Continuing Professional Development | Business Success | Clinical Skills

vet 360

Vol 1.0 | Issue 01 | March 2023

CPD Article

The Approach to the Respiratory Distress Patient

Business

Understanding the Income Statement of a Vet Practice

The Role of Physiotherapy in Veterinary Lung Rehabilitation

Also in this issue

Feline Glaucoma | The Many Facets of Hypertension

FORTEKOR®
Get more from life

Elanco™

Slow down the progression of **chronic diseases in dogs and cats with Fortekor™**

New Formulation **Fortekor™** is administered just once a day for the treatment of congestive heart failure in dogs and chronic renal insufficiency in cats. Initiating therapy early can have multiple benefits.

Dogs with chronic heart failure[1]

- Visibly improves quality of life by blocking the RAAS
- Slows disease progression and prolongs life expectancy

Cats with chronic kidney disease[2,3,4]

- Reduces proteinuria and slows progression of CKD
- Helps maintain and improve cats' quality of life
- May help cats live for longer
- Helps improve appetite

AVAILABLE
In 5 mg & 20 mg tablets for cats and dogs

Reference
1. The BENCH Study Group. J Vet Cardiol 1999; 1: 7–18.
2. King JN et al. J Vet Intern Med 2006; 20: 1054–1064.
3. Mizutani H et al. J Vet Intern Med 2006; 20: 1074–1079.
4. Maschio G et al. NEJM 1996; 334: 939–945.

S3 **Fortekor™ 20mg** Reg. no.: 97/5.3.2/10 (Act 101/1965), **Contains:** Benazepril
S3 **Fortekor™ 5mg** Reg. no.: 97/5.3.2/9 (Act 101/1965) **Contains:** Benazepril
Label contains complete use information including cautions, and warnings. Always read, understand and follow the label, and use directions.

Registration holder: Elanco™ Animal Health (Pty) Ltd. Reg. no.: 2019/163893/07. N1 Business Park, Old Pretoria Road, Kosmosdal Ext 7, Gauteng, 0157.
Tel: +27 12 657 6200. **Fortekor™, Elanco™** and the diagonal bar logo are trademarks of **Elanco™** or its affiliates. **Elanco™ Helpline:** 0861 777 735. PM-ZA-23-0045

Editor's Note

Dear colleagues,

It feels as if the year is only just underway and we're already in March! I hope the start of 2023 has been a good one for you, your families and your practices.

This edition of Vet360 focuses on the heart and lungs, covering topics from pulmonary hypertension to blood pressure monitoring in dogs and cats, as well as discussing the role physiotherapy can play in helping us manage our patients with pulmonary disease. There is also a guide to glaucoma in cats, an opthalmological problem which could stem from hypertension. We also bring you two CPD articles, one on managing the respiratory distress patient and one on pulmonary hypertension and cor pulmonale. In addition, you'll see a familiar name returning to our pages: Liesel van der Merwe has scanned the latest journals and gives us a summary.

I want to thank the range of veterinary and paraveterinary professionals who give so generously of their time and knowledge to make Vet360 an interesting and relevant read, every time. We love learning from them!

On that note, I have to give a big a-paw-logy to an author from the September 2022 edition. Katherine McIver wrote a thorough, practical guide to developing antimicrobial stewardship programme in South African practices, and a gremlin crept in and stole her name from the printed version of the article! The correct -- non-anonymous -- version is available online at https://vet360.vetlink.co.za/ . I highly recommend checking it out. You can also find our full back-catalogue of articles there, and subscribers to Vet360 can complete their CPD quizzes online to gain structured CPD points.

I hope 2023 continues to bring you and your practices success and that you enjoy the read!

Marianne

vet360

VET360 aims to be a leader in the field of continuing veterinary development in Southern Africa by providing veterinary professionals from diverse disciplines with tools to help them meet the challenges of private practice. The magazine aims to make information accessible, both paper and electronic, and provide clinical, business and other veterinary information in a concise form to enable the practitioner to rapidly acquire nuggets of essential knowledge

Editor
Marianne Lomberg BVSc (Hons)
Tel: 072 328 7636 | Email: marianne@mariannelomberg.com

Index

Vet360 is a source of current, relevant educational material for practising veterinary professionals in Southern Africa. Published Bi-monthly in hard copy and electronically on www.vet360.vetlink.co.za.

Distribution: Southern Africa, 3 500 copies.

Editor: Marianne Lomberg BVSc (Hons)

Layout and design: Annalize de Klerk

Publisher and Owner: Vetlink Publications

Other Publications by Vetlink: Vet360 Mobile App, Livestock Health and Production Review, Hooo-Hooo, Equine Health Update

Publisher: Madaleen Schultheiss, madaleen@vetlink.co.za

We welcome any comments, contributions, topic suggestions and letters for publication. Send them to:
The Editor, PO Box 232, GROENKLOOF, 0027
Tel: (012) 346 1590, 082 575 6479. Fax: 086 671 9907
Email: marianne@mariannelomberg.com
(Dr Marianne Lomberg)

Advertising Enquiries: AgriConnect
Ilse Liveris
Senior commercial consultant
072-708-4401 / ilsel@agriconnect.co.za

Soekie du Toit
Commercial consultant
078-947-6916 / soekie@agriconnect.co.za

We are currently distributing to SAVA members. A win-win situation for SAVA, Vetlink and the reader. Please to subscribe to ensure access to the CPD MCQ.
Subscription: www.vet360.vetlink.co.za

SAVA South African Veterinary Association
Suid-Afrikaanse Veterinêre Vereniging

vetlink

Understanding the Income Statement of a Vet Practice

Andrew Christie
BComm (Business Management)

This is the second article examining the financial statements of a practice. The first article outlined how assets, liabilities and equity make up the balance sheet, what the key items are, and what they can tell a practice owner. This article introduces the income statement[1] and what it measures, as well as the important information it reveals about the practice.

The income statement is generally regarded as the most important of the financial statements as it measures the financial performance of a practice. This seems fairly straightforward as it simply compares the sales and expenses to establish whether a profit or a loss has been made. However, there are nuances that need to be considered, and the accounting terms are sometimes misleading or just generally misunderstood by anyone who isn't a Chartered Accountant (CA).

What does the Income Statement measure?

As mentioned, the income statement (IS) measures the financial performance of a practice by deducting expenses from revenue and other income. This is easy to prepare, and most vet management systems can do this. In fact, often I create the ISin Excel from information provided by practice managers. It is important to remember that there are different types of IS –at one end is the income statement prepared and finalised by members of the appropriate professional body, while at the other end could be a rough document scrawled on the back of a Hill's notepad. The principles remain the same, however, and so this article can be applied to both formal and informal IS.

What does it mean?

Like the Balance Sheet, the IS is published with comparatives from the period before.[2] This means that anyone using the IS can see what changes have occurred. Additionally, there may be Notes which expand on information where necessary. For example, the calculation of Cost of Sales might be shown in the Notes.

[1] The 'Income Statement' can be replaced with a 'Statement of Comprehensive Income' in the annual financial statements. The main difference is that the overheads are not listed in the Statement but are included in the Notes.

Income Statement		
	Notes	2022
Revenue	1	9 500 000
Less: Cost of Sales	2	4 000 000
Gross Profit	3	5 500 000
Add: Other Income	4	400 000
Less: Overheads	5	4 115 000
Advertising		10 000
Bank Fees		100 000
Cleaning		20 000
Consulting & Accounting		70 000
Depreciation		160 000
Director's Salaries		850 000
Donation		5 000
Freight & Courier		5 000
General Expenses		10 000
Generator		45 000
Insurance		35 000
Legal Expenses		5 000
Motor Vehicle Expenses		25 000
Office Expenses		10 000
Printing & Stationery		20 000
Rates & Taxes		50 000
Rent		420 000
Repairs and Maintenance		20 000
Salaries		2 150 000
Subscriptions		40 000
Telephone & Internet		48 000
Travel		5 000
Wages		12 000
Operating Profit		1 785 000
Less: Taxation	7	499 800
Profit After Taxation		1 285 200
Less: Dividends	8	875 200
Retained Profit for the Year		**410 000**

Very importantly to remember is that the IS items show amounts after the deduction of VAT (Vatex amounts). This is because VAT needs to be accounted for separately.

[2] For the sake of simplicity only one year's figures have been shown here.

Revenue

Revenue is the total earning generated by the sales of products and services of the practice. In a listed company, this could be called 'headline earnings', while I often just refer to it as sales.

Revenue is recognised when an invoice is issued. This can be frustrating when a vet knows that an invoice will only be paid late by a certain customer, since it will be included in the tax calculation.

Cost of Sales

Cost of Sales (COS) refers to the cost of making the sales. It mainly includes drugs used or dispensed, consumables such as cotton wool, and, in small animal practices, pet food and merchandise. It is calculated:

Stock on Hand 01 March 2022		R1,500,000
+	Purchases for the year	R3,500,000
	Stock on Hand 28 February 2022	**R1,000,000**
=	Cost of Sales	R4,000,000

COS is called a variable expense - if Revenue doubles, one expects, then, the cost of making those sales to double as well. This is a something that I watch very closely with my clients – if sales increases by, say, 10% but COS increases by 15% then the cost of making the increase in sales is higher. Sometimes the higher increase in COS means that the Gross Profit actually decreases – the practice would have done better to not have increased turnover.

Gross Profit

Very simply, Gross Profit (GP) is the amount of revenue that a practice retains after paying its trade creditors for the drugs and merchandise etc. reflected in COS. This is a very important figure as it indicates how well stock is being managed – is too much merchandise being held, are too many of a particular type of drug being held? Often vets argue that they have to have huge amounts of consumables because they are used so quickly. My answer is always the same –Are you in business to have stock or to make money?

Other Income

Other Income refers to money generated by the practice other than that generated by operations. For example, in the example above, an old x-ray machine was sold for R400,000 after it was replaced. Because practices aren't in the business of buying and selling used veterinary equipment, it is not recognised as revenue. Nevertheless, it is income which has to be shown on the IS.

Overheads

Overheads are the other expenses of a practice – the fixed expenses. They are called fixed expenses because they don't change much, if at all, if turnover increases. For example, in most cases, rent remains the same as the landlords just want their money - they don't care if turnover is good or poor.

Generally, Salaries are the highest item in overheads. Salaries include the tax and UIF paid on behalf of employees since they are also salary expenses.

Director's Salaries are also generally quite high. If they are not mentioned specifically, they may be included in Salaries. Because the two salary items are high, when I compare changes in overheads, I like to separate the overheads into 'Salaries', Director's Salaries', and 'Other Overheads'.

Depreciation is a non-cash item since no money is paid. Consider this practice which we know purchased a new x-ray machine. Say the cost was R800,000. It is not listed as an expense, even though they paid the supplier. This is because the x-ray machine is a non-current asset and therefore shown on the balance sheet.

Depreciation allows the purchase price to be written off over its useful life. Say the R800,000 can be used for 5 years. This means that the depreciation is R800,000 ÷ 5 = R160,000 for each of the 5 years. Depreciation can be calculated in a few different ways and the 'useful life' is normally defined by SARS. It's complicated and so best left to your accountant to calculate.

In a nutshell, depreciation increases a practice's expenses which in turn reduces the amount of tax that has to be paid.

Operating Profit

Operating Profit is the profit that is made through the operations of the practice – Revenue less Variable and Fixed Expenses. This figure generally gives us the best indicator of how a practice is performing.

Taxation

This is the Company Taxation that is paid on the operating profit and does not refer to VAT or any other taxation. For tax years ending on or after 31 March 2023, this will be 27%.

Dividends

One of the challenges for the owner of a practice is how they should draw their earnings from the practice. They can choose to get paid a salary or they can choose to draw dividends from the business or a mixture of the two. It can get very complicated and best dealt with by your tax consultant or accountant.

Retained Profit for the Year

Retained profit for the year refers to the profit that is kept in the business for future projects or investments. We can see in the Income Statement above that the owners have elected to take out R875,200 as dividends, but have left in R410,000 for a planned expansion or new vet management system etc.

Since it is being kept in the business, it is effectively money that the owners have invested in the practice and is therefore also captured as Equity on the balance sheet.

Transpharm

WE TAKE CARE OF YOUR HEALTH

TRANSPHARM IS A PHARMACEUTICAL, SURGICAL, VETERINARY WHOLESALER AND DISTRIBUTOR SERVICING:

- Pharmacies
- Hospitals
- Doctors
- Veterinarians
- Dentists
- Homeopaths
- Allied Healthcare Professionals

WIDER RANGE
+ 30 000 PRODUCTS

BETTER PRICES
+ 800 SUPPLIERS

FREE DELIVERY
NO MINIMUM ORDER REQUIRED

OPEN AN COD ACCOUNT OR 30 DAY ACCOUNT
Any registered company can also open an account with us and start saving!

ONE OF SOUTH AFRICA'S LEADING PHARMACEUTICAL WHOLESALERS AND DISTRIBUTORS

GAUTENG
orders@transpharm.co.za | clientservice@transpharm.co.za
TEL: 012 377 9000
WESTERN CAPE
infowc@transpharm.co.za | TEL: 021 929 2120
KWAZULU-NATAL
kznorders@transpharm.co.za | TEL: 0861 TPHARM (874276)

WWW.TRANSPHARM.CO.ZA

CATCH US ON WWW.TRANSPHARM.CO.ZA
our exclusive online ordering platform

Feline Glaucoma

Dr Izak Venter
BVSc, MMedVet (Ophthal)
Digital Veterinary Ophthalmology Services
www.dvos.co.za
Facebook DVOS VETS

Introduction

Feline glaucoma can be an extremely frustrating disease to deal with. One of the reasons being that as many as 70 % of cats are blind at the time of initial presentation. Acutely painful and congestive glaucoma is rarely recognized in cats. Another reason is that very few of the available antiglaucoma medications are actually effective in cats. Fortunately, in comparison to the canine eye, the feline eye is relatively resistant to glaucomatous damage and, surprisingly, vision may be preserved even in some chronically glaucomatous cats with buphthalmos.

Rather than being a disease entity in itself, glaucoma is considered to be a group of diseases with abnormally elevated intraocular pressure as a common feature and symptom.

A sustained increased intraocular pressure [IOP] triggers a cascade of events in the optic nerve and retina, leading to progressive optic nerve damage, retinal degeneration and eventual blindness, even if the intraocular pressure has returned to normal values.

Caution should be exercised in diagnosing glaucoma based on a single, elevated IOP reading. Elevated IOP in the absence of clinical evidence of glaucomatous optic nerve and retinal damage is termed ocular hypertension.

Aqueous humor dynamics in cats

Maintenance of normal IOP in cats relies on a delicate balance between aqueous humour outflow and production. Aqueous humour is produced at the level of the nonpigmented epithelium of the ciliary processes.

In cats, more than 97% of the aqueous humour exits the eye by the conventional route: through the pupil from the posterior to anterior chamber, then via the trabecular meshwork and collector channels of the angular aqueous plexus into the intrascleral venous plexus and ultimately, the general circulation.

Less than 3 % of aqueous humour outflow occurs by the uveoscleral route in cats(in other words through the iris and ciliary body stroma to the suprachoroidal circulation and vortex veins, and through the sclera to the episcleral tissue). [Figure 1]

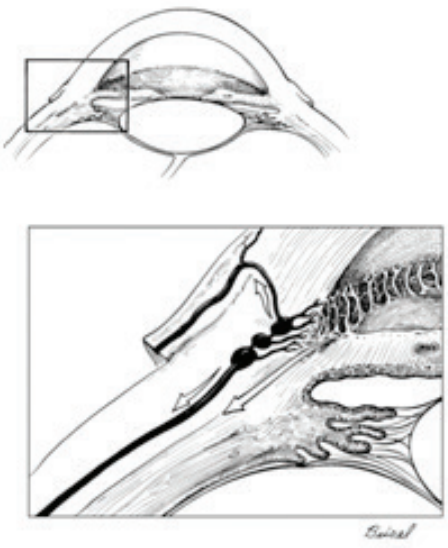


Figure 1: *Normal iridocorneal angle. The conventional outflow of aqueous humour through the pectinate ligaments, ciliary cleft and uveal trabecular meshwork into the anterior ciliary veins, conjunctival veins, and into the vortex veins. Unconventional outflow pathways through the stroma of iris and ciliary body into the vortex veins. (University of Georgia, ophthalmology teaching illustrations.)*

Increased IOP is always the result of decreased outflow and never increased inflow of aqueous humor. The "sink model" describes this balance of inflow and outflow and makes it easy to understand that glaucoma is always a result of a "plugged drain" and never a "tap" that is opened too wide.

Epidemiology and classification of feline glaucoma

Compared to dogs, glaucoma is a relatively uncommon clinical diagnosis in the cat. The incidence may, however, be underestimated as many feline cases go unrecognized. Glaucoma appears to be a rather common indication for enucleation in cats, representing 29% of feline globe submissions to the Comparative Ocular Pathology Laboratory of Wisconsin (COPLOW).

Glaucoma is often classified according to whether the inciting cause of aqueous outflow obstruction is 'primary' or 'secondary'. The primary glaucomas have no other identifiable antecedent ocular disease and are further subclassified into open- and closed-angle glaucomas as well as congenital glaucoma. Secondary glaucoma is more common than primary glaucoma and results from antecedent ocular or systemic disease processes, most commonly neoplasia and uveitis.

Primary glaucoma

Compared with the dog, primary glaucoma is rarely observed in cats, although a breed predisposition has been described in the Siamese, Persian, European Shorthair and Burmese cat. In most cats with primary glaucoma, the iridocorneal angle is open. Narrow- or closed-angle glaucoma has been reported in Burmese cats, but is exceedingly rare.

Congenital glaucoma usually manifests at a very young age and is associated with buphthalmos, which can be quite marked because of the decreased scleral rigidity in young animals(see Figure 2). Other ocular malformations, for example microphakia, macrophakia, pectinate ligament dysplasia, multiple uveal cysts, and persistent pupillary membranes, may also be present.

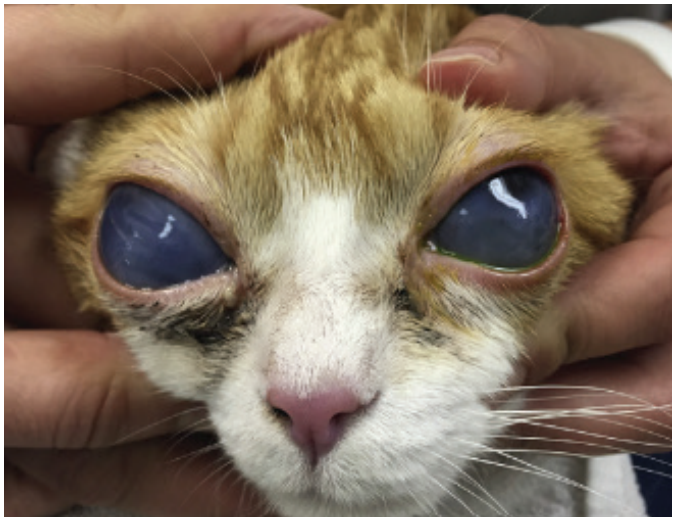


Figure 2: *Severe bilateral pronounced buphthalmos, dense oedema, and secondary exposure keratitis in a 7-month-old domestic shorthair cat with congenital glaucoma. [McLellan GJ, Teixeira LBC Feline Glaucoma. Vet Clin Small Anim. 45 (2015) 1307–1333].*

Open-angle glaucoma is characterized by an insidious, gradually progressive globe enlargement in middle aged and older adult cats. The disease may be unilateral or bilateral but is often asymmetricalin presentation. No abnormalities are noted within the iridocorneal angle or trabecular meshwork. The resistance to aqueous outflow is most likely at the level of the scleral venous plexus and vortex veins.

Causes of secondary glaucoma

Intraocular neoplasia

Glaucoma secondary to intraocular neoplasia is the most common type in cats and accounted for more than half of the feline glaucoma cases in a review of the COPLOW archive. The three most commonly diagnosed neoplasms in glaucomatous cats are melanoma, lymphoma and post traumatic sarcoma.

Neoplasia may lead to the blockage of the outflow pathways in the trabecular meshwork and ciliary cleft, associated with pre-iridal fibrovascular membranes and subsequent angle closure. Development of secondary glaucoma in melanoma patients usually occurs at an advanced stage of the melanoma.

Uveitis

Uveitis, in particular lympho-plasmacytic uveitis, is the second most common cause of glaucoma in cats. Pathogenic mechanisms that can contribute to the development of glaucoma in cats include:

- Inflammatory infiltrates obliterating the aqueous outflow pathways.
- Formation of preiridal fibrovascular membranes.
- Peripheral anterior and / or, posterior synechiae.
- Chronic inflammation can also contribute to vitreal prolapse or lens luxation.

Trauma

Both penetrating trauma and blunt force trauma can result in secondary glaucoma. Blunt trauma may lead to hyphaema or distortion of anterior segment anatomy. Penetrating ocular trauma usually leads to severe uveitis and consequent glaucoma. It is important that owners are educated about the increased risk of neoplasia [Feline Post Traumatic Sarcoma] development in cats with eyes that have been traumatized.

Hyphaema

Intraocular haemorrhage or hyphaema is common in older cats, related to systemic hypertension in older cats. Other causes for hyphaema in cats include trauma, systemic bleeding disorders and intraocular neoplasia. Cats with single episodes of haemorrhage resulting in only a partial hyphaema are unlikely to develop glaucoma, and blood cells are generally cleared from the anterior chamber over a matter of days.

Recurrent hyphaema, on the other hand, leads to "8-ball hyphaema" that completely fill the anterior chamber. Too many blood cells cause a mechanical obstruction of the iridocorneal angle and trabecular meshwork, with glaucoma as a result.

Feline aqueous humour misdirection syndrome [FAHMS] or malignant glaucoma

FAHMS is an unusual form of insidious glaucoma primarily occurring in older cats. This condition is characterized by a uniformly very shallow anterior chamber (see Figure 3). The degree of IOP elevation in affected eyes ranges from mild to severe, with modest elevations being most typical, and in the early stages IOP may actually be normal. In severely affected eyes, a pronounced myopia (as much as 16.5 D) was previously identified, and was attributed to the anteriorly positioned lens.

The most likely pathophysiology of FAHMS is the posterior rather than anterior direction of aqueous humour. It has been postulated that aqueous humour is misdirected posteriorly through breaks in the anterior vitreous face that act as one-way valves, resulting in it becoming trapped within 'pools' in the vitreous cavity.

Anteriorly displaced vitreous becomes further compressed and tightly apposed against the lens and ciliary processes. This tight apposition of vitreous and lens further diverts aqueous humour into the vitreous cavity in a vicious cycle, hence the term malignant glaucoma sometimes being used.

Anterior displacement of the lens zonules and ciliary processes intensifies the pupil block. With progressive increase in IOP the lens iris diaphragm is being displaced forward, collapsing the ciliary cleft, compressing the trabecular meshwork, and narrowing the iridocorneal angle.

Figure 3. *Very shallow anterior chamber visible as the result of anterior displacement of the lens and iris. This is a classic clinical sign of FAHMS. [McLellan GJ, Teixeira LBC Feline Glaucoma. Vet Clin Small Anim. 45 (2015) 1307–1333].*

Lens-associated secondary glaucoma

Primary lens luxation in cats is rare and seldom plays a direct role in the pathogenesis of glaucoma in this species. Feline lens luxation is more often recognized secondary to chronic uveitis and glaucoma.

Intumescent cataracts may lead to secondary glaucoma through the obstruction of aqueous flow through the pupil by the swollen lens. This is also less common in cats compared to dogs,

and may be the result of the elliptical or slit-shape of the feline pupil rendering it resistant toconventional mechanisms of lens-associated pupil block.

Clinical signs

As mentioned in the introduction, acute, congestive glaucoma which is so typical for the dog, is almost never observed in the cat. The clinical signs are more subtle and therefore easily overlooked. Unfortunately, at the time of presentation most eyes with glaucoma have already developed buphthalmos.

Observant owners may notice anisocoria due to relative pupil dilation in the affected eye. Affected cats seldom show obvious signs of severe ocular pain, even in the face of very high IOP, and glaucomatous cats generally maintain a normal appetite. The clinical signs of glaucoma are summarized in table 1.

Table 1: *Clinical signs of glaucoma*

Acute glaucoma	Mydriasis
	IOP >25 mmHg
	Conjunctivitis
Chronic glaucoma	Buphthalmos
	Haab's striae
	Exposure keratitis
	Focal or generalized corneal oedema
	Mydriasis
	Blindness
	Negative pupillary light reflex
	Absent menace response
	Negative dazzle reflex
	Anterior lens luxation
	Optic nerve cupping
	Retinal degeneration
	Lens luxation

Cornea

Corneal oedema in acute glaucoma is almost invariably present in dogs but is not as frequently seen in cats. This could be the result of a more efficient pump mechanism of the corneal endothelium against the hydrostatic pressure in the aqueous humour. In chronic glaucoma, generalized corneal oedema ismore often present.

As the cornea stretches during the development of buphthalmos, linear ruptures in Descemet's membrane, called Haab's striae, may occur. These are visible as lines of intense cornealoedema (see Figure 4).

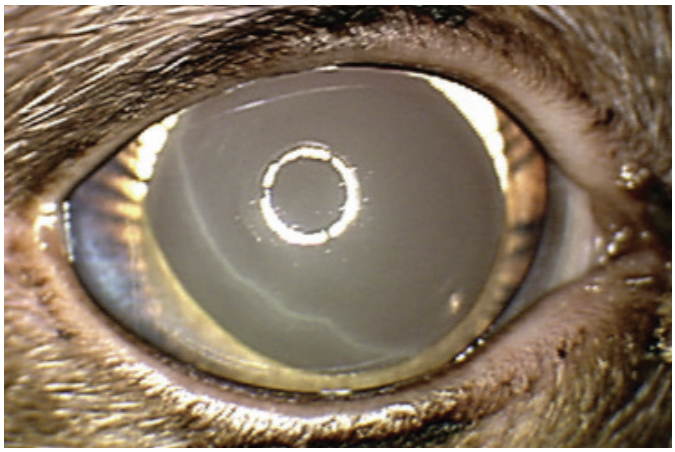


Figure 4: *A very clear Haab striae, which represent tears in Descemet's membrane, due to globe stretching is visible. Other clinical signs of glaucoma visible in this photo are slight buphthalmos and iris hypoplasia. [McLellan GJ, Teixeira LBC Feline Glaucoma. Vet Clin Small Anim. 45 (2015) 1307–1333].*

Uvea

Sustained elevated intraocular pressure will cause paralysis of the iris musculature and a fixed, dilated pupil. Mydriasis is often the only symptom observed in acute glaucoma in cats. In chronic glaucoma, iris atrophy may become apparent.

Retina & optic nerve

Panretinal degeneration, which is a common feature of glaucoma in dogs and isseen as retinal vascular attenuation and increased tapetal reflectivity, is seldom observed in cats, except in very longstanding cases.

Optic disc cupping can be difficult to identify by ophthalmoscopy in cats, because the feline optic nerve head lacks myelin and appears depressed relative to the plane of the surrounding retina, even in normal animals.

Diagnosis

Normal cats exhibit a pronounced circadian rhythm in IOP, with highest values at night (by about 4–5 mmHg) and a gradual decline in IOP occurring during the day. Intraocular pressure in cats is also influenced by the age of the subject, being considerably lower in geriatric cats as well as young kittens within the first few weeks of life.

Marked asymmetry in IOPs, with a difference of 12 mmHg or more between eyes, or an IOP of 25 mmHg or more in older cats (measured with a Tono-pen XL), should prompt a thorough evaluation for ocular abnormalities including glaucoma.

Prior application of a mydriatic drug may lead to substantial increases in IOP in both normal and glaucomatous cats. Differences in measurements obtained by different tonometer types are also clinically significant. It is therefore important that a consistent tonometer type and model is used for clinical monitoring.

A number of tonometers have been investigated in cats including
- Schiøtz indentation tonometer
- Pneumotonometer
- Mackay-Marg type applanation tonometers (Tono-pen, Tonopen XL)
- Perkins applanation tonometer
- Induction/impact or rebound tonometer (TonoVet).

Most applanation tonometers dramatically underestimated IOP above about 30 mmHg. The TonoVet rebound tonometer is more accurate than the Tono-Pen, particularly at IOPs >30 mmHg; does not require application of topical anesthetic; is well-tolerated by cats, and may be considered most suitable among current, commercially available tonometers, for diagnosis and monitoring of glaucoma in cats.

Gonioscopy

Gonioscopy is seldom used by most general practitioners, but is a valuable adjunctive diagnostic procedure in glaucoma patients. The anterior chamber is deeper and the opening of the iridocorneal angle in cats is considerably wider than in humans and dogs. The individual fibers of the pectinate ligament that span the anterior opening of the ciliary cleft, are very fine and relatively sparse in normal cats in comparison to the typical appearance of the canine drainage angle.

The iridocorneal angle and opening of the ciliary cleft are best evaluated by gonioscopy, using a goniolens. However, it is possible to evaluate a significant portion of the feline drainage angle by direct observation using focal illumination and magnification. A normal otoscope works well for this.

Although primary closed-angle glaucoma is very uncommon in cats, gonioscopy may aid in the identificationof closure of the iridocorneal angle, neoplastic infiltrates, or neovascularization in cats with secondary glaucoma.

Treatment

Treatment should be directed at the underlying disease process leading to secondary glaucoma, whenever possible. Glaucoma therapy in cats must be tailored to the individual, and response to therapy must be closely monitored. Regular tonometry is therefor extremely important as part of the treatment plan.

There are a number of important considerations to keep in mind when formulating a treatment plan for a feline glaucoma patient.
- Is the glaucoma primary or secondary?
  - ❍ Direct treatment at the primary disease process whenever possible

- Has the underlying aetiology been determined?
  - ❍ Enucleation or even exenteration may be indicated if intraocular neoplasia is suspected.
  - ❍ For glaucoma secondary to uveitis, attempt to identify underlying cause and treat specifically.
  - ❍ Miotics may be contra-indicated due to their potential to contribute to further instability in the blood–aqueous barrier.
- What is the likely potential for vision in the affected eye, and is the contralateral eye at risk?
  - ❍ For irreversibly blind, painful eyes, analysis of the cost/benefit ratio will generally favour enucleation.
  - ❍ Although cats do not show obvious signs of pain in chronic glaucoma, it may still be in the patient's best interest to enucleate blind buphthalmic eyes.

Medical treatment of glaucoma

Medical treatment of glaucomatous eyes either reduce the production of aqueous humour production "closing the tap", or increase outflow of aqueous humour "unblocking the sink".

The treatment of feline glaucoma specifically can be extremely frustrating as a number of products giving good results in canines and humans are either not effective in cats, or contraindicated due to severe side effects.

Carbonic anhydrase inhibitors (CAIs)

CAIs are used for the management of glaucoma in many species. This drug class includes topical preparations (such as dorzolamide hydrochloride 2% ophthalmic solution and brinzolamide 1% ophthalmic suspension) as well as oral formulations (such as acetazolamide, methazolamide, and dichlorphenamide). CAIs lower IOP by inhibiting the production of aqueous humour in the eye.

Water within the nonpigmented ciliary body epithelium hydrates carbon dioxide to form carbonic acid (H_2CO_3), which then dissociates into protons (H^+) and bicarbonate ions. The responsible enzyme is carbonic anhydrase. The newly formed bicarbonate ions then move into the posterior chamber of the eye, thereby creating an osmotic gradient. As a result, water from the ciliary stromal vasculature migrates into the posterior chamber to form aqueous humour. This process is interrupted by CAIs as these drugs block 98%-100% of the available carbonic anhydrase enzyme (CA) within the ciliary body. CAIs are one of the most effective antiglaucoma medications available and may reduce intraocular pressure by up to 50%.

Topical dorzolamide 2% has been shown to significantly lower IOP in both normal and glaucomatous cats when administered three times daily. Brinzolamide 1% does not significantly lower IOP in normal cats when applied twice daily, but does significantly lower IOP in glaucomatous cats when administered three times daily, albeit to a lesser degree than dorzolamide.

Systemic CAIs inhibitors may be toxic in cats and should only be administered for a short period of time and in lower dosages. Reported side effects include vomiting, decreased appetite, lethargy, and rapid breathing because of drug-induced metabolic acidosis.

The use of dorzolamide 2% ophthalmic solution impairs hydrogen secretion from the distal renal tubule. This induces distal renal tubular acidosis in the cat, which manifests clinicopathologically as hyperchloraemic metabolic acidosis with hypokalaemia and alkaline urine.Routine electrolyte monitoring is advised in all feline patients treated with dorzolamide 2% ophthalmic solution. Topical CAI application is further associated with conjunctival irritation and hypersalivation in some cats. In spite of this, dorzolamide remains

the first-line therapy of choice for glaucoma in cats, regardless of the underlying pathogenesis of glaucoma, as CAIs do not contribute to pupil block or intensify any pre-existing uveitis.

Beta-blockers

Nonselective beta-receptor blocking agents such as timolol maleate, reduce IOP, thus decreasing aqueous humour formation. They have no effect on aqueous humour outflow.

Topical application of a single dose of timolol maleate 0.5% can lead to a mean IOP reduction of about 22% in the treated eye. However, it induce miosis in the treated eye, therefore intensifying some forms of pupil block glaucoma, and is contraindicated in patients with uveitis. Timolol may further cause bradycardia and bronchoconstriction and is contraindicated in cats with feline asthma or cardiac disease.

Timolol is commercially available in combination with dorzolamide. No significant additive ocular hypotensive effectsare seen with a combination of the CAI, dorzolamide 2% and timolol 0.5%, relative to the effect of treatment with dorzolamide 2% alone.

Cholinergics

Cholinergic drugs, for example Pilocarpine, lower IOP and induce miosis. Contraction of the longitudinal ciliary body musculature increase conventional aqueous humour outflow. Pilocarpine is a useful drug for the treatment of open-angle glaucoma. It is important to note that Pilocarpine is contraindicated in eyes with secondary glaucoma associated with uveitis. Many cats do not tolerate topical pilocarpine very well. For these reasons this product is very seldom indicated.

Adrenergic agonists

Because of their systemic adverse effects, including lowering of heart rate and vomiting, adrenergic agonists are not recommended for the management of glaucoma in cats.

Prostaglandin analogues

Prostaglandin analogues are widely used and extremely effective in certain canine glaucoma patients. Species differences in prostanoid receptor distribution within different ocular tissues have major implications for the effects, and efficacy of topical prostaglandin analogue therapy for glaucoma in cats. Prostaglandin analogues such as latanoprost and travaprost causes intense miosis in cats, that is attributable to the presence of FP receptors in the iris sphincter muscle of cats. In dogs and humans, FP receptors are responsible for the IOP lowering effects of prostaglandin analogues. FP receptors are lacking in the ciliary body of cats and EP receptors are predominantly responsible for relaxation of feline ciliary muscle. The result is that prostaglandin analogues have no significant IOP-lowering effect in normal cats.

Topical corticosteroids

Topical corticosteroid therapy (usually with either 0.1% dexamethasone or 1% prednisolone) is a mainstay of therapy for chronic uveitis as well as non-herpetic ocular surface disease in cats. However, topical administration of corticosteroids can increase IOP by about 5 to 10 mmHg in some normal cats. Should steroid-induced ocular hypertension occur, a change in the topical anti-inflammatory drug to a topical non-steroidal product is indicated.

Surgical treatment of glaucoma

Enucleation

For irreversibly blind eyes, eyes where there is a suspicion of intraocular neoplasia and chronic glaucoma due to previous ocular trauma, enucleation must be considered. Enucleated globes should always be submitted for histopathological diagnosis.

In cats, enucleation is strongly preferred over evisceration with intrascleral prosthesis (ISP) placement because the success rate of ISP placement in this species is lower than in dogs. The author has seen a patient developing a Feline Post Traumatic Sarcoma after the placement of an intrascleral prosthesis.

Cyclodestructive procedures

Cyclosdestructive procedures include laser cyclophotocoagulation, cyclocryotherapy as well as Intravitreal gentamicin,and have been described as a means to reduce aqueous production. The success of cyclocryotherapy is considerably lower in glaucomatous cats than in dogs, and often repeated treatments may be required. These procedures incite significant inflammation andare contraindicated in cats with glaucoma secondary to uveitis or neoplasia.

Intravitreal injection of gentamicin for pharmacologic ablation of the ciliary epithelium is widely considered to be contraindicated in cats, due to the potential for the development of life-threatening Feline Post Traumatic ocular Sarcoma.

Anterior Chamber Shunt Procedures

Gonio-implantation surgery in glaucomatous cats is not routinely done as treatment failure is common. Because chronic uveitis is commonly associated with development of secondary glaucoma, pre-existing inflammation often contributes to shunt failure related to anterior tube occlusion.

Summary

Glaucoma in cats typically follows an insidious, gradually progressive clinical course and is most often secondary to other ocular or systemic disease, especially intraocular neoplasia. A thorough examination to identify the underlying cause is crucial. Treatment of glaucoma presents a clinical challenge in cats due to the limited number of effective or safe products available. Enucleation is generally the most appropriate treatment for cats with irreversibly blind, painful eyes.

References

1. Czepiel TM, Wasserman NT. Hypokalemia associated with topical administration of dorzolamide 2% ophthalmic solution in cats. Veterinary Ophthalmology. 2021;24:12–19
2. Dietrich U. Feline Glaucomas. Clin Tech Small Anim Pract. 2005; 20:108-116
3. McLellan GJ, Teixeira LBC Feline Glaucoma. Vet Clin Small Anim. 2015; 45: 1307–1333.
4. McLellan GJ, Miller PE. Feline glaucoma. A comprehensive review. Veterinary Ophthalmology. 2011; 14, Supplement 1, 15–29

Turning the world of Metronidazole

upside down

Pharmapack Awards 2018

METRONIDAZOLE LIQUID SUSPENSION
Flexible dose

EASILY ADMINISTERED
Pharmapack Awards Winner

2 SIZES FOR ALL DOGS
30 ml and 100 ml

WELL ACCEPTED [6] BY DOGS
Flavoured

NEW

Flavoured Liquid Suspension Metronidazole for Dogs!

With the award-winning smart cap for accurate dosing and protected administration!

Eradia®

FOCUS ON METRONIDAZOLE

Chronic diarrhoea can have different origins and may respond to antibiotics like metronidazole[1]

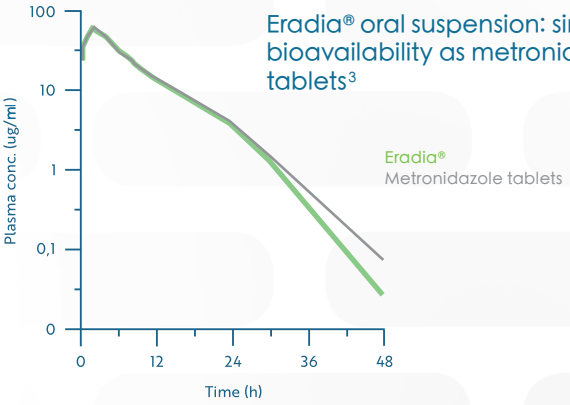
136 dogs with chronic or persistent diarrhoea

ERADIA® : A METRONIDAZOLE-BASED ORAL SUSPENSION

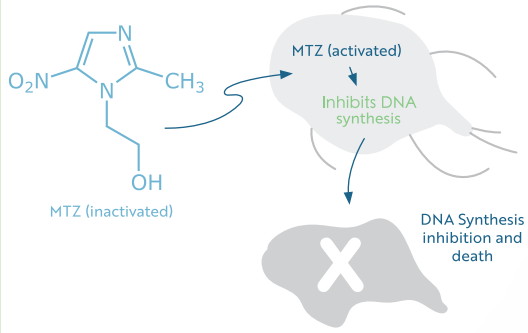
PRIMARY ENTEROPATHY **90 %**

- Other e.g. Neoplastic
- Chronic Inflammatory Enteropathy **71 %**
  - Dietary responsive
  - Antibiotic responsive
  - Inflammatory Bowel Disease
- Infectious **13 %**
  - E.g. Giardia, Clostridia, Mixed Infection

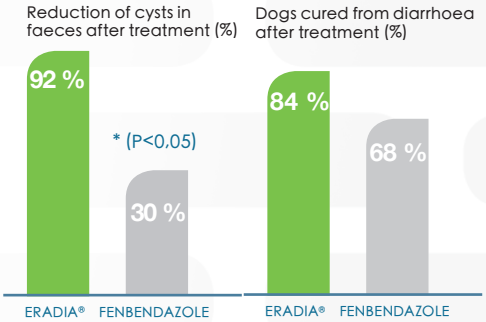
Eradia® oral suspension: similar bioavailability as metronidazole tablets[3]



Metronidazole is active specifically against micro-organisms: Giardia, obligate anaerobic bacteria[2]



Eradia® oral suspension is efficient in decreasing cyst excretion and curing diarrhoea in Giardia infected dogs[4]



Metronidazole & fenbendazole: 50 mg/kg/day as recommended.

73 dogs treated with Eradia®

58 dogs treated with fenbendazole

VERY SATISFYING ACCEPTABILITY[5] **100 %** intake **80 %** total consumption

REFERENCES

(1) Volkmann M, Steiner JM, Fosgate GT, Zentek J, Hartmann S, Kohn B. Chronic Diarrhea in Dogs – Retrospective Study in 136 Cases. Journal of Veterinary Internal Medicine. 2017;31(4):1043-1055. (2) Samuelson J. Why Metronidazole Is Active against both Bacteria and Parasites. Antimicrobial Agents and Chemotherapy. 1999;43(7):1533-1541. (3) Pellet T, Toutin C, Fournel S, Gruet P. Bioequivalence study between an oral suspension and a tablet formulation of metronidazole following a single oral administration in dogs. EAVPT congress 2018. (4) Faure L, Fournel S, Nicolas C, Rigaut D. A Field Clinical Study to Confirm the Efficacy & Safety of a Metronidazole-based Oral Suspension in Dogs Naturally Iinfested byGiardiasis: Comparison to Fenbendazole. IJARVM 2018. (5) Nicolas CS, Martins F, Pujol C. Evaluation of palatability of a metronidazole-based oral suspension in dogs when poured on the food and at different stages of shelf-life. In: Proceeding of the 5th World Congress on Advanced Clinical Trials and Clinical Research. J Clin Trials. 2018;8:45.
(7) Nicolas CS, Martins F, Pujol C. Evaluation of palatability of a metronidazole-based oral suspension in dogs when poured on the food and at different stages of shelf-life.

Eradia®, Reg. No: 19/17.4/27 (Act 101/1965), Composition: Each 1,0 ml Solution contains 125 mg Metronidazole.

Virbac (Pty) Ltd (Reg. No: 1990/003743/07) Private Bag X115, Halfway House 1685. T (012) 657-6000 | F (012) 657-6067. za.virbac.com

Shaping the future of animal health

Virbac

The Approach to the Respiratory Distress Patient

Dr Marinell Breytenbach
BSc, BVSc (Hons)
Resident in Emergency and Critical Care

Respiratory distress is, to use the technical terminology, Scary Stuff, for both the patient and the attending veterinary team. That is because, of all the basic physiological needs, the need for oxygen to reach the tissues in the body is the one which will most rapidly cause death if it is disrupted. While oxygen is carried to the tissues in the blood, it enters the blood through alveolar ventilation.

An animal which, for any reason, is not receiving adequate alveolar ventilation, will show dyspnoea and other signs of respiratory distress. As a veterinary team, it is our job to rapidly establish the cause of the distress and return our patient to a state of adequate alveolar ventilation, before the damage caused to tissues by the lack of oxygen becomes irreversible or fatal.

Defining adequate oxygenation

Three different terms are used when referring to oxygen in the blood, or lack thereof.

The Partial Pressure of Oxygen (PaO2) is a measure of the partial pressure of oxygen dissolved in arterial blood plasma. It is meaured by a blood gas analyser and is the most accurate indicator of the lungs' ability to move oxygen from the atmosphere into the blood (alveolar ventilation). At sea level, normal PaO2 is 80 – 110 mmHg.

Normally functioning haemoglobin molecules bind the oxygen dissolved in blood plasma, leading to the second commonly used measure: Arterial Oxygen Saturation (SaO2).

This measure, also done on arterial blood and measured with a blood gas analyser, shows the percentage of haemoglobin molecules in arterial blood which are saturated with oxygen.

A third measure is even more commonly used, but unfortunately even less accurate in hypoxaemic patients: the Peripheral Arterial Oxygen Saturation or SpO2. This measure, taken by pulse oximeter in the periphery of the patient's body, is correlated with SaO2, but not in a linear fashion, and can be impacted by the pulse oximeter's probe placement as well as other factors affecting haemoglobin health and peripheral perfusion.

Hypoxaemia

Hypoxaemia is defined as PaO2 of less than 80 mmHg OR SaO2 lower than 95%.

Due to the high affinity of healthy haemoglobin for oxygen, drops in SaO2 lag behind decreases in PaO2, which is why the levels associated with hypoxia differ.

Table 1 *shows the correlation between PaO2 and SaO2:*

	PaO_2	SaO_2
Normoxaemia	100	98
Hypoxaemia	<80	<95
Severe Hypoxaemia	<60	<90

By the time SpO2 drops below 90, the patient is experiencing serious, potentially life-threatening hypoxaemia.

SaO2 or SpO2 below 90 should always be treated as serious and clinically relevant.

Causes of hypoxaemia include:

1. Decreased oxygen content of inspired air (FiO2). Room air contains a Fraction of Inspired Oxygen (FiO2) of 21, meaning that 21% of the room air is composed of oxygen.
2. Hypoventilation, a reduced breathing rate
3. Diffusion impairment, for example that caused by secretions filling the lung alveoli and preventing gas from diffusing into the blood that bypasses them
4. Ventilation – perfusion mismatch, such as occurs during pulmonary hypertension or asthma
5. Intrapulmonary shunt, which diverts oxygenated blood away from its regular flow into the body

Clinical signs of respiratory distress

Cyanosis springs to mind when we consider signs of respiratory distress, but in fact, it is a very late stage symptom, which only occurs when there is a significant (>5g/dL) of desaturated heamoglobin in the blood perfusing the tissues.

In order to give patients an optimal chance of returning to nomoxaemia, it is essential to recognise earlier signs of respiratory distress:

- Excessive panting
- Inability to settle / generalised distress
- Abnormally long or deep breathing
- Abnormal or excessive movement of the chest and abdomen associated with inspiration or expiration
- Standing with the elbows abducted
- Standing with the neck outstretched
- Open-mouth breathing in cats

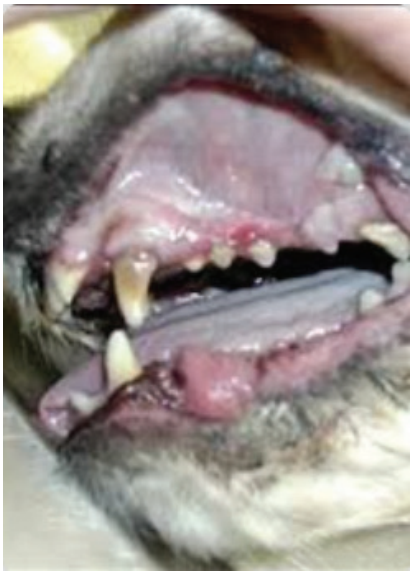


Figure 1: *Cyanosis, which is a result of deoxygenated haemoglobin in the tissues, is a very late stage sign of respiratory distress.*

Management of the respiratory distress patient

Rapid management of a patient presenting with respiratory distress is crucial to a successful outcome. Simultaneously, the underlying cause needs to be determined, while the patient is stabilised, supported and prepared for procedures.

Signalment

A patient's breed, type or age can predispose it to different conditions which could cause acute respiratory distress. Table 2 gives a summary of signalments associated with different causes of respiratory distress:

Breed	Common respiratory problem
Yorkshire terrier	Tracheal collapse Chronic bronchitis Bronchomalacia MMVD causing pulmonary oedema
English bulldog	Brachycephalic Obstructive Airway Syndrome (BOAS) Heart valve disease
Doberman pincher	DCM –causing pulmonary oedema and pleural effusion
Labrador retriever	Laryngeal paralysis
Pug	BOAS Lung lobe torsion
Cocker spaniel	DCM –causing pulmonary oedema and pleural effusion Bronchiectasis Chronic bronchitis
West highland white terrier	Pulmonary fibrosis Chronic bronchitis
Golden retriever	Spontaneous pneumothorax
Siberian husky	Eosinophilic bronchopneumopathy (EBP)
Norwich terrier	While not a brachycephalic breed, have upper airway abnormalities, including redundant supra-arytenoid folds, laryngeal collapse, everted laryngeal saccules, and a narrowed laryngeal opening
Toy poodle	Tracheal collapse Chronic bronchitis Chronic heart valve disease
Siberian husky and Alaskan malamute	Spontanious pneumothorax (Bulla and belbs)
Afghan hound	Lung lobe torsion
Young puppies	Noncardiogenic pulmonary edema secondary to electric cord injury, near drowning. Anticoagulant rodenticide Bordetella bronchiseptica pneumonia Aspiration pneumonia
Hunting dogs	Pyothorax
Young cats	Nasopharyngeal polyps Upper respiratory infection
Pointed cats (Siamese, Birman, Ragdoll)	Allergic airway disease ("asthma")
Cats	Pyothroax

Patient History

A thorough patient history is also key to discovering the cause and progression of the problem. Patient history for an animal in respiratory distress should include:

- Vaccination status
- Deworming status
- Travel history
- Are other pets in the household affected
- Is the patient in contact with animals outsode the home, for example going to daycare or a recent stay in kennels
- When did the first symptoms appear
- What was the progression of symptoms
- Does it vary in intensity between evening and morening
- Are symptoms equally present at rest and during ecercise
- Any possiblity of toxin exposure
- Any history of possible trauma
- What is the animal's exercise tolerance
- For cats, do they have a history of hairball problems
- Are any other health problems present
- What medication is the animal taking

Initial management

In addition to signalment and history, the following should be done as part of the initial management of a patient presenting with respiratory distress:

1. Recording an accurate weight to aid with correct dosage of medication

2. A hands-off exam, to include
   a. Respiratory rate
   b. Respiratory effort
   c. Breathing patterns
   d. Posture

3. A hands-on exam, to include
   a. Mucous membrane examination
   b. Auscultation

4. Oxygen supplementation

5. Establishing IV access – unless doing so will stress the animal and aggravate its condition

6. Administering an anxiolytic or sedative if appropriate, since hypoxia is truly distressing

7. Induction, once stable

8. Procedures as required to correct the cause

your pet's first choice
Omega

YOUR CLIENTS HANG ON TO YOUR EVERY WORD.

RECOMMEND NOTHING LESS THAN A **PREMIUM PET FOOD.**

- Rich in **ostrich** (min. 15%)
- High in **EPA & DHA** (min. 3 000 mg/kg)
- **Joint-supporting nutraceuticals** (fish oil, chondroitin and glucosamine)
- **Prebiotics** (dried beet pulp and inulin)
- **L-Carnitine** to promote fat metabolism
- Meticiously prepared in a **FSSC 22 000 certified** facility

PROUD MEMBER OF THE
PET FOOD INDUSTRY
ASSOCIATION OF SOUTHERN AFRICA

Available at the following veterinary wholesalers: Lakato, Midlands, VetServ and MCP

www.omegapetfoods.com

Reg No (Act 36/1947): V21407 (South Africa) and NFF2326 (Namibia)

Types of oxygen supplementation

Both invasive and noninvasive methods of oxygen supplementation exist and depending on the patient's tolerance and level of need, different options may be indicated:

Noninvasive methods

Flow-by oxygen (figure 2)

This is arguably the simplest technique. A tube connected to oxygen is simply held 2-3 cm from the patient's nostrils. If the oxygen supply is set to a flow rate of 2 – 3 l per minute, this can increase FiO2 from the 21% of ambient air to between 24 and 45%.

Flow-by oxygen is simple, well-tolerated and very useful during initial triage of the patient. It is, however, wasteful and because of this should be replaced with a more oxygen-sparing method of supplementation.

Figure 2: *Flow-by oxygen is ideal for use during initial triage, but is wasteful so should not be the long-term method of supplementation.*

Face Mask (figure 3)

Supplementing oxygen through a face mask is another very simple technique which can be used in the short term. Holding a tight-fitting mask over the patient's muzzle with an oxygen flow rate of 8 – 12 l/min, can increase FiO2 to 50-60%. The only equipment needed for face mask supplementation is a range of differently-sized masks, so this setup is relatively easy and inexpensive. Another advantage is that most of the patient's body is still free for the clinical evaluation. Unfortunately, face masks are not well tolerated by all patients, and the high flow of oxygen over the muzzle may damage the eyes, so this is also a short-term solution for most patients.

Oxygen hood (Figure 4)

An oxygen hood, constructed out of a standard Elisabethan collar and cling film, with the oxygen hose poking into the hood from the collar, is something that can be used in any veterinary practice. In addition to being practical and built out of materials any vet should have on hand, it is generally well tolerated in the short term. To use it, start by flooding the hood with oxygen at an initial flow rate of 1 – 2 l / min, reducing after a few minutes to 0.5 – 1 l/min. This will provide FiO2 of between 30 and 40%.

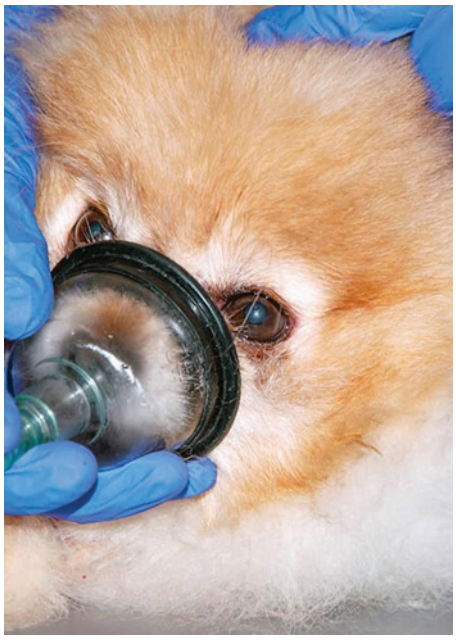


Figure 3: *Face masks can increase FiO2 to 50 or 60%, but are not well tolerated by all patients.*

A note of caution is that both CO2 and moisture can build up quickly inside the enclosed environment of an oxygen hood and become a new source of patient distress. Be sure to monitor the patient carefully and to periodically open and "refresh" the hood.

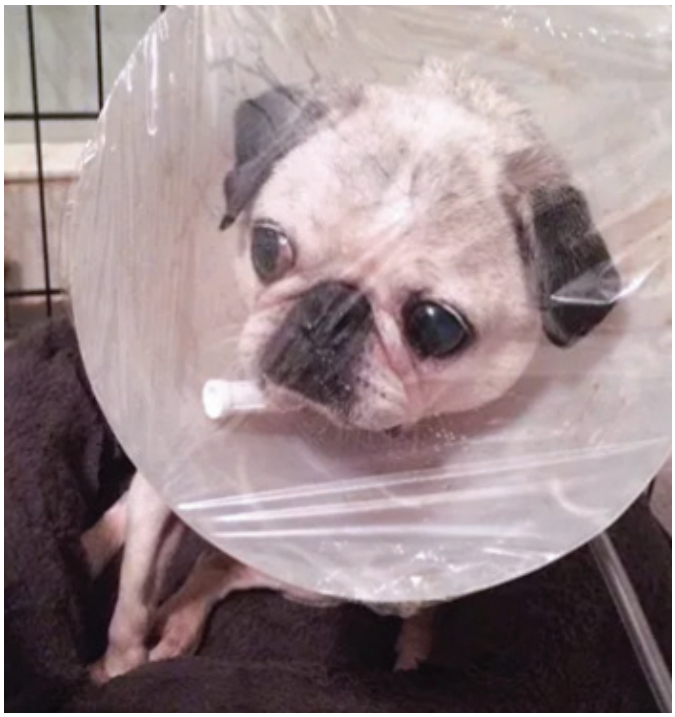


Figure 4: *Oxygen hoods can be easily constructed in any practice and are generally well tolerated. Be sure to monitor the patient for signs of distress due to CO2 or moisture build-up and to open and refresh the hood periodically.*

Oxygen cage

Oxygen cages provide customised and ongoing control of oxygen concentration, temperature and humidity, ensuring patient comfort while FiO2can be as high as 60%. Unfortunately, they are expensive, access to the patient is limited while it is inside the cage, and only small dogs and cats can comfortably fit. This makes them a less practical solution for smaller veterinary practices with limited space and equipment budgets.

Invasive menthods of oxygen supplementation

As the name suggests, invasive methods of oxygen supplementation delivers it directly to the animal. They are not as quick to set up, but lead to lower oxygen wastage in general and are more suitable to long-term therapy.

Nasal prongs (figure 5)

Nasal prongs deliver oxygen directly into the nostrils, making them ideal, especially for larger dogs that cannot fit in an oxygen cage or who would require enormous flow-rates with flow-by oxygen supplementation. This method is only beneficial if oxygen flow from the nose to the lungs is unobstructed, so it is important to determine the cause of respiratory distress before placing nasal prongs. While they are generally well tolerated by patients, they can be easily removed by pawing at the nose. The exact FiO2supplied is also difficult to ascertain.

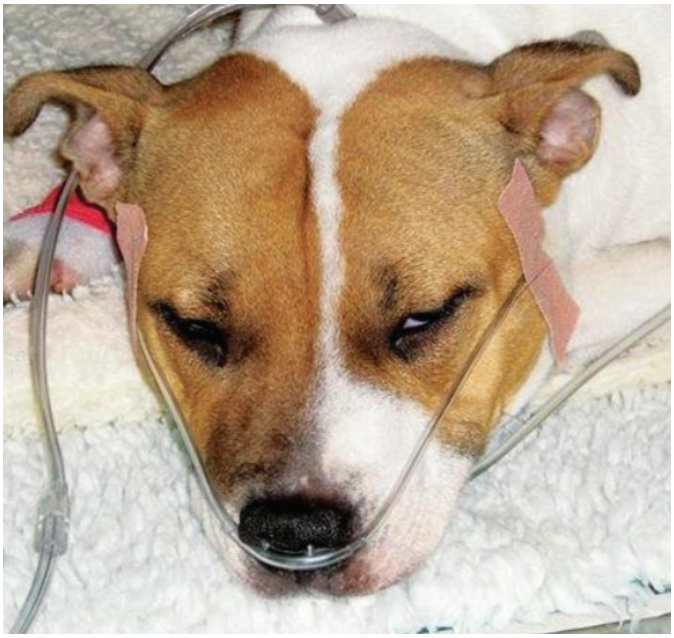


Figure 5: *Nasal prongs are easy to place, inexpensive and generally well tolerated. They are only helpful if there is open air flow from the nostrils to the lungs, though.*

Nasal and Nasopharyngeal oxygen

This method, which involves a cannula fed deeper into the nose or nasopharynx, is ideal for patients who are not spontaneously breathing through their nose or who require oxygen supplementation for longer than 24 hours. A lower flow rate is required for good effect: 50 – 150 ml/kg/min can yield a FiO2 of 30 – 70%.

Oxygen must be humidifed if it is administered using this method, however, and even then, higher flow rates can cause irritation.

Transtracheal oxygen (figure 6)

Transtracheal oxygen is indicated where there is complete upper airway obstruction either due to the presenting trauma or due to patient conformation leading to permanent open-mouth breathing, In this case, such as sometimes happens with severely brachychephalic dogs, nasally supplemented oxygen would simply be blown out of the mouth or nose again rather than being taken into the lungs. Directly supplementing oxygen into the trachea of these patients can be the most effective way of ensuring it actually reaches the lungs. However, placing and maintaining a tracheostomy tube is a skilled and labour-intensive procedure.

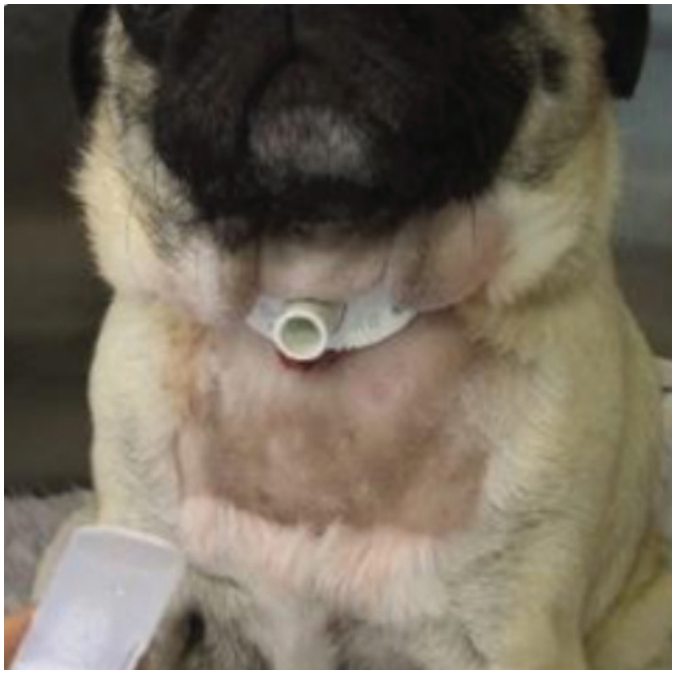


Figure 6: *Transtracheal oxyen supplementation may be the best choice for animals with upper airway obstruction or permanent open-mouth breathing. Placing and maintaining a tracheostomy tube is a special skill and a labour-intensive procedure, though.*

Anxiolytics or sedatives to use in respiratory distress patients

The sensation of not getting enough air is terrifying for humans and therefore it is logical to interpret the distress of our patients as having a similar cause. Anxiolysis can assist a respiratory distress patient in settling enough to allow for effective diagnosis and treatment. However, not all sedatives are appropriate due to cardiorespiratory suppression. A few of the useful agents and caution for their use follows:

Acepromazine (ACP)

This can be used as an anxiolytic agent in dogs which have a stable cardiovascular syste. Dosages are 0.005 – 0.02 mg/kg IV and 0.01-0.05 mg/kg IM. It has a long duration of action, from four to over six hours, which may be desirable in otherwise stable but anxious

patients. Caution, however, has to be exercised since this agent cannot be reversed and could lead to hypotension. It also has a long time to take effect – up to 15 minutes after IV administration and even longer after IM administration.

Butorphanol

This is the agent of choice in cats with respiratory distress. It is an opioid with good sedative effects and a relatively short duration of activity (as little as 1 – 2 hours). It cannot, however, be easilty reversed. Another effect of butorphanol to be mindful of is cough suppression. Depending on the cause of the respiratory distress, this may or may not be desirable. Dosage is 0.1 – 0.4 mg/kg IM or IV.

Medetomidine

This short-acting sedative can be useful, since it can be titrated to effect and reversed with atepamezole. It is contra-indicated for patients with underlying or contributing cardiovascular disease, since it causes bradycardia and other undesirable cardiovascular suppression. The dose is 0.005 – 0.01 mg/kg IV.

Midazolam

Although it is used for precisely this purpose (anxiolysis) in humans, primates and production animals, midazolam is not anxiolytic in dogs, cats or horses, and could even cause paradoxixal excitement, In addition, it can cause respriratory depression. For this reason, it is mentioned as a drug not to be used if other choices are available.

Induction of patients in respiratory distress

If a patient is still too distressed to allow the supplemental oxygen therapy to take effect, if they are at risk of complete respiratory arrest and need to be intubated, or if they need a tracheostomy tube to be placed, safe and rapid induction is required. In this case, propofol is the agenc of choice, used at 0.5 – 1 mg/kg IV, titrated to effect. Great care must be taken because, even though it is relatively safe and short acting, even propofol can cause cardiopulmonary depression in these already compromised patients.

Visualising the airway

Once initial triage has been carried out, it is essential to be able to find the cause of the problem. This is done mainly though different methods to visualise the airway:

Direct visualisation

In a sedated patient, with the aid of a laryngoscope, evaluate the upper airway fully to find all points of concern:

- Soft palate
- Ventricles
- Larynx structure and movement
- Masses
- Tonsils

Endoscopy

This is a very useful tool for visualising both upper and lower airways:

- Nose
- Mouth
- Larynx
- Pharynx
- Trachea
- Bronchus and bronchioles

Any abnormality should be sampled through biopsy, cytobrush or broncho-alveolar lavage.

Radiography

Radiographs can be used to evaluate the structure and integrity of the:

- Trachea
- Bronchi and bronchioles
- Lung parenchyma
- Peural space
- Chest wall
- Heart

Orthogonal views are needed, and if there is any suspicion of metastatic disease in the patient history, three views must be taken to ensure chest metastases are not missed.

Fluoroscopy

This real-time imaging technique is particularly beneficial if patient signalment and symptoms suggest dynamic disease such as tracheal collapse.

Computerised tomography

CT scans show far greater detail than radiography and is extremely useful to look at the nasopharyngeal area, the trachea and the thoracic cavity.

Sonography

Sonographic assessment can also be very helpful to characterise the pathology within the thoracic cavity.

Arterial blood gas measurements

While it is not a visualisation technique as such, arterial blood gas measurements can be instrumental in understanding the cause and severity of the patient's hypoxaemia, and in monitoring their recovery during treatment.

Table 3 gives normal blood gas values for dogs and cats.

	Arterial Values	Venous Values
DOGS[1]		
pH	7.395 ± 0.03	7.352 ± 0.02
PO_2(mm Hg)	102.1 ± 6.8	55 ± 9.6
PCO_2(mm Hg)	36.8 ± 2.7	42.1 ± 4.4
HCO_3 (mmol/L)	21.4 ± 1.6	22.1 ± 2
BE (mmol/L)	-1.8 ± 1.6	-2.1 ± 1.7
CATS[2]		
pH	7.34 ± 0.1	7.30 ± 0.08
PO_2(mm Hg)	102.9 ± 15	38.6 ± 11
PCO_2(mm Hg)	33.6 ± 7	41.8 ± 9
HCO_3 (mmol/L)	17.5 ± 3	19.4 ± 4
BE (mmol/L)	-6.4 ± 5	-5.7 ± 5

Table 3: *Normal Arterial and venous blood gas values for dogs and cats.*

MALASEB™
MEDICATED SHAMPOO

MEDICATED SHAMPOO
Reg. No. G4202 Act 36/1947

MEDICATED SHAMPOO FOR CATS AND DOGS.

CONTAINS CHLORHEXIDINE AND MICONAZOLE IN SHAMPOO.

Malaseb™ is registered to treat:

- *Hotspots*
- *Dry itchy skin*
- *Symptoms associated with ringworm*
- *Skin conditions associated with bacterial and yeast infections*

DOGS

Use twice a week when the infection is active, then once every 1-2 weeks when the infection is under control.

CATS

Use twice a week for ringworm treatment. Treatment may be required for 8-12 weeks.

CONTACT: **RINALDO BEKKER**
Agent/Sales Consultant
+27 (0) 63 113 3942
rinaldobekker.ah@gmail.com

good chemistry

AECI® ANIMAL HEALTH

A quick guide to some common causes of respiratory distress

Upper airway

Upper airway causes of respiratory distress can be due to abnormal structure or function of the nose, mouth, larynx, pharynx, or trachea – basically anywhere between the nostrils and the main bronchi. Tell-tale signs of upper airway involvement is loud, noisy breathing.

Common differential diagoses are:

- Brachycephalic Obstructive Airway Syndrome
- Tracheal collapse
- Laryngeal paralysis
- Foreign bodies in the upper airway
- Masses or swellings obstructing the upper airway

A quick guide to managing hypoxia due to upper airway disease

1. Supplement oxygen
2. Reduce anxiety with ACP or butorphanol
3. Intubate / do a tracheostomy depending on the status of the airway
4. Apply a cold-water wash to the upper airway
5. Decrease inflammation of the upper airway – regardless of inciting cause, the struggle of trying to breathe is likely to have caused congestion and inflammation. In the case of an allergic reaction or response to an insect bite or sting, this may be all that is required to support the patient's recovery.
6. Administer steroids if longer-term anti-inflammatory action is indicated. Useful steroids include:
   a. Dexamethazone
   b. Methylprednisolone sodium succinate
   c. Prednisolone
   d. Flumethasone

Lower airway

These causes of respiratory distress are found in the bronchi and bronchioles. Animals will often show that the lower airway is affected by having expiratory dyspnoea or increased effore on exhalation – giving an abdominal push on exhalation is typical. On auscultation, increased lung sounds or wheezes may be heard as air struggles to pass through narrowed airways.

Differential diagnoses to consider:

- Asthma
- Bronchopneumonia
- Bronchitis

A quick guide to managing hypoxia due to lower airway disease:

1. Supplement oxygen
2. Use a bronchodilator:
   a. Terbutaline
   b. Theophylline
   c. Salbutamol
3. Administer steroids to control the inflammation. The same agents as for upper airway disease can be used.

Pulmonary Parenchyma

This type of respiratory distress is caused by fluid or solid matter filling the alveoli and interstitial spaces, interfering with blood-gas exhange. Breathing will be quiet but laboured, with synchronous chest movement. On auscultation moist lung sounds are heard and a heart murmur may be present.

Differential diagnoses to consider:

- Pneumonia
- Oedema (cardiogenic or non-cardiogenic)
- Lung lobe contusion
- Neoplasia

A quick guide to managing hypoxia due to disease of the pulmonary parenchyma:

1. Supplement oxygen
2. Provide anxiolysis / sedation with butorphanol
3. Treat the effusion (antibiotics in the case of infection, furosemide in case of oedema)
4. Intubate and ventilate

Pleural space

This includes conditions affecting and altering the pleura, mediastinum or diaphragm. Affected animals will also breathe quietly with increased effort, but a tell-tale sign of pleural space disease is that the movement of the chest wall and the abdomen will be asynchronous (distinguishing it from diesases of the lung parenchyma) – during inhalation, the chest will move out, but the abdomen paradoxically inwards, and during exhalation the chest will contract but the abdomen will expand. On auscultation, reduced lung sounds are audible.

Differential diagnoses include:

- Pleural effusion
- Mediastinal masses
- Pneumothorax
- Diaphragmatic hernia

A quick guide to managing hypoxia due to disease of the pleural space:

1. Supplement oxygen
2. If air or fluid is present in the pleural space, drain it (dorsal for air, ventral for fluid)

Chest wall

Usually traumatic, damage to the ribs and muscles of the chest wall can also cause hypoxia due to the animal being unable to fully inflate its lungs. Breathing is typically fast and shallow, and often the site of injury can be seen, pinpointed by an area of abnormal chest movement.

Rib fractures and intercostal muscle tears are the two main differential diagnoses.

A quick guide to managing hypoxia due to chest wall trauma:

1. Supplement oxygen
2. Provide analgesia – a lot of the dyspnoea may be simply due to pain
3. If air you have an open wound, plug it. You may need an emergency thoracocentesis to remove excess air in addition

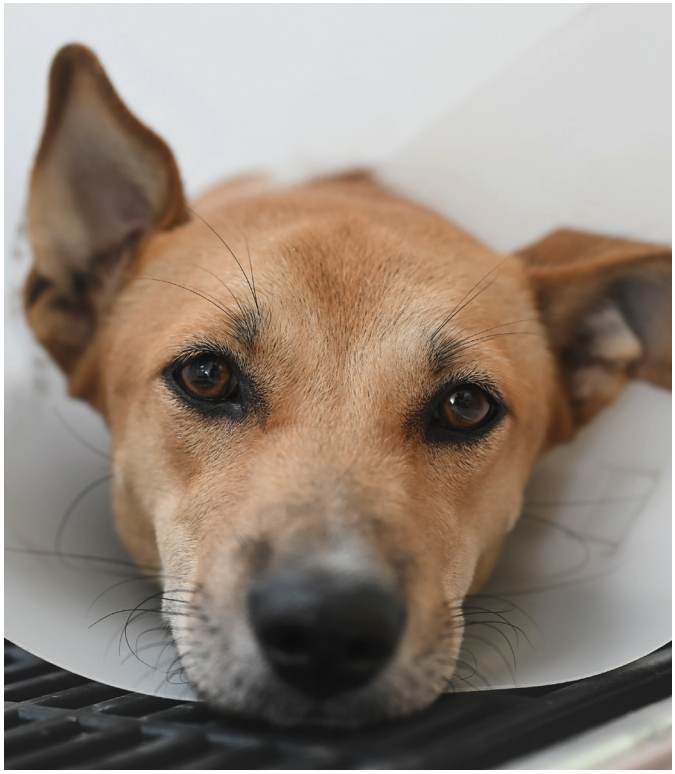
to plugging the hole so that more air does not infiltrate with every breath.

4. Proceed to surgery once the patient is stable enough, if indicated.

Bear in mind that some cases may have more than one conributing cause, in more than one part of the airway, which causes sudden decompensation. However, following these steps, you should be able to get your respiratory distress patients breathing freely again.

This article was adapted from a CPD lecture given to veterinarians from Pretoria and surrounding areas as part of a monthly lecture series hosted by Valley Farm Animal Hospital with support from generous sponsors. The lectures are open to CCS vets as well as vets in private practice, and aim to increase confidence in handling a variety of conditions in general practice.

Our thanks to Dr Breytenbach and to Valley Farm Animal Hospital for sharing their expertise with our readers. Veterinarians who would like to attend the monthly lecture series can contact admin@valleyfarmvet.co.za for more information.



The Role of Physiotherapy in Veterinary Lung Rehabilitation

an interview with Amy Louw

Amy Louw

South African vets are in the fortunate position of having a team of paraveterinary professionals available to help us with multimodal patient management.

Veterinary physiotherapy is one of the registered paraveterinary professions which can support conventional medical or surgical treatment.

But how can physiotherapy help respiratory patients? And if you would like to refer one of your patients to a physiotherapist for part of their treatment, what can you and your client expect?

Vet360 spoke to Amy Louw, a human a veterinary physiotherapist and one of the main lecturers in the BSc Veterinary Physiotherapy degree at Equine-Librium College, to find out more.

Vet360: What exactly is respiratory physical rehabilitation?

Amy: Respiratory physical rehabilitation uses specific physiotherapy techniques to improve lung function, facilitate the removal of secretions from the lung fields, improve gaseous exchange, and normalise respiratory patterns.In addition, veterinary physiotherapists can assist patients by implementing a controlled exercise program to improve exercise tolerance once the acute condition has resolved.

An equine patient being nebulised for a minor respiratory infection.

Vet360: Which are the most common respiratory conditions that you treat in dogs, cats and horses?

Amy: Although cardiorespiratory patients do not typically make up a large percentage of our case load, a wide range of animal patients can benefit from receiving cardiorespiratory physiotherapy.

These include in-hospital patients that may be compromised due to respiratory illness, such as pneumonia; or those that are recumbent due to other disorders and have developed secondary respiratory illnesses.

In addition, animals that suffer from seasonal allergies or chest infections will benefit from receiving respiratory physiotherapy during their recovery. The animal always needs to be under veterinary care prior to starting with respiratory physiotherapy treatment.

The most common respiratory conditions we can be involved in the management of include: Pneumonia, Asthma, Brachycephalic obstructive airways disease, Respiratory compromise from allergies and Inflammatory airway disease

Vet360: How many sessions does a typical patient come to you for?

Amy: Animals with acute respiratory conditions are generally unwell and require very short, but frequent sessions, so ideally once or twice a day for 5-7 days initially. The animal will continuously be reassessed and frequency determined by the specific patient's needs.

Animals that are otherwise healthy, generally require 2 – 3 sessions a week, once again guided by their response to treatment.

Vet360: What modalities do you use to treat respiratory patients?

Amy: Well, it really depends on the referring complaint and the respiratory assessment, which is done during the first physiotherapy session.

A full respiratory assessment includes: evaluation of the patient's current state – what they are physically able to do; what other injuries do they have; what medication are they on; having a look at chest X-rays where applicable; and discussing the management of the patient with the primary or referring veterinarian.

Auscultation and palpation of the lung fields will then be done to determine where there is reduced airflow and/or secretions. Once the assessment is complete, the physiotherapist will determine which techniques and modalities are appropriate to use for that specific case.

The following techniques can all be used with respiratory patients: ***Manual therapy,*** including percussions and vibrations. Percussions involves the therapist rhythmically "clapping" with cupped hands over the patient's chest wall. The aim of this is to loosen secretions within the chest cavity. Vibration is when the therapist shakes their hands while remaining in contact with the chest wall (usually on expiration), which assists with moving the dislodged secretions into larger airways. A mechanical vibration device can also be used with caution.

Nebulisers are used for inhalation therapy to deliver specific medications (veterinary prescribed) to aid in the treatment of respiratory dysfunction, such as asthma or pneumonia. There are both small animal and equine nebulisers.

Postural drainage – small animal patients can be placed in particular positions to allow gravity to assist with the drainage of secretions from the different lung segments.

Coughing is important to clear the secretions from the lungs once they have been mobilised. In small animal patients, physiotherapists can assist the animal to cough by applying gentle pressure to the trachea.

Education – physiotherapists can educate owners regarding environmental changes that can assist with the resolution of the respiratory disorder.

Exercise is the most efficient way of increasing perfusion and improving respiratory function. As soon as it is safe for the animal to perform gentle exercises (short standing and walking sessions), the physiotherapist will encourage the animal to move while continuing to monitor their respiratory condition.

In small animals that are non-ambulatory, passive range of movement and changes of body position will assist with preventing further complications from being recumbent.

Vet360: How does combining physiotherapy with traditional medical management benefit patients?

Amy: Patients that receive physiotherapy for cardiorespiratory dysfunction generally have fewer long-term complications, are able to return to function faster, require less time in hospital, and develop fewer secondary complications arising from being immobile.

Vet360: Where can vets find a veterinary physiotherapist in their area?

Amy: Equine-librium has clinics throughout South Africa. Contact details of the specific clinics can be found on the Equine-librium website: www.equine-librium.co.za, alternatively on info@equine-librium.co.za

In addition a list of SAVC registered and authorised Veterinary Physiotherapists can be found on the South African Animal Physical Rehabilitation Associations website: www.saapra.co.za

The Many Facets of Hypertension

Joan Capuzzi, VMD
dvm360, dvm360 August 2022, Volume 53, Issue 8

High blood pressure silently weaponizes flowing blood, turning key organs into targets. By detecting the often subtle clues to its havoc, veterinarians can recognize cats and dogs at risk.

The cardiovascular system supplies the body with life-sustaining blood by generating adequate intravascular pressure to perfuse the tissues. If the pressure slumps, the organs may not receive suitable perfusion. But if it swells, they can suffer barotrauma.

The rate and force at which blood courses through the body combine with the diameter and elasticity of the vessels to create blood pressure (BP). Systolic BP (the maximal pressure against the walls of the arteries) is produced by cardiac contraction. Diastolic BP (the minimal pressure against the arterial walls) occurs as the heart refills.

How high is high?

Studies show systolic BP (SBP) ranges from 131 +/-20 to 154 +/-31 in clinically healthy dogs[1-5] and runs slightly lower in healthy cats.[6,7] Elevated pressures can cause target organ damage (TOD) that, according to the Veterinary Blood Pressure Society, tracks along with the severity of hypertension. Pressures above 150/95 pose risk for end-organ injury and may warrant intervention as follows[8]:

- Normotensive (minimal TOD risk): SBP < 150 mm Hg
- Prehypertensive (low TOD risk): SBP 150-159 mm Hg
- Hypertensive (moderate TOD risk): SBP 160-179 mm Hg
- Severely hypertensive (high TOD risk): SBP > 180 mm Hg

Target organs

The organs most vulnerable to hypertensive harm are the eyes, brain, heart, and kidneys.

Eyes

Ocular conditions, including retinal detachment, papilloedema, hyphaema, and glaucoma, are the most common hypertensive injuries in cats. Prevalence rates for hypertensive retinopathies and choroidopathies in cats are as high as 100%[9-12] but are also significant in dogs.[13,14] Retinal detachment is the most frequently observed finding in both species, and acute onset of blindness may be the presenting complaint.[15-17]

Brain/cerebrovascular system

Hypertensive encephalopathy can occur in dogs[18] and cats.[19] Clinical signs, possibly related to haemorrhage and/or infarct, include seizures, altered mentation, weakness, and vestibular problems.[20,21] Neurologic issues have been reported in up to 46% of hypertensive cats.[10,19] They are more likely to occur with a sudden jump in BP, a pressure exceeding 180 mm Hg, or both; they often resolve if hypertension is treated early.[22]

Heart/cardiovascular system

Hypertension raises the load against which the heart must contract, sometimes leading to thickening of the heart muscle and cardiomegaly. In cats and dogs, this most often manifests as left ventricular concentric hypertrophy.[23] In cats, sustained hypertension causes cardiomyopathy at a rate of 60%[19] but rarely progresses to heart failure.[10,20,24]

Systolic heart murmurs and arrhythmia may be detectable on examination, with some patients presenting with exercise intolerance and—less frequently—epistaxis[25-27] associated with vascular hypertensive injury.

Kidneys

Elevated systemic BP can damage the delicate vascular beds in the nephrons. Renal vessels then constrict, and glomerular filtration drops, resulting in sodium retention and—in a destructive feedback loop—water retention that can further elevate BP.[28]

Increased glomerular pressure leads to sclerotic changes in the glomeruli, enabling protein to leach out. Proteinuria likewise hastens progression of chronic kidney disease[28,29] and has been

Photo by Kelly Ehrmann

associated with shorter survival times in hypertensive cats[30,31] and dogs.[32,33] In dogs, the severity of hypertension was shown to correspond directly to the magnitude of proteinuria and shortened survival times.[34-36]

Although it ravages sensitive vascular networks in key organs, hypertension usually does not announce itself with a clinical bang, such as a nosebleed or a seizure, sudden blindness, or even polyuria/polydipsia. Hypertension is alarmingly silent, and its damage to target organs occurs in increments over time.

Triad of hypertension

Hypertension is categorized into 3 types: situational, primary, and secondary.

- **Situational hypertension** is fleeting and brought on by environmental stressors. The psychological jolt generated by a visit to the veterinarian can prompt the autonomic nervous system to drive up BP. Anxiety-induced rises in BP can lead to erroneous diagnosis and unnecessary treatment.

- **Primary (or "essential") hypertension** occurs without underlying triggers.

- **Secondary hypertension**, which accounts for more than 80% of cases in dogs and cats,[5,15,37] is characterized by persistently

elevated BP concurrent with precipitating disease (or therapeutic agent).

Aetiologies include acute[35] and chronic kidney disease,[31,38] hyperthyroidism,[38] hypothyroidism,[32] hyperadrenocorticism,[39,40] hyperaldosteronism,[41] phaeochromocytoma,[42] and diabetes.[43]

Of these, chronic kidney disease has the strongest association with hypertension: both cause and effect for one another, the 2 are inextricably linked. Among other things, diseased kidneys are unable to properly couple the excretion of salt and water with changes in blood volume. And they inappropriately activate the renin-angiotensin-aldosterone system (RAAS), which further spikes blood volume.

The main iatrogenic causes of secondary hypertension are glucocorticoids,[39,40] mineralocorticoids,[41] and erythropoietin.[44]

Tallying tension

The American College of Veterinary Internal Medicine (ACVIM) advocates BP screenings every 6 months for animals with predisposing underlying diseases.[1] Readings should also be performed in geriatric (over age 9 years) and/or obese cats and dogs, as well as those exhibiting clinical signs compatible with hypertensive end-organ damage, such as retinal haemorrhage, ataxia, dyspnoea, and proteinuria.

Measurements should be conducted in a quiet room with the owner present, if possible. The patient should not be sedated and allowed to acclimatise to the examination room for 5 to 10 minutes before testing, and minimally restrained in sternal or lateral recumbency.

Although direct BP measurement—which involves arterial catheterisation—is the gold standard, it is not practical. In the clinical setting, noninvasive, indirect techniques are used: Doppler devices utilise ultrasound waves that detect blood flow; oscillometric units monitor arterial pulse waves.
Proper cuff selection is key to obtaining accurate measurements. For Doppler and oscillometric techniques, the cuff width should be 30% to 40% of the circumference of the cuff site (leg or tail).

ACVIM recommends obtaining 5 to 7 values and discarding the first. Measurements should be repeated until values are consistent. Values are then averaged into a single number.

Readings are not always straightforward, and can be altered by measurement method, operator experience, patient position, gender, age, breed, and temperament.[45-50] Ageing dogs have been shown to experience a 1 to 3 mm Hg per year, and cats 0.4 +/- 0.1 mm Hg per 100 days.[51]

In sight hounds (ie, greyhounds), pressures run 7 to 20 mm Hg higher than in other canine populations,[47,52] attributed to situational rather than true hypertension.

Animals deemed truly hypertensive should be retested at least twice over 4 to 8 weeks. For severe hypertension, however, testing should be completed within 2 weeks to meet the risk of target organ damage. If organ injury, such as retinopathy, is already present, treatment should be initiated after the first measurement session, with results confirmed over subsequent visits.

Easing the pressure

Where secondary hypertension is present, the underlying disease should be treated first. Although it may lower BP and render the patient's hypertension more amenable to treatment, this alone will rarely make the patient normotensive. Medications are usually required:

Angiotensin-converting enzyme (ACE) inhibitors (ie, enalapril, benazepril): RAAS inhibitors, antiproteinuric, usually initial drugs of choice in dogs,[53] less effective in cats.[37,54]

Angiotensin II receptor blockers (ie, losartan, telmisartan): RAAS inhibitors, antiproteinuric, first-line antihypertensive agents in dogs.[55,56]

Aldosterone antagonists (spironolactone): RAAS inhibitors, antiproteinuric, potassium-sparing diuretic, effective in dogs and cats.[57]

Calcium-channel blockers (amlodipine): Inhibit vasoconstriction; treatment of choice in cats, used as monotherapy or combined with ACE inhibitors or angiotensin-receptor blockers[9,10,19]; in dogs, used in multiagent therapy only, combined with ACE inhibitors.[58]

α-adrenergic blockers (prazosin, phenoxybenzamine): Produce vasodilation; adjunctive therapy for dogs when initial antihypertensive treatment is inadequate.[42]

β-blockers (atenolol): Slow heart rate, lower BP; add-on treatment for dogs and cats when initial antihypertensive agent falls short of desired effect.[9,19,59]

Direct vasodilators (hydralazine, acepromazine): less commonly used to manage hypertension, except for emergency situations.[20]

Thiazide diuretics (hydrochlorothiazide): used in a minority of patients in whom volume expansion is clinically apparent.[10]

Hypertension related to certain conditions may be best addressed using specific types of agents, such as α-adrenergic and β-adrenergic blockers in cases of phaeochromocytoma or aldosterone-receptor blockers in animals with adrenal tumors.[21]

Medical management of hypertension can be augmented by weight loss in obese patients, and dietary salt restriction. In managing hypertensive patients, the goal is to achieve gradual drops in systolic pressure to less than 170 mm Hg.

BP checks should be performed every 2 weeks, along with corresponding changes in dosages and/or drugs. Once regulated, SBP should be rechecked every 3 months and blood work performed annually.

***Joan Capuzzi, VMD**, is a small animal veterinarian and journalist based in the Philadelphia, Pennsylvania area.*

References available online - www.vet360.vetlink.co.za

CPD Questions

www.bit.ly/vet360-apple https://bit.ly/vet360cpd

Go to www.vet360.vetlink.co.za or download the new Vet360 App to answer the questions CPD reserved for Vet360 subscribers (R360 per annum) Call 012 346 1590 or visit www.vet360.vetink.co.za to sign up

1. What is the relationship between SPo2 AND SaO2?
a. Linear
b. Inversely
c. Directional but not linear
d. Exponential

2. If the SpO2 reading is <80% , what is the SaO2?
a. SaO2 of 98 mm HG
b. SaO2 of 99 mm HG
c. SaO2 of 100 mm HG
d. SaO2 of <95 mm HG

3. PaO2 is a function of:
a. Partial pressure of oxygen in the alveoli. This alveolar partial pressure of oxygen is the driving force for the diffusion of oxygen across the alveolar membranes, through pulmonary capillary walls, and into the arteriolar blood flow and erythrocytes for transport throughout the body into peripheral tissues.
b. A PaO2 test measures partial pressure of oxygen, therefore the oxygen pressure in arterial blood. The PaO2 reflects how well oxygen is able to move from the lungs to the blood.
c. is the concentration of oxygen in the gas mixture.
d. None of the above

4. What is the definition of hypoxemia?
a. Is defined as a decrease in the partial pressure of oxygen in the arterial blood , measured in mm HG or saturation less than 95%.
b. Partial pressure of oxygen less than 80 %
c. Saturation less than 95 mm Hg
d. None of the above

5. Cyanosis can only be seen if:
a. If the patient is anemic
b. If the patient is open mouth breathing
c. If you have a V/Q mismatch
d. If the deoxygenated hemoglobin is more than 5gm/dl

6. Tight fitting Facemask will increase FiO2 with %?
a. 30-40%
b. 40-50%
c. 50-60%
d. >60%

7. DD's for upper airway disease?
a. Cat with snuffles
b. Labrador with laryngeal paralysis
c. Cat with asthma
d. A and B

8. DD's for pulmonary disease?
a. MMVD , stage C , in a 7 year old Maltese with crackles in the lungs
b. MMVD , stage A , in a Doberman pincher
c. MMVD , Stage B1 in a Yorkie , with a tracheal collapse
d. None of above

9. What is the difference between A lines and B lines on a TFAST?
a. Horizontally oriented hyperechoic A-lines are equidistant lines caused by reverberation artifacts of the lung margin, or reverberation artifact from the normal pulmonary–pleural interface. While B lines are reverberation artifacts originating from the visceral pleura lines appear as hyperechoic vertical lines extending from the pleural line to the edge of the far field image
b. A-lines are also known as shred signs and B lines are known as fractal signs.
c. There is no such terminology in TFAST
d. All of above

10. How does the SpO2 reader get the reading?
a. Whole blood is passed between two electrodes through an aperture so narrow that only one cell can pass through at a time. The impedance changes as a cell passes through. The change in impedance is proportional to cell volume, resulting in a cell count and measure of volume.
b. Based on competitive enzyme-linked immuno-sorbent assay technology
c. A and B
d. The probe selectively reads the (pulsatile) arterial blood flow of the tissue; oxyhaemoglobin absorbs more infrared light and deoxyhaemoglobin absorbs more red light at different wavelengths. The pulse oximeter then gives a reading based on the difference between these two absorbances.

Exercise Induced Collapse in Dogs

Charné Rossouw-Claassen
Division Manager, Animal Genetics

Exercise induced collapse (EIC) is a genetically inherited disorder that was first described in the Labrador retriever. Most affected dogs will have their first episode before the age of 3 and will typically start off by experiencing weakness in their rear limbs.

Should the dog continue to exercise, the symptoms will continue to worsen until the exercise is halted. The condition is not painful and will typically resolve after 5 – 25 minutes of rest.

The condition has been linked to a recessively inherited mutation in the dynamin-1 (DNM1) gene. This gene, which is almost exclusively expressed in the spinal cord and brain, helps regulate the formation of endocytic vesicles. These vesicles contain neurotransmitters which helps with synaptic communication.

The single nucleotide change results in a change in amino acid, which leads to fewer endocytic vesicles being produced. It is believed that this reduced potential for synaptic communication results in a reversible loss of the dog's motor functions.

EIC usually presents after 5 – 10 minutes of strenuous exercise, such as hunting, running or fetching. Clinical signs may include:

- Wobbly gait
- Dragging the back legs while running or walking
- Hypermetria (picking the feet up too high)
- Stiff front legs while collapsed
- Collapsing while running
- Temporary inability to move head and limbs after exercising
- No other symptoms between collapses
- Quick recovery (5 – 25 minutes)
- Confusion (rare)
- Seizures and death (rare)

If it's suspected that a dog suffers from EIC, a genetic test is available at several laboratories to confirm diagnosis. To rule out any other underlying conditions, a full blood count and biochemical profile can be performed to confirm that the dog's organs are properly functioning.

Thyroid hormone levels can also be tested as well as screening for other muscle diseases in the dog. A special heart monitor can be fitted to the dog for a day or two to track heart rhythms to rule out a heart problem.

The most effective treatment for EIC is to avoid activities that are known to trigger the episodes, especially in hot, humid weather. Dogs should be given moderate exercise and monitored for signs of an episode. Exercise should be stopped at the first sign of symptoms and water used to bring down the dog's body temperature.

In some studies, reducing the dog's level of excitement/ exercise drive (such as neutering or administering phenobarbital) has helped to reduce the amount of episodes. It does however, not directly treat the cause.

The frequency of episodes will also reduce as the dog ages and becomes more docile. Owners can manage their dog's episodes by remaining observant and taking their pet for regular check-ups if the dog is on any prescription medication.

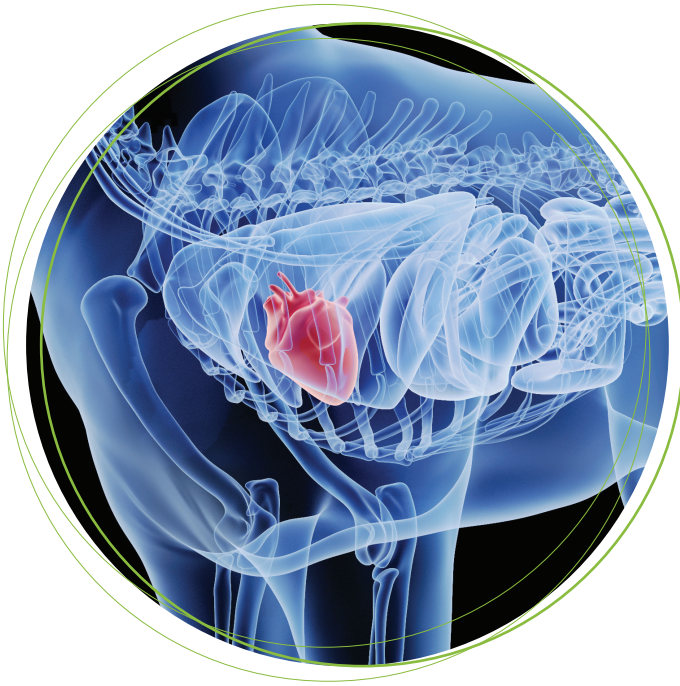
Although there is currently no cure for EIC, future generations can be guarded against it by not breeding carriers to carriers. It is therefore important to advise breeders to test their stud animals before starting to breed with them to ensure clear, healthy puppies.

References:

- Patterson, E. et. al. 2008 A canine DNM1 mutation is highly associated with the syndrome of exercise-induced collapse. Nature Genetics Volume 40(10): 1235- 1239.
- Collapse During Exercise in Labrador Retrievers 2009. PetMD Editorial https://www.petmd.com/dog/conditions/cardiovascular/c_dg_exercise_induced_collapse_labrador_retriever
- Daynamin-1 associated exercise-induced collapse 2016. College of Veterinary Medicine. https://vetmed.umn.edu/research/labs/canine-genetics-lab/genetic-testing/exercise-induced-collapse

Pulmonary Hypertension and Cor Pulmonale

Quick Guide for the Practitioner



Prof Johan Schoeman BVSc, MMedVet(Pret), PhD(Cantab), DSAM, Dipl. ECVIM
Department of Companion Animal Clinical Studies
Faculty of Veterinary Science
University of Pretoria

Cor Pulmonale is defined as right-sided heart failure caused by pulmonary disease. It is typified by radiographic or echocardiographic evidence of right ventricular overload.

By definition, pulmonary hypertension (PH) must be present in cor pulmonale for the right heart to fail. There are many different causes of PH and pulmonary vascular obstruction secondary to heartworm (*Dirofilaria immitis*) is the most common worldwide cause in dogs. Since this disease is essentially absent in South Africa, we have to investigate other potential causes of PH in our dogs. Hence, in our setting, the most common cause of PH would most likely be pulmonary venous hypertension secondary to severe chronic mitral valve disease (CMVD). Practitioners will likely fail to consider the effect of left heart failure and the attendant markedly increased pulmonary venous pressure on the right heart, unless they have been sensitized regarding the existence of this form of pulmonary hypertension - so called post-capillary PH.

So, how is pulmonary hypertension classified and how does it develop?

It is helpful to consider that the right ventricle is the sole source of preload for the left heart. In other words, the whole of the right ventricular output percolates through the lungs and ultimately ends up in the left atrium. Accordingly, any back pressure in this very low pressure system will increase right ventricular afterload. Unlike the thick-walled left ventricle, the thin-walled right ventricle is not designed to handle a marked increase in ventricular afterload. In fact, whilst the left ventricle is handling a regular afterload pressure of 120 mmHg, the right ventricle is typically handling peak systolic pulmonary pressures of only 20 mmHg, i.e. approximately 6 x less pressure. Consequently, when this pressure increase to values between 30 - 50 mmHg it is classified as mild PH, whilst pressure > 50 - 70 mmHg is classified as moderate PH and pressures > 70 mmHg is classified as marked PH.

PH can simply be classified based on the anatomical aetiology as pre- and post-capillary or better still, as a five group system based on the underlying pathologic process. This system was originally developed for people, but has recently been amended for veterinary aetiologies (Table 1). In short, the five groups are: Group I - pulmonary arterial hypertension due to arteriolar disease; Group II - pulmonary venous hypertension due to left heart disease; Group III - chronic lung disease and/or hypoxia; Group IV - thromboembolic disease; Group IV - from unclear or multifactorial aetiologies.

To understand the pathophysiology of PH, one first has to appreciate the fact that the pulmonary vascular bed is a low-pressure, low-resistance, high-capacitance system. Upon leaving the right ventricle, the blood flows through the pulmonary artery (PA) into a network of thin-walled pulmonary arteries, capillaries and veins before entering the left atrium via a number of pulmonary veins. As a result, PA pressure is determined by, *inter alia*, RV output (aka pulmonary blood flow), pulmonary vascular resistance and, last but not least, pulmonary venous pressure. Following on from the above, pulmonary hypertension develops when there is an imbalance

between pulmonary arterial vasoconstriction, vasodilation, platelet activation and smooth muscle cell proliferation.

In this regard, vasoconstriction is induced by alveolar hypoxia, endothelin-1and serotonin. Conversely, prostacyclin and nitric oxide (NO) are potent vasodilators. Nitric oxide is also an inhibitor of platelet aggregation induced by aforementioned serotonin as well as an inhibitor of vascular smooth muscle proliferation. Once formed, NO exerts its vasodilatory effect through the activation of cyclic guanosine monophosphate (cGMP). Ultimately, this vasodilation induced by cGMP is limited through inactivation of cGMP by phosphodiesterase-5 (PDE5) isoenzymes. Because of this, much of our therapy aims to block the PDE 5 isoenzymes - see treatment later.

How is pulmonary hypertension diagnosed?

Most canines with PH are middle-aged to older small breed dogs, concordant with the most common aetiologies of PH such as chronic mitral valve disease (Group II) and chronic small airway disease (Group III). In addition, terrier breeds may be over-represented due to their predisposition to chronic pulmonary fibrosis (Group III). Since PH ultimately cause reduced cardiac output, exercise intolerance and syncope are often noted by clients, in addition to coughing and tachypnoea. Physical examination may reveal the presence of ascites, crackles, wheezes, harsh respiratory sounds and left- or right sided murmurs in the majority of cases. An abnormally loud or split second heart sound (S2) may the auscultated by the astute clinician, owing to slightly later closer of the pulmonic valve at the end of systole.

However, the ultimate diagnostic tool in small animal practice is echocardiography because cost and availability preclude the use of right heart catheterization and the direct measurement of PA pressure. On account of this, PA pressure has to be *inferred* by determining either tricuspid valve regurgitant(TR) flow or pulmonic valve insufficiency(PI) flow. In the absence of pulmonic stenosis (a congenital condition, predominantly diagnosed in young dogs), the regurgitant velocities through the valves of the right heart allows estimation of PA pressure through the use of the modified Bernoulli equation, i.e. pressure gradient = 4 x (peak velocity)^2. As such, a peak TR velocity of >2.8 m/sec (peak TR pressure gradient >31 mmHg) and a peak PI velocity of > 2.2 m/sec (peak PI pressure gradient > 19 mmHg) strongly suggest PH. In reality, it would be wise to consider the following caveats: the accuracy of these predicted pressures depend upon operator skill, degree of TR and PI, patient compliance and physiological factors such as excitement and RV systolic function.

Moreover, in the absence of doppler estimations of TR and PI, other echocardiographic findings such as RV concentric or eccentric hypertrophy, RA enlargement, septal flattening and potential RV systolic dysfunction are highly indicative of PH.

How do we treat and ultimately achieve vasodilation in pulmonary hypertension?

Since PH can develop from numerous causes, treatment should be aimed at the specific underlying aetiology, the scope of which is beyond this short communication. In humans, PH can be treated with endothelin antagonists and prostacyclin analogs. However, in dogs PDE-5 inhibitors such as sildenafil, tadalafil and vardenafil are commonly prescribed. They increase vasodilation by increasing cGMP and ultimately NO concentrations in the lung vasculature - refer back to the development of PH. Sildenafil is well-tolerated in dogs at 1-2 mg/kg PO q8-12 hours. In addition, the PDE-3 inhibitor, Pimobendan, has also been demonstrated to reduce measurable PH over time.Never therapies have also been evaluated in dogs. These include the inhibitor of platelet-derived growth factor, Imitanib. Six dogs with congestive heart failure have been treated with Imitanib at 3 mg/kg q 24 for 30 days and they showed a consequent reduction in TR velocity, left atrial size and plasma atrial natriuretic peptide concentrations.

In sum, PH should be considered in dogs with CMVD, since up to 40% of these patient will ultimately develop concomitant pulmonary hypertension, especially in those presenting with syncope in addition to their clinical signs referable to increased left heart back pressure, i.edyspnoea and pulmonary crackles. To be specific, the presence of a TR pressure gradient of > 55 mmHg was a significant predictor of poor outcome[1].

Table 1: *Classification of Pulmonary hypertension*

1. **Pulmonary arterial hypertension (PAH) due to pulmonary arteriolar vascular disease**
   - Pulmonary vascular parasitic disease
     - *Angiostrongylus vasorum* (French heartworm)
     - *Dirofilariaimmitis* (heartworm disease)
   - Congenital systemic-to-pulmonary shunts
     - Arterial septal defect (ASD), Patent ductus arteriosus (PDA), Ventricular septal defect (VSD)
   - Necrotizing vasculitis / arteritis
   - Idiopathic
2. **Pulmonary hypertension with left heart disease (pulmonary venous hypertension)**
   - Mitral valve disease • Myocardial disease
   - Miscellaneousleft-sided heart disease
3. **Pulmonary hypertension with pulmonary disease / hypoxemia**
   - Chronic obstructive pulmonary disease
   - High-altitude disease
   - Interstitial pulmonary fibrosis
   - Neoplasia
   - Reactive pulmonary artery vasoconstriction (from pulmonary oedema and hypoxemia)
   - Tracheobronchial disease
4. **Pulmonary hypertension due to thrombotic and / or embolic disease**
   - Thromboembolism
     - Cardiac disease
     - Corticosteroid administration
     - Disseminated intravascular coagulation
     - Endocarditis (pulmonic / tricuspid valve)
     - Hyperadrenocorticism
     - Immune-mediated haemolytic anaemia
     - Indwelling venous catheters
     - Neoplasia
     - Pancreatitis
     - Protein-losing disease (nephropathy or enteropathy)
     - Sepsis
     - Surgery
     - Trauma
     - *Dirofilariaimmitis* (heartworm disease)
5. **Miscellaneous**
   - Compressive mass lesions (neoplasia, granuloma)

References

1. Borgarelli et al. Prevalence and prognostic importance of pulmonary hypertension in dogs with myxomatous mitral valve disease. Journal of Veterinary Internal Medicine, 2015; 29: 569-574

CPD Questions

www.bit.ly/vet360-apple https://bit.ly/vet360cpd

Go to www.vet360.vetlink.co.za or download the new Vet360 App to answer the questions CPD reserved for Vet360 subscribers (R360 per annum) Call 012 346 1590 or visit www.vet360.vetink.co.za to sign up

1. Which ONE of the following conditions is an example of post-capillary pulmonary hypertension?
a. Angiostrongylus vasorum infection
b. Patent ductus arteriosus
c. Mitral valve disease
d. Chronic obstructive pulmonary disease
e. Pulmonary thromboembolism

2. Which ONE of the following drugs is an example of a PDE-5 inhibitor?
a. Losartan
b. Sildenafil
c. Pimobendan
d. Imatinib
e. Benazapril

3. Which ONE of the following echocardiographic measures is diagnostic for the presence of mild pulmonary hypertension?
a. A TR velocity of > 2.8 m/sec
b. A PI velocity of > 1.5 m/sec
c. Marked left atrial dilation
d. Pulmonary oedema detected on th oracic radiographs
e. Presence of dyspnoea

4. Which ONE of the following echocardiographic findings is NOT consistent with pulmonary hypertension?
a. RV concentric hypertrophy
b. RA enlargement
c. Septal flattening
d. A PI velocity of > 2.5 m/sec
e. A TR velocity of < 2.0 m/sec

5. Which ONE of the following disease conditions can lead to pulmonary hypertension due to thrombotic or embolic disease?
a. Chronic obstructive pulmonary disease
b. Patent ductus arteriosus
c. Mitral valve disease
d. Pulmonary fibrosis
e. Hyperadrenocorticism

6. Which ONE of the following statements regarding pulmonary hypertension is WRONG?
a. The condition occurs more commonly in middle-aged to older small breed dogs.
b. Mitral valve disease is a frequent cause of PT.
c. Chronic obstructive airway disease can predispose to PT.
d. In many cases a clear splitting of the first heart sound (S1) can be auscultated.
e. Terrier breeds are over-represented with PT.

7. Which ONE of the following factors is less likely to influence pulmonary artery pressure?
a. RV output (aka pulmonary blood flow),
b. Pulmonary vascular resistance
c. Pulmonary venous pressure
d. Systemic blood pressure
e. Pericardial fluid accumulation

8. Which ONE of the following statements regarding Cor Pulmonale (CP) is WRONG?
a. CP is a failure of the lungs secondary to reduced LV cardiac output.
b. CP is defined as right-sided heart failure caused by pulmonary disease.
c. CP is typified by radiographic or echocardiographic evidence of right ventricular overload.
d. Pulmonary hypertension (PH) must be present in CP for the right heart to fail.
e. Pulmonary vascular obstruction secondary to heartworm (Dirofilaria immitis) is the most common worldwide cause in dogs.

9. Which ONE of the following statements regarding the determination of pulmonary artery pressure is WRONG?
a. In veterinary medicine, it is practical to infer PA pressure by determining tricuspid valve regurgitant(TR) flow.
b. In veterinary medicine, it is practical to infer PA pressure by determining pulmonic valve insufficiency(PI) flow.
c. In the absence of pulmonic stenosis, the regurgitant velocities through the valves of the right heart allows estimation of PA pressure
d. PA pressure is determined through the use of the modified Bernoulli equation, i.e. pressure gradient = 2 x (peak velocity) ^{4}.
e. A peak pulmonic insufficiency (PI) velocity of > 2.2 m/sec (peak PI pressure gradient > 19mmHg) strongly suggest PH.

10. Which ONE of the following drugs is likely to be contra-indicated in the treatment of pulmonary hypertension?
a. Pimobendan
b. Sildenafil
c. Furosemide
d. Phenylpropanalamine
e. Benazepril

Journal Scan

Compiled by Dr Liesel L van der Merwe BVSc MMedVet(Med)

Clinical evaluation of a combination therapy of imepitoin with phenobarbital in dogs with refractory idiopathic epilepsy

BMC Veterinary Research (2017)13:33.
DOI 10.1186/s12917-017-0597-z

Why they did it: The study was performed to obtain information about the efficacy and safety of imepitoin as an add-on therapy in patients receiving other antiepileptic drugs in patients showing drug resistance.

What they did: The study was a prospective, controlled, single center (a neurological referral hospital), open label study in client owned dogs with drug resistant idiopathic epilepsy. Insufficient seizure control was defined as 1 seizure event - generalised tonic-clonic seizure, cluster seizure event or status epilepticus - at least 6 weeks prior to inclusion. The patients were divided into several groups:

Cohort A were patients treated with phenobarbitone with or without co-medication with Potassium bromide (KBr) and/or levetiracetam. These patients needed to have been on phenobarbitone for at least 8 weeks and needed a serum phenobarbitone level of 10 – 35 ug/ml (43 – 150 mmol/L).

Patients receiving concurrent KBr needed to be on treatment for at least 3 months and have blood levels in the range 0.5-2.5mg/ml. Levetiracetum could be have used either as baseline therapy or as pulse therapy. Cohort A patients were started at the labelled dose of imepitoin at 10mg/kg BID and titrated upwards to 30mg/kg BID as required.

Cohort B were patients, with identical entry criteria as those in A, who were started on a lower staring dose of imepitoin, 5mg/kg BID

Cohort C were patients who were being treated with imepitoin with / without addon levetiracetum for a minimum of 6 weeks and in the upper therapeutic range of >20 mg/kg BID, who were not responding to treatment, and who were treated with add-on phenobarbitone at a low starting dose of 0.5mg/kg BID. Dose adjustments were allowed after 2 weeks of treatments, with phenobarbitone levels, to allow steady states to be reached. The maximum dose of phenobarbitone allowed during the trial was 6mg/kg.

Owners kept a seizure diary and follow up visits were scheduled for 4weeks, 12 weeks and 24 weeks. During each follow-up visit the seizure diary was evaluated as well as a hematology, biochemistry profile and phenobarbitone and potassium bromide blood levels taken. If seizure activity had occurred, the dose of only the add-on medication was increased.

Termination of involvement in the study was allowed if seizure response was insufficient despite the maximum dose of the add-on treatment had been reached (imepitoin at 30mg/kg BID and phenobarbitone at 6mg/kg BID), if phenobarbitone levels were > 35ug/ml (> 150mmol/L) , if severe side effects occurred or if owners elected euthanasia.

The primary determination of efficacy was defined as the reduction in monthly seizure frequency during treatment as compared to the defined baseline period of 42 days for each individual dog. The monthly frequency of days with cluster seizures was calculated separately.

A total of 34 dogs were enrolled: 16 dogs in cohort A, 11 dogs in cohort B and 7 dogs in cohort C. Animals were sequentially admitted to the different cohorts, so no randomization occurred. This did affect the baseline characteristics of the patients in the various groups. Animals in cohort A appeared to suffer from more severe disease with more patients (87.5%) showing cluster events versus 54.5% in cohort B. Australian shepherd dogs, Borders Collie dogs and Belgian shepherds were overrepresented (14/34 dogs)

What they found: Add on treatment resulted in a reduction in monthly seizure frequency (MSF) means was 15.46% in cohort A and 39.41% in cohort C. An increase in MSF of ≥50% was seen in 31.25% of dogs in cohort A and in 9% of dogs in cohort B.

Statistical analysis of MSF in Cohorts A-C evaluating the overall treatment effect on the entire group was significant (p=0.048), while the difference between the 3 groups was not significant (p=0.068). The add-on treatment effect did not reach significant for any individual cohort. The add-on therapy had little effect on cluster seizure therapy.

Nineteen patients completed the trial to 24 weeks: 5 dogs in cohort A, 9 dogs in cohort B and 5 dogs in cohort C. There was no significant difference from baseline monthly seizure frequent (MSF) and monthly cluster frequency (MCF) between the completers and the whole trial population, however the treatment effect was more pronounced in the completer population reaching 73% (p = 0.072) in cohort A.

Statistically the add-on treatment effect reached significance for the completer population (p=0.002) and for cohort A and B combined (p=0.009) A trend towards reduction in cluster activity was observed but was not statistically significant.

The treatment dose of imepitoin was titrated to treatment effect: mean dose in cohort A completers was 26.3±3.4 mg/kg/day, and the non-completers 48.3±4.6 mg/kg/day in the non-completers. Mean dose in cohort B completers was 29.3±4.7 mg/kg/day and 60.1±3.1 mg/kg/day in cohort B. Phenobarbitone add-on doses in cohort C varied between 1.2 – 6 mg/kg/day and did not reach therapeutic levels in this cohort. In cohort A and B serum phenobarbitone levels were mostly in the therapeutic range of 10 – 35ug/ml (43 – 150 mmol/L), with the range being exceeded in a few animals. All animals which received KBr had plasma levels in the therapeutic range. Add on treatment with imepitoin did not significantly change serum values of phenobarbitone or KBr.

Addition of KBr to patients on monotherapy with phenobarbitone, followed over 12 months resulted in a decreased seizure frequency in 83% of dogs and a 53% reduction in seizure frequency compared to the 12 months prior to addon therapy. The overall treatment effect of add-on imepitoin is similar at 51.3% (p=0.009)

Take home message: Combination of imepitoin and phenobarbitone is well tolerated in dogs. Due to lack of randomization and possible placebo effect - results need to be interpreted cautiously. The information in this study may be skewed as patients in cohort A had more severe disease. Patient numbers were also relatively small which is always a problem with statistical interpretation when multiple variables come into play

A clinically meaningful reduction in seizure frequency (≥50%) was obtained in 36 – 42% of all animals. A lower starting dose of 5mg/kg BID for add-on imepitoin was better tolerated than the standard 10mg/kg BID starting dose and was generally titrated up to an average of 15mg/kg BID. The combination of add-on imepitoin to phenobarbitone or add-on phenobarbitone to imepitoin was not effective in decreasing frequency of cluster seizures. Cluster seizures are thus an important indicator for drug resistance.

Prognostic relevance of left cardiac enlargement in dogs with preclinical myxomatous mitral valve disease. Grosso G, Vezzosi T et al

Journal of Veterinary Cardiology (2023) 45 , 50 – 58

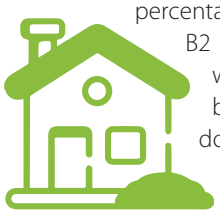
Why they did it: Myxomatous valve disease (MVD) is the most common cardiac disease in dogs and the recent ACVIM guidelines have revised the definition of stage B into Stage B1 and Stage B2. Measured variables include the left atrium-to-aorta ratio (LA:Ao) and the left ventricular end diastolic value (internal diameter), normalised for body weight, LVIDn. Dogs with normal LA and LV dimensions or with only increase in one of the above variables are classified as B1, dogs with both LV and LA enlargement are classified as B2. The primary aim of the study was to evaluate prognosis in stages B1 and B2 in the revised definition and also to assess the prognostic relevance of left atrial enlargement (LAE) and left ventricular enlargement (LVE)E in stage B2.

What they did: This was a retrospective multicentre trial. All dogs needed complete signalment recorded and had to have been diagnosed with echocardiographic examinations performed by a board certified cardiologist or supervised resident. Long term outcome was assessed by reviewing the database for re-evaluations and by telephoning owners or referring veterinarians. In case of death or euthanasia the date and cause were recorded and classified as cardiac or non-cardiac. Survival times were grouped as cardiac or all cause deaths. Sudden death was classified as cardiac.

What they found: 440 dogs with asymptomatic MVD were included. According to the ACVIM classification 276 dogs were in stage B1 and 164 dogs were in stage B2. For those dogs in stage B1, 72% of dogs had a systolic left apical murmur and 28% of dogs had no murmur whereas all dogs in stage B2 had a murmur detected. Of the stage B1 dogs 173 (63%) had no left cardiac enlargement, 73 (26%) had only LAE and 30 (11%) had only LVE. All B2 dogs had both LVE and LAE as per ACVIM definition. At inclusion 91% (251) of dogs in B1 were not on any medication, 15 dogs (5%) were on pimobendane and 8 dogs (3%) were on ACE inhibitors and 2dogs (1%) were being treated with ace inhibitors and spironolactone. In stage B2 53 dogs (32%) were being treated with pimobendane, 35 dogs (21%) were being treated with a combination of pimobendan and ACE inhibitors, 26 dogs (16%) were being treated with a combination of pimobendane, ACE inhibitors and spironolactone, 21 dogs (13%) were only on ACE inhibitors and 29 (18%) were not on any treatment.

Sixty two percent of dogs (273 dogs) were still alive at the end of the study – 43% experienced cardiac related deaths and 57% non-cardiac related deaths. 23 of the 72 dogs experiencing cardiac related death were in stage B1 and 49/72 in stage B2. The main causes were cardiac related euthanasia (35/72; 49%), cardiogenic pulmonary oedema (26/72; 36%) and sudden death (11/72; 15%). The mean survival time (MST) considering cardiac related death, was significantly different between stage B1 and stage B2 dogs with stage B1 being 2 344 days (1 905-2 783 days), 95% CI and stage B2 being 1 341 days (984 -1 698, 95% CI). Dogs with a heart murmur auscultated had a shorter MST for all-cause mortality: dogs in StageB1 with a heart murmur had a MST of 1 317 days and those without a heart murmur had a MST of 2 085 days. Independent predictors of outcome were determined using a multivariable Cox proportional hazards regression analysis multivariate regression analysis. Age, LA:Ao, and LVIDn were independent predictors of cardiac related death in stage B. Age had a hazard ratio (HR) per one-year increase of 1.14, LA:Ao ration had a HR of 3.64 per one unit increase; LVIDn had a HR of 7.34 per one unit increase.

Take home message: Stage B1 dogs have a long mean survival time for cardiac related deaths of almost 7 years and also had a low percentage of cardiac related deaths (23/276). Stage B2 dogs had a MST or approximately 3 years, with the number of cardiac related deaths being double (49/276). Among stage B1 dogs LAE and the presence of a heart murmur had prognostic significance considering all cause mortality.



Hill's
Transforming Lives

While some hear a meow, you hear your calling.

And we understand that the call isn't always easy to answer, no matter how much you love animals. That's why we're working harder than ever to support you and make nutrition conversations easier:

1. Redesigned packaging with easy-to-explain benefits
2. 100% satisfaction guarantee so you can recommend with confidence
3. New, streamlined portfolio with new varieties and simplified sizing structure

PD H&M/Helicopter/Vet360

KYRON LABS

KYRON PROBIOTICS

& THE TREATMENT OF DIARRHOEA

PROTEXIN
Reg. No. G1265 Act36/1947

Multi-strain Probiotic

Backed by over 20 years expertise & research

60g | 250g | 1kg

PRO-KOLIN+
Reg. No. V31686 Act36/194

Restores gut microflora

Dual source pre-biotic

Optimised levels of medicinal Koalin clay

Protects irritated intestinal walls

15ml | 30ml

PRO-LYTE
Reg. No. V23194 Act36/1947

Replaces lost electrolytes

Minimises villous atrophy

Replenishes & restores beneficial gut microflora

Restores energy levels

10 x 80g sachet bucket

10 x 20g sachet box

*For full product details please visit our website!

KyronLabs | @kyronlabs

www.kyronlabs.co.za

Health and beauty for all animals®

Registration Holder:
Kyron Laboratories (Pty) Ltd
Co. Reg. No. 1990/004442/07
29 Barney Road, Benrose, 2094, South Africa | Tel: +27 11 618 1544 | info@kyronlaboratories.com