tuberosity are all important when determining the best way to manage patellar instability. The patellar position and stability within the trochlea is a combination of alignment, muscular tension and patency of the static stabilizers. This includes the joint capsule and medial and lateral patellofemoral bands which, although they run from bone to bone and are therefore ligamentous, are anatomically a thickened and more structurally solid portion of the adjacent and surrounding joint capsule. Any degree of patellar luxation is abnormal in the dog, either spontaneously or with pressure. The feline stifle has more inherent patella laxity. In the cat, a Grade I patellar luxation (either direction) may be normal. As the severity of patellar luxation increases the underlying osseous abnormalities worsen, making their assessment more important in severely affected patients.

Tibiofemoral Disorders

The primary stabilizing structures of the stifle are the four main transarticular ligaments, the cranial and caudal cruciate (CrCL, CdCL) and the medial and lateral collaterals (MCL, LCL). The secondary stabilizers are the menisci, the joint capsule and associated periarticular soft tissues. There is fifty years of research papers and publications on the canine stifle, its loss of stability, the techniques used to diagnose pathology and instability and the multiple treatments that have been employed to attempt to restore anatomic stability to the stifle once it becomes unstable. To attempt to cover this mountain of information is unwise but there are some key issues that should be considered by the clinician when they are examining a patient with a suspected tibiofemoral injury in particular in terms of the CrCL;

- **Acute vs. Chronic**: the reality of the canine cruciate injury is that there is very commonly an underlying chronic inflammatory process that precedes clinical recognition of an issue. The presence of joint capsule fibrosis, osteophytes and enthesophytes confirms chronicity of the joint pathology. Radiographs will confirm this situation and can be used for this purpose. The loss of a normal-sized and distinct extra-articular fat pad on a lateral radiograph indicates that there is likely the combination of joint effusion AND joint capsular fibrosis.
- **Active vs. Inactive**: A dog with a visible lameness and discomfort on stifle exam has an active issue with the joint. A partial active CrCL tear will have mild to moderate instability with hyperextension discomfort/pain. A complete tear commonly does not have hyperextension pain but will have more pronounced instability on examination. The degree of discomfort on examination (mild, moderate, severe) should be documented.
- **Stability:** As with patellar instability, it is important that an assessment of the stability of the four primary stabilizers be performed. Ligament and tendon injuries are classified as Grade 1 through Grade 4 depending on the degree and type of biologic and structural trauma present. The ability to elicit significant valgus or varus opening of the medial or lateral joint line indicates MCL or LCL injury. In Grade 1 sprains of either ligaments or tendons (desmitis or tendonitis) there is no loss

of structural integrity, pain and mild swelling predominate. As the Grade of injury increases so does the laxity present and can be described as mild, moderate or severe instability correlating with Grade 2 and 3 partial tears and a Grade 4 complete tear. In the case of the CrCL and CdCL their structural integrity is tested with a combination of the cranial drawer test and the tibial compression test (which detects cranial thrust) when examining the CrCL and caudal drawer when examining the CdCL. CdCL injuries are rare but do occur occasionally. An important additional assessment is the degree of internal tibial rotation possible. The CrCL, menisci, joint capsule, and overall joint confirmation dictate rotational stability. An acute traumatic complete (Grade 4) CrCL tear will commonly have a significant internal rotational instability. In partial tears or more chronic complete tears the remnant CrCL ligament fibers and thickened fibrosing joint capsule will commonly act to limit internal rotation.

Tibiofemoral stabilization

The decision to perform static stabilization (extracapsular suture, intra-articular graft) vs. dynamic stabilization (TPLO, CBLO, TTA) is a difficult one that is dictated by several basic factors. Patient size, activity and age are a good starting point. Owner's goals, financial limitations, time limitations, and inherent ability to follow post-operative directions are also significant factors. The research findings have continued to accumulate and over the past five years major questions in terms of the degree and effectiveness of all these techniques have been asked, and in some cases, answered. The work continues but our understanding of the complexity of achieving excellent function post-operatively in an appropriate time period with a reasonable surgical approach has matured. There is no single technique that applies to all patients. The presence of co-morbidity in terms of underlying and active synovitis and osteoarthritis, patellar stability and meniscal damage will affect outcome and must be considered before sweeping prognostic statements are made. Veterinarians who offer and perform stabilization surgery at the general practitioner or specialist level must continue to assess their understanding of stabilization techniques, their appropriateness in offering and performing stabilization surgery and most importantly have open, honest, well informed, and professionally defendable conversations with clients regarding their options for their injured pet. A failure to do this is an individual failure on the part of the consulting veterinarian and a failure on the part of the veterinary profession to act in an ethical and unbiased way.

As the research data has accumulated on the currently employed techniques, we have begun to realize that restoration of the native femoro-tibial relationship and stability is not currently possible. The change to an understanding and use of 'acceptable stability' as a goal which allows functional stability to be present is our new reality.

MEDICINA TRANSFUSIONAL, PRODUCTOS SANGUÍNEOS Y SU INDICACIÓN

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ABSTRACT BODY:

Aunque, las transfusiones son una forma simple de transplante y no carecen de posibles complicaciones; la hemoterapia puede ser la diferencia entre la vida y la muerte, y no debe ser nunca limitada por los riesgos potenciales. Los hemoderivados permiten la administración de agentes específicos para cada patología, minimizan las posibilidades de sensibilización, reacciones adversas, la sobrecarga de volumen y permiten un mejor aprovechamiento de la sangre.

HEMODERIVADOS

Sangre entera: Cuando se administra inmediatamente después de su colecta, provee GR, plaquetas y todos los factores de la coagulación. Mantener refrigerado a 1-6ºC por hasta 35 días en CPDA-1, 1ml c/9ml de sangre. Volumen 450-500ml

Células rojas empacadas CRE: Se obtienen por centrifugación o sedimentación de una unidad de sangre y extracción del plasma. Si el sistema presenta una bolsa con expansor para eritrocitos, se retira completamente el plasma y se agrega la solución a los GRs. En caso contrario se deja 1cm de alto del plasma sobrenadante. Volumen 220-250ml. El Hto. de las CRE es 65-80%, un Hto.>80% aumenta la fragilidad de los GR y las complicaciones hemolíticas. Conservar a 1-6ºC por 35 días.

Plasma: El *plasma fresco congelado* (PFC) se obtiene por centrifugación a 5000g por 5min a 6ºC y se congela a –18ºC antes de 6hs de la colecta. Volumen 220-250ml. Contiene los factores lábiles y estables de la coagulación, incluyendo factor von Willebrand, FVIII, ATIII y los factores estables dependientes de la vitamina K. Mantiene todas sus propiedades por un año, debe administrarse antes de 2hs desde su descongelamiento. El *plasma congelado* es el que se congela pasadas 6hs de su colecta o PFC luego de 1 año de almacenamiento. Puede permanecer congelado por 5 años (desde la colecta) y ser utilizado hasta 5 días después de descongelarlo. Se utiliza como coloide y para proveer los factores dependientes de vitamina K. El *plasma estoqueado* se extrae de sangre estoqueada, no posee factores de la coagulación. Está indicado como coloide y en la hipoproteinemia severa.

Crioprecipitado y criodeprivado: Se obtienen mediante descongelamiento controlado del 90% del PFC a 1-6ºC. El líquido o *criodeprivado* (200-225ml) es ordeñado a una de las bolsas satélites en el sistema de colección múltiple y vuelve a congelarse a -18 ºC. Excelente coloide sin factores la coagulación, dura 5 años desde la donación. El 10% restante es el *crioprecipitado* (20-25ml), contiene todos los factores del PFC y se almacena congelado a -18ºC por 1 año desde la donación.

Concentrados plaquetarios: mantener a 20-24ºC bajo agitación constante. Proveen una cantidad limitada de plaquetas y su vida media es 72hs.

ELECCIÓN DEL HEMODERIVADO

La elección del producto más apropiado se basa en la condición clínica del paciente, presencia de enfermedades subyacentes, estado ácido base y disponibilidad de los diferentes hemoderivados.

En los pacientes anémicos, la necesidad de transfundir células rojas depende de la reserva cardiorrespiratoria, la tasa y magnitud de la hemorragia y del consumo de O2. Los pacientes con signos de shock severo, colapso, taquicardia, taquipnea, pulso débil, caída abrupta del Hto. del 20% en perros y 12–15% en gatos, la hemoglobina y los sólidos totales luego de una fluidoterapia adecuada son candidatos para transfusión. Se transfundirán los pacientes con [Hb] <7g/dl, [Hb] entre 7-10g/dl asociado a hipoxia tisular o necesidad de cirugía y hemorragia no controlada.

Los productos plasmáticos y plaquetarios están indicados en la deficiencia de factores de la coagulación, ATIII y plaquetas funcionales, fibrinólisis excesiva, hipoproteinemia y como aporte coloidal.

Glóbulos Rojos

Se busca el máximo beneficio con el mínimo riesgo, considerando la disponibilidad, la presencia o no de coagulopatías, el estatus hemodinámico del paciente, los órganos y sistemas lesionados y la presencia de enfermedades previas.

En la pérdida aguda de sangre se puede administrar tanto CRE como Sangre entera. Las CRE reducen el riesgo de la sobrecarga de volumen y son de elección en pacientes con falla cardiaca congestiva, trauma encéfalo-craneano o contusión pulmonar. Debido a una mayor concentración de 2,3-DPG y un menor contenido de ácidos y amonio en los pacientes graves y en aquellos con insuficiencia renal o hepática es preferible administrar GR con estoqueado menor

a 5 días. Si se necesitan factores lábiles de la coagulación, administrar sangre entera fresca, PFC y/o crioprecipitado.

Prevención de complicaciones inmunomediadas: Las células rojas, leucocitos y algunos componentes plasmáticos pueden resultar antigénicos induciendo respuestas inmunomediadas como anafilaxia y hemólisis. Las respuestas hemolíticas agudas por incompatibilidades de grupo sanguíneo pueden comprometer seriamente la vida. Los signos clínicos incluyen inquietud, fiebre, taquicardia, urticaria, vómitos, hemoglobinemia y hemoglobinuria, shock, hipotensión severa, CID, falla renal aguda y muerte. La prevención es el único tratamiento exitoso y 100% seguro.

Debido a la producción de potentes hemolisinas, haciendo referencia al locus DEA-1, los perros son identificados como A^- o A^+. Los donantes A^+ resultan seguros sólo para receptores también positivos. Los receptores A^- o de grupo sanguíneo desconocido deberían recibir sangre A^-. Las reacciones hemolíticas en la primo-transfusión son raras en el perro. Sin embargo, la transfusión de sangre A^+ a un paciente A^-, resultará en la rápida formación de anticuerpos que reducirán la vida media de los GRs donados, y provocarán reacciones hemolíticas severas ante una nueva transfusión no compatible realizada después de 72hs.

Los gatos presentan tres grupos sanguíneos A, B y AB. Los raros gatos AB son "receptores universales". En los del grupo B la incidencia de alloanticuerpos naturales es cercana al 100% y motivan reacciones hemolíticas severas si reciben sangre del grupo A o AB. Sólo un 30% de los gatos del grupo A presentan anticuerpos naturales anti-B estos generan reacciones hemolíticas retardadas que no implican un riesgo serio, pero acortan la vida media de los GRs.

El Rapid test^R es un test de aglutinación y hemólisis, los pacientes con anticuerpos auto-aglutinantes darán falsos positivos, por lo que deben recibir siempre sangre A^-, en los perros. El Quick test^R BT identifica anticuerpos monoclonales y es seguro en la identificación de grupo en todos los pacientes.

El uso de donantes compatibles no previene todas las reacciones inmunomediadas. La *prueba de compatibilidad cruzada o "crossmatching"* detecta anticuerpos hemolisantes y/o hemoaglutinantes e identifica incompatibilidades séricas donante-receptor dentro y fuera de los grupos sanguíneos, disminuyendo los riesgos de reacciones inmunomediadas. Un resultado negativo antes de una primo-transfusión no identifica compatibilidad de grupo y no excluye la posibilidad de sensibilizarlo para futuras transfusiones. Al no incluir los glóbulos blancos o las plaquetas, tampoco previene todas las reacciones inmunomediadas. No resulta un test efectivo en pacientes que presenten Ac autoaglutinantes o AHI. Se centrifugan dos tubos de micro hematocrito correctamente identificados con sangre del donante y el receptor, se quiebran donde se delimita plasma y eritrocitos. En un portaobjeto identificado como "compatibilidad mayor" (CM) se coloca una gota de plasma del receptor y una gota de GR del donante. En otro portaobjeto se coloca una gota de plasma del donante y una gota de GR del receptor, prueba de compatibilidad menor (cm). En otros dos portaobjetos se realizan los controles negativos, enfrentando el plasma y los GR del donante (D) y el plasma y los GR del receptor (R). Se incuban a temperatura ambiente durante 5-15min, y se observan macro y microscópicamente en busca de aglutinación y/o hemólisis.

ADMINISTRACIÓN DE HEMODERIVADOS

Los hemoderivados se administran por vía IV o IO. Evitar la administración conjunta de fluidos que contengan Ca porque pueden inducir la coagulación del hemoderivado. Las CRE conservadas en expansor pueden diluirse para disminuir el daño mecánico durante la administración. La colecta y administración

$$\text{Volumen a transfundir} = \text{PC kg} \times \text{vol. sanguíneo ml/kg} \times \frac{\text{(Hto. deseado – Hto. del paciente)}}{\text{Hto. del donante}}$$

$$\text{Volumen a transfundir} = \frac{\text{PC kg} \times \text{(Hb deseada – Hb del paciente)}}{\text{Hb del donante}} \times 70$$

PC: Peso Corporal vol.sanguineo: 90ml/kg perro, 35ml/kg gato

de productos que contengan plaquetas y factores de la coagulación debe realizarse con materiales libres de látex. La dosis inicial de los productos celulares se estima en 20ml/kg de sangre entera o 10ml/kg de CRE. Un cálculo más certero es el siguiente:

Los pacientes con coagulopatías recibirán una dosis inicial de 5-20ml/kg de PFC o crioprecipitado 1U/10kg continuando hasta resolver su cuadro clínico. Los hemoderivados deben ser calentados en un baño a 37ºC. El uso de microondas o mayor temperatura inducirá lisis celular e inactivación de los factores de la coagulación. Los productos plaquetarios deben mantenerse a 22-24ºC bajo agitación. Todos los productos sanguíneos deben administrarse utilizando un filtro para transfusión, en menos de 4hs, en menos de 2hs para administrar los factores lábiles de la coagulación (PFC y Crioprecipitado). Se sugiere la administración de 0.25-0.5ml/kg/minuto en los 1os 30min para observar la aparición de reacciones adversas. La velocidad de transfusión la dicta el paciente, considerando el defecto presente, su peso y la presencia de enfermedades concomitantes. Se debe registrar la temperatura, la FC, FR, el color de las membranas mucosas y el tiempo de llenado capilar antes de iniciar la transfusión y a los 15, 30 y 60 minutos de iniciada, para detectar en forma temprana la aparición de reacciones adversas.

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IMPLEMENTING ONE HEALTH IN LATAM VETERINARY CLINICS

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ABSTRACT BODY:

The concept of One Health is nothing new and comprises collaboration between human and animal healthcare in the context of the health of the shared environment. This relationship is often referred to as the 'One Health Triad'. Although the One Health paradigm often focuses on the relationship of human health to diseases of production animals and wildlife, small companion animals have an essential role within the One Health model. One area in which physicians and small companion animal veterinarians can readily work together is in addressing diseases that can be transmitted between animals and humans (zoonoses). The optimum control and prevention of companion animal zoonoses involve a coordinated One Health approach. Every practitioner should try to connect with local human physicians. Effective interactions between a veterinary practitioner and the local human healthcare practice would be the ideal expression of the One Health principle. Control and prevention of zoonoses are best achieved by enhancing communication between physicians and veterinarians to ensure patients know the risks of and how to prevent zoonoses in themselves, their pets, and other people.

Although small companion animal practitioners often deal with zoonotic diseases in their patients, we still find out that they do not always engage with One Health. Small animal practitioners sometimes forget that they are trained in public health and must consider public health issues in their daily contact with clients. Every practitioner should explain to their clients the importance of preventing infectious diseases, such as the ones preventable by vaccination and transmitted by vectors. A study published in Australia has shown how poorly informed people are about such infections, their risks of exposure, and how infrequently their veterinarian or physician gives them relevant information. A similar study was carried out in Brazil's endemic area for visceral leishmaniasis. The results showed that the level of knowledge of the population about this zoonosis is precarious.

In Latin America, the main reason a client takes his "healthy" pet to practice is to be vaccinated. Veterinary practices have benefited from the annual administration of vaccines. Unfortunately, most clients believe vaccination is the most important reason for annual veterinary visits. The WSAVA Vaccination Guidelines Group (VGG) strongly supports the concept of regular (usually annual) health checks, which removes the focus on annual revaccination. The annual health check may still encompass the administration of selected vaccines, especially the ones that protect against zoonotic infections such as rabies and leptospirosis. Still, the pet does not need to receive every vaccine available since several of them have a duration of Immunity (DoI) of many years and don't need to be given every year. Small animal veterinarians are crucial in educating about pet care, responsible pet ownership, and safe interaction with dogs and cats. They must also stress the importance of all aspects of an individualized health care program, including appropriate diagnostic testing and the control of parasites and zoonotic diseases. A thorough history should be taken to understand the lifestyle and disease risks. There are numerous companion animal zoonoses for which domestic dogs or cats act as a means of transmission or reservoir of infection; or, in some instances, as sentinel species for human infection.

As part of this annual health check visit, veterinarians need to address the importance of parasite control (i.e., roundworms, hookworms, tapeworms, heartworm, ticks, and fleas). Parasite control must be continued in all adult pets, including the indoors, and maintained year-round. Ticks are one of the most important vectors for the transmission of infectious agents to people and their pets (i.e., *Anaplasma phagocytophilum*, *Bartonella* spp., Borrelia spp., *Ehrlichia* spp., *Rickettsia rickettsii*). In most cases, environmental acquisition of tick-borne disease is more likely than acquisition from a pet. However, if a previously infected tick or a tick that acquires infection from a pet detaches before ingesting a complete blood meal, it could subsequently transmit the agent after reattaching to a human. This is rare because most attached ticks feed to repletion before detaching, laying eggs, and dying. Pets can also serve as a transport host by carrying ticks from the outdoor environment into the house, where the tick could drop off from the pet and attach to a human. Fleas can also transmit many infectious agents (i.e., *Bartonella* spp. *Mycoplasma* spp., *Rickettsia felis*, *Yersinia pestis*). Bartonella species infections are particularly significant in immunocompromised individuals. "Cat scratch disease" is an infection caused by *Bartonella henselae* that spreads when an infected

cat licks a person's open wound or bites or scratches a person hard enough to break the skin's surface.

Control of leishmaniasis in Latin America represents one of the most significant challenges for One Health. It provides a clear example of the necessity of human and veterinary medicine to work together to develop strategies for managing and eliminating this disease. The role of the small companion animal veterinarian is to promote education and awareness, prevention, and treatment of infected animals when possible. Sporotrichosis is another significant zoonotic disease prevalent in Latin America, especially in Brazil. Feline sporotrichosis caused by *Sporothrix brasiliensis* has been reported in Brazil's South and Southeast regions since the 1990s and has led to massive zoonotic transmission. The role of the companion animal veterinarian is vital in the management of this important zoonosis.

A key goal of the evolving One Health paradigm includes surveillance of infectious diseases in domestic animals to anticipate the emergence of new zoonoses and protect humans. Although this is limited in LATAM countries for pet-related infections and zoonoses, small animal companion practitioners should take every opportunity to be involved and contribute data from our practices.

Another major area of One Health focuses on antimicrobial resistance and responsible use of antimicrobials in human and veterinary medicine. The small companion animal practitioner should actively subscribe to the principles that underline responsible use.

An important One Health concept is to consider not just the pet (or person) but the entire human–animal family during a veterinary or medical consultation. The close relationship between people and their pets and the intimate sharing of the indoor environment has led to shared lifestyle diseases, for instance, obesity. Despite the high prevalence of overweight and obesity in the human and companion animal populations, few successful interventions are proven to combat this complex multifactorial problem. One key strategy involves effective communication between human and veterinary healthcare professionals with patients and clients about obesity. The veterinarian has a crucial role in promoting an active family lifestyle and may participate in or establish community dog walking programs to promote physical activity in people and their pets.

Small companion animal practitioners can also become directly involved in or initiate community-based One Health programs, such as providing healthcare for homeless people and their companion animals, which may never receive veterinary attention and can be reservoirs of zoonotic infection.

An excellent example of a One Health initiative is the Los Angeles County "Healthy Pets Healthy Families" Initiative. Healthy pets can lead to healthy people! With this in mind, the initiative monitors local animal health trends and generates goals and interventions to improve animal and human health. To do so, HPHF formed a coalition of local partners and established goals for seven focus areas: bite prevention, disaster preparedness, obesity prevention, secondhand smoke, spay and neuter, vaccine-preventable diseases, and zoonoses & parasite prevention. Those are examples of initiatives that small animal practitioners could implement in Latin America.

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WHY ANTIBIOTICS SHOULD NOT BE PART OF YOUR TREATMENT OF ACUTE DIARRHEA IN THE DOG

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ABSTRACT BODY:

For a long time, it has been a common habit to treat dogs presenting with acute diarrhea with antibiotics. The purpose of this lecture is to demonstrate that there are only rare and clearly identifiable circumstances under which antibiotic treatment is helpful in dogs with acute diarrhea. For instance, the use of systemic antibiotics in dogs infected with parvovirus is often necessary.

In the last decade, several studies have shown the effects of various antibiotics such as amoxicillin (with or without clavulanic acid), metronidazole, and tylosin on the fecal microbiota in healthy dogs. While some of these antimicrobials can cause diarrhea, in general, according to their spectrum of activity, their effects are characterized by a decrease in diversity, a reduced abundance of beneficial species, and an increased abundance of potentially harmful microorganisms. The changes are most pronounced while animals are receiving antibiotics, but often persist for weeks after the drugs are stopped. In addition, this dysbiosis also affects the fecal metabolome with an increase in lactate and a decrease in secondary bile acids.

Based on studies from human medicine, microbiome resilience after antibiotic disruption depends on key characteristics such as type, timing, duration, and spectrum of the antibiotic course as well as microbiome modulating factors such as age, underlying disease, antibiotic resistance pattern, and diet.

Antibiotics have traditionally been used in dogs with acute diarrhea, particularly if the diarrhea was bloody. In recent years, several studies have documented that antibiotics offer no significant benefit in these circumstances. The use of amoxicillin and clavulanic acid in dogs with acute hemorrhagic diarrhea syndrome (AHDS) had no positive impact on clinical improvement or length of hospital stay.

The same was documented in a study of dogs with uncomplicated (non-bloody) diarrhea. In addition, the fecal dysbiosis index, a measure of the gut microbiome health, did not normalize faster in dogs receiving antibiotics. Importantly, the proportion of resistant fecal E. coli increased during antibiotic treatment, as expected, and remained high for at least 3 weeks after discontinuation.

However, it should be noted that the use of antibiotics is recommended in a subset of dogs with AHDS. This group consists of dogs that show signs of sepsis (e.g., fever or hypothermia, hypotension, marked neutrophilia or neutropenia, hypoglycemia, hyperbilirubinemia), disseminated intravascular coagulation (DIG), or massive intestinal protein loss, or none after 24-48 h of treatment getting better. In this instance, intravenous administration of broad-spectrum antibiotics such as amoxicillin and clavulanic acid (or equivalent), possibly together with a fluoroquinolone, for a few days is recommended

Possible alternatives to antibiotics include prebiotics, probiotics, and fecal microbiota transplantation:

Prebiotics are designed to promote desirable gut microbiota. They are found in several commercially available foods designed for dogs with acute gastrointestinal conditions. Soluble fibers such as psyllium can also be added to the diet (the dose ranges from 0.5-2.5 g/kg body weight with each meal, starting at the low end of the dose range). They may be particularly helpful in dogs with colitis as they can unfold several beneficial functions for the colonic enterocytes and have a positive impact on the fecal metabolome.

The benefits of probiotics have recently been highlighted in several situations. Dogs with acute diarrhea recovered faster after receiving a probiotic mix than after treatment with metronidazole. In dogs with ADHD, a highly concentrated multistrain probiotic shortened recovery time compared to a placebo and accelerated the normalization of the gut microbiome.

Fecal microbiota (FMT) transfer is an important treatment modality for people infected with *Clostridioides difficile*. FMT also has the potential to promote the restoration of normal gut microbiota in small animals. FMT has been used successfully in dogs with acute diarrhea. A recent study comparing the use of FMT to metronidazole in dogs with acute uncomplicated diarrhea reported no significant differences in clinical improvement. However, FMT resulted in faster recovery of both the fecal microbiome and metabolome.

Interestingly, a working group from the European Network for Optimization of Veterinary Antimicrobial

Treatment (ENOVAT) recently presented preliminary results of its guidelines for use of antibiotics in dogs with acute diarrhea. Based on the available evidence from the literature and, partially, on their expert opinion, the group members recommended against use of antibiotics in dogs with non-bloody or bloody diarrhea with mild or moderate systemic disease. However, they supported the use of antibiotics in dogs presented with acute diarrhea accompanied with severe systemic disease. Their recommendations on the use of prebiotics were equivocal. A full report from the working group is expected in the coming year.

In conclusion, these studies underscore the fact that antibiotic administration has important implications for gut microbiota that need to be considered in decision-making. The prescription of antibiotics should be evaluated on a case-by-case basis and not used as a standard treatment in dogs with acute diarrhea. Ultimately, the decision to prescribe antibiotics depends on the clinical presentation, laboratory test results and the experience of the veterinarian. Alternatives such as prebiotics, probiotics and possibly FMT should be considered, even though there is not yet much evidence to support their beneficial impact in dogs with acute diarrhea.

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QUERATOCONJUNTIVITIS SECA Y CALENTAMIENTO GLOBAL

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ABSTRACT BODY:

QUERATOCONJUNTIVITIS SECA Y CALENTAMIENTO GLOBAL

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La queratoconjuntivitis seca (QCS) u ojo seco es una alteración en la película lacrimal precorneal (PLP), la cual afecta a cualquier raza de caninos y en los casos muy severos puede llevar a ceguera en los pacientes.

La QCS es una enfermedad multifactorial caracterizada por una PLP inestable que no es capaz de mantener la salud del epitelio corneal, lo cual genera inflamación y dolor ocular.

La PLP está compuesta de tres capas o estratos y que cumplen la función de lubricación, nutrición, protección y refracción. El estrato más interno, en contacto con la córnea, es el de mucina, que es producida por las células caliciformes, el estrato medio es el acuoso y es producido por las glándulas lacrimales y el estrato más externo en el lipídico, que es producido por las glándulas de Meibomio.

Existen varias causas para la presentación de QCS, dentro de las cuales se encuentran la agenesia o hipoplasia de las glándulas lacrimales, la conformación del cráneo (braquicefálicos), alteraciones inmunomediadas, hipotiroidismo, daño al nervio facial, distemper, atopía, fármacos e iatrogenia. En la presentación clínica se observa conjuntivitis recurrente e inespecífica, vascularización, pigmentación y engrosamiento corneal. El diagnóstico de QCS se lleva a cabo a través de la prueba de Schirmer, el tipo I determina la producción de lágrimas a través de la irritación producida por la tira de papel en contacto con la córnea; el tipo II (basal) se realiza con la aplicación de anestésico local (proparacaína) antes de la colocación de la tira de papel. Otra forma es a través de la determinación del tiempo de ruptura de la película lacrimal precorneal (TRPLP), la cual se lleva a cabo usando una gota de fluoresceína sobre la córnea, determinando el tiempo de duración de la misma sobre el epitelio corneal, se estima que el tiempo promedio debe ser 15 segundos. Bajo esta forma, se puede establecer el tipo de deficiencia sea cuantitativa, que evalúa la producción de las glándulas lacrimales, o cualitativa, que evalúa la producción de las células caliciformes y de las glándulas de Meibomio.

La deficiencia cualitativa determina la inestabilidad lacrimal o estrés evaporativo. En nuestro centro (Ophthalmo Pet), hemos venido observando con mayor frecuencia este último tipo de deficiencia, por lo que, conjuntamente con un centro aliado (UNOVE), tenemos un trabajo en preparación (estudio aún no publicado), los primeros resultados señalan que las razas Shih tzu, Cocker, Pug, Maltés y Yorkshire terrier como las que presentan menor TRPLP y que va entre los 0 y 5 segundos, cabe resaltar que un número significativo de estos caninos presentan diferentes tipos de problemas de piel, siendo el más frecuente la atopía.

Existen variados esquemas para el tratamiento de una u otra forma de deficiencia lacrimal, dentro de los cuales se encuentra el reemplazo de la PLP (alcohol polivinílico, dextranos, metilcelulosa, viscoelásticos y petrolato), el uso de inmunomoduladores (ciclosporina, tacrolimus o el pimecrolimus), los antiinflamatorios (dexametasona, prednisolona o flurbiprofeno), antibióticos (neomicina/bacitracina/polimixina, tobramicina, ciprofloxacina), suero sanguíneo, acetilcisteína o lavados con soluciones estériles y el uso de células madre.

Merece una mención especial los estudios llevados a cabo en el Perro sin pelo peruano (PSPP), los cuales como manifestación particular presentan un acúmulo de moco de color grisáceo en el canto medial. En un primer reporte llevado a cabo por Y. Aparcana (2007) se realizó la prueba cuantitativa, estableciéndose que el 18.7% de PSPP eran sospechosos o presentaban QCS. En un segundo estudio llevado a cabo por J. Flores y F. Canales (2009), se realizaron las pruebas cuantitativa y cualitativa en los PSPP, estableciéndose para la primera que el 14% de PSPP eran sospechosos o presentaban QCS y para la segunda que el 93.% de los PSPP presentaban mala calidad lacrimal y/o predisposición a QCS, siendo el TRPLP de 5-15 segundos para los casos sospechosos y de 0-4 segundos en los casos positivos. En el 2010, F. Canales publicó un reporte de caso para determinar células caliciformes en un PSPP con Schirmer I de 0 mm para ambos ojos y de TRPLP de 5 -9 segundos, tratado con ciclosporina 2% en gotas por 6 meses sin resultados positivos, se tomaron muestras del fórnix conjuntival ventral y de la conjuntiva palpebral ventral, fueron coloreadas con hematoxilina-eosina

(HE) y azul alsaciano (AB) determinándose la presencia de mucopolisacáridos en las células calciformes, lo cual determina la funcionalidad, el índice de células caliciformes para el fórnix ventral fue de 0.35 (normal alto) y de 0.08 (normal bajo) para la conjuntiva palpebral ventral. En conclusión, según los reportes, el PSPP presenta buen índice para la prueba cuantitativa y presenta predisposición para QCS debido a inestabilidad lacrimal, probablemente debido a hipofunción de las glándulas de Meibomio relacionada a displasia ectodermal propia de la raza.