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CPD Article
**Performing Open
Surgical Liver Biopsy**

Business
**Understanding the Balance
Sheet of Vet Practice**

**Qualitative Tear Film
Deficiency (Qtfd)**

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Editor's Note



Dear colleagues,

As the year draws to a close, I hope you'll enjoy this final 2022 edition of Vet360 when it lands in your mailbox along with the Christmas cards.

This month, we focus on the liver, with a general practitioner's guide to taking open surgical liver biopsies and a fascinating case study about a very South African problem: cycad toxicity. We also have a business article from our regular contributor, Andrew Christie, another chance to deepen our vision of the eye with an article on Tear Film Deficiencies from Izak Venter, as well as the first in a series on decision making in gastrointestinal foreign bodies, focused on radiology, and more.

At the end of the year, I want to give thanks to all the people and companies who make it possible to bring Vet360 to you. Thank you to the teams at Vetlink and Agriconnect who work behind the scenes to turn ideas into a beautiful magazine. Thank you to the companies who support our industry by advertising or sponsoring sections; to our world-class local and international contributors who share their knowledge with us so freely, and last, but not least, to you, our readers and subscribers.

I wish you a holiday season filled with joy and time with family, and I look forward to sharing more insightful information with you in 2023.

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

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Understanding the Balance Sheet of a Vet Practice



Andrew Christie
BComm (Business Management)

This is the first in a series of three articles that will examine the financial statements of a practice. This, the first, will explore the balance sheet while the second will look at the income statement. The final article will introduce ratio analysis as a means of extracting information from the financial statements.

Few aspects of the financial statements are as misunderstood as the balance sheet. Items seem to have different names, some of the entries don't make logical sense – even the term "balance sheet" has been replaced by "Statement of Financial Position".

In actuality, the balance sheet yields extremely valuable information for the owners and management of a vet practice. This article seeks to demystify the balance sheet, allowing better financial decisions to be made.

What does the Balance Sheet measure?

Any vet practice has things that it owns, which are called 'assets'. These assets can only have been purchased in one of two ways – money that the owner has put in, called 'equity', or by borrowing the money, called 'liabilities'. In other words:

$$\text{Assets} = \text{Equity} + \text{Liabilities}$$

And that's all that a balance sheet is – the first half shows the assets and the second half shows the equity and liabilities that paid for the assets.

Consider the example on the next page:



1 This article will use the term 'balance sheet' as it is more widely known and used outside of accounting circles.

ASSETS		2022	2021
	Notes		
Non-Current Assets		1 400 000	1 400 000
Property, Plant and Equipment	1	1 050 000	1 050 000
Goodwill	2	350 000	350 000
Current Assets		341 000	290 000
Trade and other receivables	3	91 000	80 000
Inventory	4	200 000	170 000
Cash on hand	5	50 000	40 000
TOTAL ASSETS		1 741 000	1 690 000
EQUITY AND LIABILITIES			
Equity		410 300	350 300
Issued Capital	6	300	300
Retained Earnings	7	410 000	350 000
Non-Current Liabilities		870 000	910 000
Loan - Property	8	750 000	800 000
Shareholder's Loans	9	120 000	110 000
Current Liabilities		460 700	429 700
Trade and other payables	10	300 000	209 700
Accrued Taxation	11	160 700	220 000
TOTAL EQUITY AND LIABILITIES		1 741 000	1 690 000

What does it all mean?

The balance sheet is published with comparatives from the period before. This means that whoever looks at the balance sheet has an idea of what changed in the period. There is a column for 'notes'. This is because often extra information is required to show calculations or explain an item in the balance sheet. These notes are found towards the end of the annual financial statements.

Assets and liabilities are split into 'Non-Current' assets/liabilities and 'Current' assets/liabilities. Non-current assets will be useful to the practice for a period longer than a year (for example, an x-ray machine) while a current asset can be converted to cash within a year (for example, stock).

Current liabilities have to be paid within the next year (for example, suppliers), while non-current liabilities have to be paid over a period longer than a year (for example, a vehicle).

Before providing a brief definition of the items in the balance sheet, I'll discuss the two which vets most often ask me about.

Goodwill

Goodwill on the balance sheet is calculated as:

The purchase price of a practice less the net asset value (total assets – total liabilities).

This isn't really a good measure of the actual value of the goodwill of the practice as goodwill changes literally on a daily basis – think about covid: when customers were confined to their homes, vet practices' turnover (and consequently value) plummeted. Then, when restrictions were eased, turnover rocketed for many practices. Both of these events would have had a huge effect on the value of the goodwill.

Furthermore, goodwill is based on things like the popularity of vets, the location of a practice, and modern techniques – these are variables which are largely impossible for accountants to quantify, let alone measure on an ongoing basis.

Since goodwill is difficult to measure, and is so volatile, many accountants for vet practices tend to reduce it each year until it has no value and is therefore not required to be on the balance sheet.

Shareholder's Loans

Often in a vet practice, the owners draw money based on what the practice can afford to pay. In the covid situation above, many owners had to reduce their salaries and dividends. At the end of the year, the amount that wasn't paid would be 'owed' to the owners and is therefore a liability to the practice. It is listed as a non-current liability as the practice may not be able to pay the owners in the coming year. This is what has occurred in the balance sheet above.

The opposite can also occur – owners may take money that isn't a salary or dividend. This becomes a non-current asset as it is money that is owed to the practice.

Other items are more straightforward:

i. Property, Plant and Equipment

Often shortened to 'PPE', this is normally the largest amount on the balance sheet and is sometimes broken down into separate components. It includes the property (if the practice owns its premises), equipment such as x-ray machines and 'furniture and fittings'. In the example above, Note 1 would show purchase prices and the depreciation for all the assets that comprise the PPE.

ii. Trade and other receivables

Also known as 'Debtors'

iii. Inventory

Also known as 'Stock'

iv. Cash on hand

Normally the cash in the bank, but also includes any asset that can be turned into cash immediately, such as money market funds.

v. Issued Capital

This is the amount of money the owners of a practice have invested in the practice. Very often with a vet practice, as with the example above, just a few hundred rands are put in by each shareholder to register the practice.

vi. Retained Earnings

Retained Earnings reflect the money that the owners have elected to keep within the practice, rather than take out as a dividend. It is also significant as it is the link to the income statement, one of the other major financial statements.

vii. Loan – Property

In this example, we can see that property was purchased by the practice and at least a portion was paid by a loan. We can also see that payments were made as the amount outstanding reduced from 2021 to 2022.

viii. Trade and other payables

Also known as 'trade creditors'.

ix. Accrued Taxation

VAT becomes payable as soon as the practice invoices a client. But to keep things simple for SARS and the practice, it only has to be paid every two months. In the balance sheet, accrued VAT is how much has to be paid to SARS at a point in time. In other words, if a practice ceased trading, it would owe SARS an amount of VAT, even if it wasn't on a submission date.

Why is the balance sheet important?

The balance sheet shows the financial health of a vet practice at a given point in time. In addition to what is shown (for example, we can see that the practice will have to pay R160,700 in accrued tax at the next submission date), information can also be obtained through the comparison of numbers (such as current assets vs current liabilities or changes in items from 2021 to 2022), or through analysis (such as calculating the average number of days it takes to sell stock) This analysis will be explored in a later article.

So what kind of questions can the balance sheet answer?

Management of the practice

- Does the practice have too many liabilities in comparison to assets?
- Does the practice have too much invested in assets such as stock?
- Is the practice struggling to receive payments from their debtors?
- Or make payments to creditors?

Level of Risk

- Would the practice get a loan?
- Will the practice get sufficient credit from suppliers?
- Does the practice have underlying financial weaknesses that will frighten away a potential buyer?

Cash

- Has the practice got enough cash to meet expenses?
- Will the practice have enough cash to pay current liabilities?

Andrew Christie (BComm (Business Management))

Overview: Andrew consults extensively on business issues to vet practices and other stakeholders within the veterinary industry, as well as conducting lectures on various aspects of business at Onderstepoort. His expertise with practice management in general and financial management in particular has made him a sought-after advisor on veterinary business issues, including practice valuations.

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Qualitative Tear Film Deficiency (Qtfd) – An Under – Diagnosed Ocular Disease in Dogs



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Introduction

In dogs, the precorneal tear film (PTF) is a crucial component for maintaining ocular surface health. The components of the PTF include a lipid, aqueous, and mucin layer.

Traditionally, a deficiency in the aqueous component of the tear film is known as quantitative tear film deficiency, or keratoconjunctivitis sicca [KCS] and is diagnosed by a decreased Schirmer tear test (STT).

Quantitative KCS contrasts with a qualitative tear film deficiency (QTFD) which results from an abnormal lipid or mucin component of the precorneal tear film. QTFD results in tear film instability with increased evaporation of tears and poor coverage of the ocular surface.

Both quantitative and qualitative tear film deficiencies cause ocular discomfort and corneal or conjunctival inflammation in the dog. Chronic keratitis may lead to visual disturbance from progressive corneal neovascularization and pigmentation.

Precorneal Tear Film [PTF]

The tear film is approximately 8-9 μm thick and consists of the three abovementioned components, namely lipid, aqueous and mucous layers as well as the glycocalyx.

For the purpose of this article the lipid, mucous layers as well as the glycocalyx are described in more detail.

Outer Layer [Lipid layer]

The outer layer is the oily layer which is produced by the Meibomian glands [modified sebaceous glands]. The functions of the lipid layer of the PTF include the following:

- Decreases the tear surface tension resulting in a stability of the tears/air interface.
- Prevents evaporation of tears.
- Provides a smooth ocular surface.
- Binds the precorneal tear film to the cornea at the lid margins and prevents tear overflow by its high surface tension.
- Lubricates the eyelid margins.

Inner layer [Mucous layer]

This layer is produced by the conjunctival goblet cells. Goblet cells are found in the upper and lower conjunctival fornix but highest numbers occur in the ventral conjunctiva. Functions of the mucus layer of the PTF include:

- Helps to bind the aqueous portion of the tear film to the ocular surface. The corneal epithelial cells are hydrophobic.
- Aids in corneal protection from shearing forces generated by eyelid movement.
- Collects foreign material.
- Decreases the surface tension of the tear film.
- Prevents attachment of bacteria to the ocular surface as well as concentrating IgA at the mucosal surface.
- Lubricates and hydrates the epithelial cells.

Glycocalyx

The glycocalyx is a 300nm layer of glycoproteins and glycolipids synthesised by the corneal epithelial cells. This layer interdigitates with microvilli of the corneal epithelial cells to allow the tear film to remain attached to the cornea. The corneal epithelium is hydrophobic, so the hydrophilic layer created by the mucus facilitates the spread of the aqueous layer evenly over the ocular surface. The mucus layer is not tightly adhered to the epithelial layer, but rather attaches to the glycocalyx and undergoes free movements across the cornea.

Diagnostic procedures

1. Schirmer tear test (STT)

The STT is a method of measuring production of the aqueous portion of the precorneal tear film [PTF]. In veterinary practice, we do a STT 1, which means the procedure is done without topical anaesthesia and therefore measures both basal and reflex tear secretion.

The STT should be the first procedure performed during the ocular examination process, because manipulating the

eye may stimulate reflex tearing, while topical anaesthetics and parasympatholytic drugs may decrease tear production and lead to inaccurate results.

The test is performed with sterile strips of absorbent filter paper. Each strip is folded at the notch and hooked over the ventral eyelid and held in place for 1 minute. The STT strip should be placed in the mid- to lateral region of the lower eyelid, where it can contact the corneal surface. When placed medially, the third eyelid can protect the cornea and falsely reduce STT results by reducing reflex tearing. The reading should be taken as soon as the strip is removed from the eye.

Interpretation:

- >15mm/min normal
- 10 – 15 mm/min suspect
- < 10 mm/min abnormal
- It is important to note that in patients with only QTFD the STT is normal.

2. Tear break-up time (BUT)

This is an easy, valuable but underutilized diagnostic test. A deficiency of the mucin or lipid layer of the precorneal tear film will lead to instability of the precorneal tearfilm and therefore an abnormally short BUT. Normal BUT in dogs is > 20 seconds.

To do the test, one drop of water / saline is applied to a sterile fluorescein strip. The fluorescein strip is then applied directly onto the dorsolateral bulbar conjunctiva. The eyelids are manually held closed. Upon opening the eyelids the tear film is observed by using a light source with a cobalt blue filter. The ideal instrument is a slit lamp biomicroscope, but a normal ophthalmoscope can also be used. A stopwatch should be used to determine the length of time until initial breakup of tear film is noted. This is seen as the formation of a dry spot or lack of fluorescein on the ocular surface.



Veterinary Ophthalmology. 2021; 24:503–508.

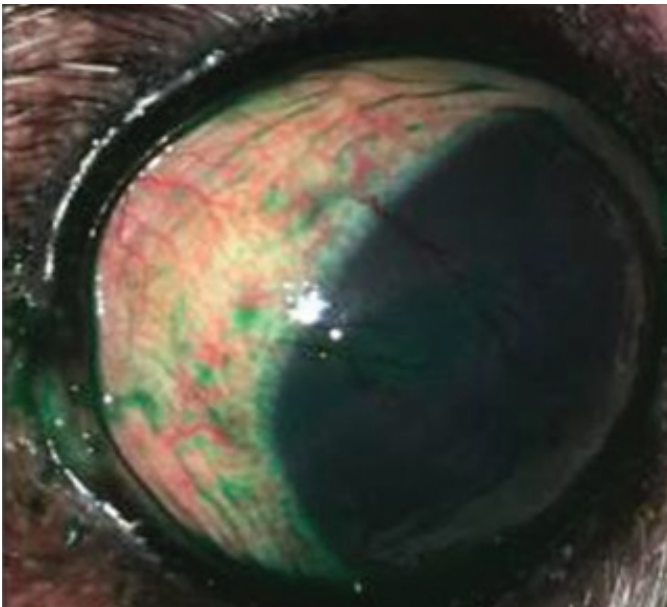
Figure 1: A wetted fluorescein strip is applied to the dorsal bulbar conjunctiva. The eyelids are manually closed and then kept open. With assistance of the cobalt blue filter, the distribution of the fluorescein in the tear film is visualized across the corneal surface. Timing must be stopped once the formation of a dry spot or lack of fluorescein is noted on the corneal surface.

3. Lissamine Green

Lissamine green is a synthetically produced, acidic, organic dye, that stains devitalised corneal and conjunctival epithelial cells.

Lissamine green should be applied after fluorescein. After flushing excess fluorescein dye from the cornea and conjunctiva with sterile eyewash, a drop of lissamine green ophthalmic dye is applied to the dorsal bulbar conjunctiva.

The eyelids are closed and then opened to ensure complete coverage of the dye over the entire bulbar conjunctiva. The bulbar conjunctiva and cornea are examined with a light source. Immediate assessment of the staining pattern must be performed because of the potential for rapid diminishing of intensity and extent of ocular staining within two minutes of dye application. Devitalised cells stain in a distinct green colour. Lissamine green is commercially available in South Africa and is manufactured by VetScripts.



Veterinary Ophthalmology. 2020;23:624–631.

Figure 2: Lissamine green positive corneal staining pattern visible on the bulbar conjunctiva.

4. Conjunctival biopsy

This is seldom done as the BUT and Lissamine green staining is very specific. A diagnosis of a mucous deficiency can be confirmed by seeing decreased numbers of goblet cells histologically.

Deficiency of the Precorneal Tear Film Mucin

Mucin deficiency is a common condition in dogs. The underlying pathophysiology includes decreased numbers

of conjunctival goblet cells or alterations in the corneal and conjunctival epithelial cell glycocalyx. Alterations in the glycocalyx are caused by chronic inflammation. This condition is sometimes not diagnosed as BUT and lissamine green staining are often not routinely done during the examination procedure.

Deficiencies of the lipid layer of the precorneal tear film.

Like mucin deficiency, this condition is probably underdiagnosed in veterinary ophthalmology. Inflammation of the eyelid margin and meibomian glands caused by Staphylococcal and Malassezia- associated blepharitis, seborrhoea, atopy and demodicosis may lead to decreased lipid production. Severe distichiasis treated with electro-epilation will also lead to destruction of the Meibomian glands.

Clinical signs

The clinical signs of QTFD is very unspecific, characterized by primarily a low-grade chronic conjunctivitis and or keratitis. The corneal epithelial surface may have a "rough" appearance and the conjunctiva will stain Lissamine green positive.

Treatment

Tacrolimus / Cyclosporin eyedrops may improve ocular mucins, independent of an increase in the aqueous component of the tear film.

The most important part of the treatment is the use of tear replacement solutions that can contribute to the stability of the PTF. There are numerous tear replacement products on the market including chondroitin sulphate, hyaluronic acid, polyethylene glycol, propylene glycol, polyvinyl alcohol as well as methylcellulose.

Chondroitin sulphate is a glycosaminoglycan that is a constituent of the extracellular matrix of mammalian cells. It is found in high concentrations in the corneal stroma, where it contributes to corneal stability, rigidity, and transparency.

Chondroitin sulphate possesses both anti-inflammatory as well as immunomodulating properties. This includes the inhibition of leukocyte directional chemotaxis, phagocytosis, and lysozyme release.

Chondroitin sulphate also has mechanical protective qualities for the conjunctiva and cornea. When in solution, chondroitin sulphate is a Newtonian fluid with a high affinity for the anterior corneal surface. This affinity for the anterior corneal surface may be partially due to muco-adhesive properties which result in prolonged contact between the chondroitin sulphate solution and the corneal surface after administration. This is an extremely important property in the veterinary field, as frequent administration of eyedrops is often impractical, or even impossible.

Newtonian fluids maintain a relatively constant viscosity at all shear rates. This is in contrast to pseudoplastic fluids (e.g. sodium hyaluronate and methylcellulose), which exhibit higher viscosity when they are under low shear rates. Low shear forces are created by eyelid and third eyelid movement over the ocular surface during the blinking action. These low shear forces are transmitted to the corneal epithelium as a drag force and can induce corneal epithelial damage. The fraction of this shear force transmitted to the corneal epithelium as a drag force increases as the viscosity of the fluid separating them increases. This is where chondroitin sulphate being a Newtonian fluid provides exceptional corneal lubrication, by minimising the transmission of shear forces to the corneal epithelium.

Chondroitin sulphate ophthalmic solutions also improve tear film stability, resulting in a rapid and prolonged recovery of stability after the tear film is disrupted. All these positive effects, but especially the improvement of tear film stability makes chondroitin sulphate the product of choice for the treatment of QTFD.



Figure 3: Chondroitin sulphate "Tears" eyedrops for the treatment of QTFD.

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ANIMAL HEALTH

Intensive Antioxidant and Regenerative Treatment Of Cycad Toxicity in a South African Dog: A Case Study



Dr Luzaan van der Laan
BVSc Hons
Valley Farm Animal Hospital

Introduction to cycad toxicity

Cycads, also called sago trees or, in Afrikaans, broodbome, are sought-after ornamental plants in South African gardens, with both the rare and protected indigenous species and their exotic counterparts being prized for their short, wide stems, large glossy leaves and striking seed pods.



Both indigenous cycads and exotic cycads such as *Cycas revoluta* are popular ornamental trees in South African gardens.

Interestingly, the pith of many species of cycad can be transformed by fermentation into a type of flour or paste, baked and then eaten, but all parts of the plant contain substances toxic to humans and animals if consumed in an untransformed state. Unfortunately for veterinary patients, the seeds, which are most commonly consumed by curious animals, contain the highest concentrations.

Three main toxins have been found:

1. Cycacin. In and of itself, is carcinogenic, teratogenic and mutagenic. However, the grave short-term danger to dogs lies in its active metabolite, methylazoxymethanol, which not only causes severe gastrointestinal irritation, but also centrilobular and midzonal coagulative hepatic necrosis, which could develop into cirrhosis.
2. Beta-methylamino-L-alanine is a neurotoxic amino acid causing ataxia in rats and symptoms similar to Parkinsons' disease in humans
3. A third, as-yet unidentified high molecular weight neurotoxin has been found to cause axonal degeneration in the CNS leading to hindlimb paralysis in cattle who ingest the plant.

In dogs, liver failure is the leading cause of mortality, with an estimated mortality rate of 32% in clinically affected patients, according to one study. For this reason, any dog presenting with clinical signs and a reasonable suspicion of cycad ingestion is treated as a serious case with a guarded prognosis.

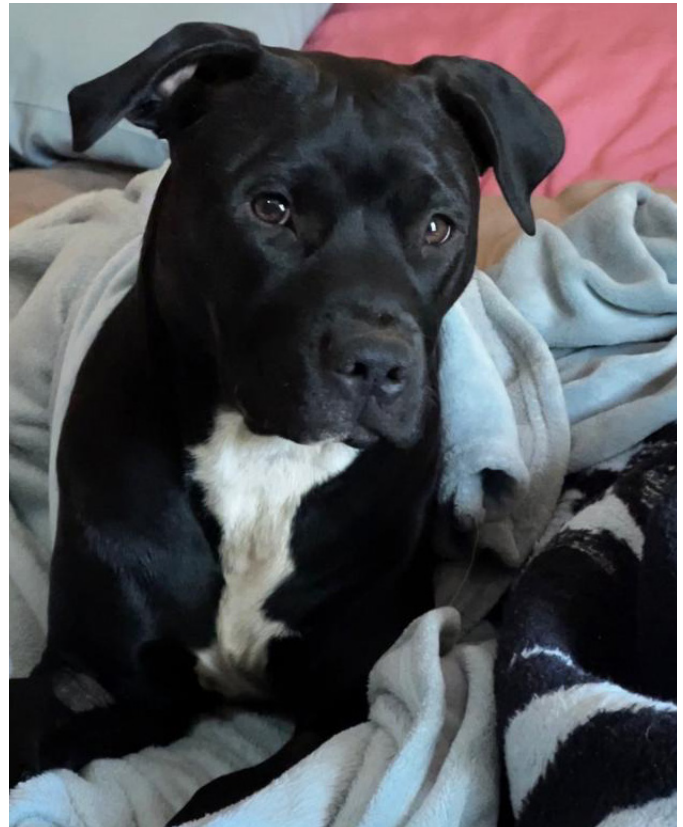
A complication of treatment is the fact that liver signs, such as elevations in serum bilirubin, ALT and ALP may only start 24 to 48 hours after ingestion, by which time the gastrointestinal symptoms may have resolved. In addition, owners of young, curious dogs may not consider the initial gastrointestinal signs to be a result of anything more dangerous than a simple dietary indiscretion.

Treatment is symptomatic and hepatosupportive, and if caught early enough after ingestion also include gastric lavage to reduce the total amount of toxin consumed. However, the relatively elevated mortality in clinically affected animals remains a cause for concern and a reason for investigating possible additional treatment modalities which could improve a patient's chances of recovery.

Case study: Luna

Clinical History & Signalment:

Luna, a 19.9kg, 11 month old spayed female Pitt Bull Terrier, was presented late evening to an After Hours Veterinarian for vomiting. She had started vomiting late morning and the pet sitter had become concerned when she noted blood tinged vomit, plant material and red cycad berries in the vomit. Activated charcoal was administered at home prior to presentation.



Physical Examination:

On physical examination during consultation, Luna was depressed and drooling. Abdominal palpation, elicited no guarding, but slight cranial abdominal discomfort. Chest auscultation was within normal limits, and skin, ears, eye and body scoring were all normal. Her temperature was 38.6 degrees, her pulse 120bpm and her respiratory rate 36 breaths per minute. A blood smear revealed mild neutrophilia but no other abnormalities and no parasites. In-house blood gas, electrolyte haematology and blood chemistry examinations were performed and revealed results within normal limits, with the exception of elevated ALT, at 142u/L (with the reference interval being 10-125uL). Abdominal ultrasound showed no free fluid, a liver ultrasonographically within normal limits and a mildly distended gall bladder.

Diagnosis:

Having ruled out other likely causes of the presenting signs, and considering the clinical history, a diagnosis of cycad toxicity was made and Luna was admitted for treatment.

Initial treatment and plan:

- Overnight, Luna was given the following standard supportive treatment.
- Fluid therapy with crystalloids, supplemented with potassium chloride
- Antiemetic support with metoclopramide and maropitant

- Esomeprazole to counter mucosal inflammation
- Buprenorphine as needed for pain control
- Silymarin as hepatoprotectant

In the morning, Luna was ambulatory and her temperature, pulse and respiration were within normal limits, but she was still nauseous and depressed.

The owners consented to an experimental treatment protocol which added intensive antioxidant and regenerative treatments to the existing protocol. The additional treatments were:

- Ondansetron for antiemetic support
- Intravenous glutathione at 140 mg/kg bid, for additional antioxidant and hepatosupportive action
- Phosphocholine, a major component of cell membranes, as a once-off intravenous hepatoprotective treatment
- Allogeneic canine Mesenchymally Derived Stem Cells, 11,000,000 stem cells delivered via splenic injection for the regenerative potential on the liver

Short-term progress

24 hours after admission and 12 hours after the additional supportive and regenerative treatments were administered, blood analysis was repeated. This time, all parameters were within normal limits, including ALT, which had dropped from 142u/L to 94u/L (see figure 1 and 2 below).

Luna was discharged two and a half days after admission with standard liver support (Hepafocus & Silymarin) and a follow-up appointment in 7 days for repeat blood tests to follow her liver's recovery.

Prognosis & Outcome:

At her seven day recheck, Luna's owner reported her as back to normal, eating playing and guarding.

On clinical examination – all parameters WNL. Her ALT level was, however, once again elevated to 239 u/L – a sign of ongoing hepatic challenge.

Intravenous glutathione was once again administered via slow intravenous continuous rate infusion, and her owners were asked to continue with the oral liver support (Silymarin & Hepafocus).

At a month post admission, Luna's owner reported her to be in excellent condition. She had also gained weight (22.8kg).

Repeat blood tests showed all clinical values to be within normal limits and abdominal ultrasound showed normal liver and gall bladder.

Liver support was discontinued and Luna will be followed up again in a year.

Discussion and key notes:

- Treating cycad toxicity aggressively from the start can avoid the premature death of so many young dogs as a result of liver failure months after ingesting cycads
- In the management of toxicities or oxidative injuries, the use of intravenous Glutathione is potent as free radical scavenger and antioxidant, and could be considered instead of the more well known antioxidants like Vit E, Acetylcysteine and Vitamin C
- Phosphatidylcholine (PC) is a major surface-active phospholipid and is widely used in human healthcare, due to, amongst other functions, its incorporation into the cell membrane, thus protecting damaged hepatocytes.
- The use of Allogeneic Adipose Derived Mesenchymal Stem Cell Therapy are becoming a scientifically proven successful option for many injuries and diseases. Allogeneic stem cells are currently undergoing clinical trials in South Africa, and could have potential in acute hepatic injury

GLU	7.55 mmol/L	4.11 - 7.95	
CREA	74 µmol/L	44 - 159	
UREA	6.2 mmol/L	2.5 - 9.6	
BUN/CREA	21		
TP	66 g/L	52 - 82	
ALB	34 g/L	23 - 40	
GLOB	33 g/L	25 - 45	
ALB/GLOB	1.0		
ALT	142 U/L	10 - 125	H
ALKP	113 U/L	23 - 212	

Figure 1: Luna's blood results at admission

ALB	33 g/L	23 - 40	
ALT	94 U/L	10 - 125	
ALKP	104 U/L	23 - 212	
TBIL	2 µmol/L	0 - 15	

Figure 2: Luna's blood results 24 hours post admission

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Detox Your Practice: How to Eliminate Toxic Team Members and Avoid Hiring Them



August 31, 2022

Michele Drake, DVM, CVA

Michelle Drake, DVM shares team management and hiring best practices that support a practice's mission, values, and culture

It's hard enough to build a practice today. It's even more challenging with an employee that's working against your goals. It's critical to eliminate toxic employees or, even better, avoid hiring them in the first place.

What exactly is a toxic employee?

This word is used in many ways. Sometimes, it refers to someone who is mean, dishonest, or creates drama. These behaviors are undoubtedly toxic. Relative to the workplace, employees are toxic to your practice if they're not aligned with your mission, values, and culture and create discord. They do not fit the profile of the type of person you believe will make your hospital a great place to work.

The worst employees—truly destructive humans—would be toxic anywhere. However, an employee could have a toxic effect even if they're not a "bad" person, if they are simply a terrible culture fit and won't get with the program. Quite often, the behavior is more insidious and quietly destructive. These employees may speak negatively about other employees or complain about policy and procedure but never want to help with solutions. They create small dramas amongst the staff instead of putting energy into providing excellent care and service.

Toxic employees harm every aspect of your practice

These behaviors break down your practice culture, leading to frustrated, demoralized employees and poorly served clients. Keeping toxic employees around causes your best employees to leave.

If an employee has a toxic situation in their current job and is offered a couple of dollars per hour more somewhere else, they're likely to leave. By harming your team's effectiveness and reducing trust from your clients, toxic employees are also deadly to your bottom line.

Think of your other employees and yourself

When a practice has misaligned people, this affects everyone. As the owner, your wellbeing is also impacted. If you have someone like this in your practice, take a moment to think:

- Does this person bring positive or negative energy into the practice?
- Is this person involved in many small dramas that take away from the hospital's mission?
- Has your practice gotten a negative review or lost a client due to this person?
- How much time and energy did it take to handle that?

- Factoring in these considerations can help you realize how much that toxic employee is really costing you in stress, time, and mental health.

Sorry, sometimes It's a DVM (or manager)

Many practice owners have a "blind spot" with associate DVMs and managers. They may say they avoid toxic employees, but they have a DVM who's been with them for 20 years and is constantly yelling at the staff, complaining a lot, or doing things their own way instead of what the practice has decided to do. Or, they may have a manager who plays favorites with employees.

Often practice owners make excuses for these individuals: "It's just how he is" or "I couldn't do without her." However, if someone will not get on board with your mission, values, and culture, they need to go—regardless of their role. It may not seem like it right now, but if that person is poisoning your culture, you're better off without them.

We all know that firing employees is one of the worst parts of practice ownership. Yet if you avoid getting rid of toxic employees in the name of being nice, you're delaying the inevitable and harming valuable coworkers in the process.

The good news

As the practice owner, you have the complete ability to resolve toxic employee issues. You must first point the finger at yourself, not others. You can develop a strong, healthy organization and ask that employees adhere to your culture and rules of conduct in the practice, and if they don't, you can fire them and find new employees who will.

Culture benefits are everywhere

The benefits go beyond just eliminating the most egregious sources of toxicity. If you, as the owner, can see someone is a poor team member, the rest of your employees know it too. They're watching and waiting to see if you'll take action or if you'll let this person continue to behave badly. When you take action to remove toxic people, this sends a message to your team that you care about them. It also reminds anyone on the "borderline" that they need to straighten up and bring their best self to work.

Yes, I know there's a labor shortage

Practice owners often tell me about a negative employee who drags the entire team down. My immediate response is, "And you fired that person, right?" Too often, the answer is some version of, "No, we need them because we can't find good people." The practice will remain stagnant and dysfunctional until the toxic employees decide to change their behavior or are asked to leave. Hire and train for culture and values, and you will have a completely different practice.

I'm proud to say we've had little trouble hiring doctors and staff. This doesn't mean that it's not time-consuming; it does require energy and attention. But even during the pandemic, we hired 4 new doctors and more than 15 staff members. We hire for culture and core values first, skills second. We've had many team members leave dysfunctional veterinary hospitals to come and work at The Drake Center, sometimes for less money. I'm happy I'm able to provide a better workplace for them. The reason many corporate groups throw out huge hiring bonuses is that they have to. They are not good places to work, and their turnover is very high. A great place to work is very valuable.

What to do now

If you think you may have 1 or more toxic employees, take the following steps:

1. Accept full responsibility. Recognize that you have the power to change the situation.
2. Clarify and write down your mission, values, and culture, and communicate these to your leadership team. Then, work to get buy-in from the entire team. You can't reprimand or fire someone for not following your values if no one knows what they are. This takes time and energy, but nothing will change until you do so.
3. When your problem employee violates these guidelines, talk with them. If talking doesn't resolve the behavior, do a formal write-up specifying exactly what behaviors were out of line and why.
4. If the person improves, that's great.
5. If the bad behavior continues, fire the employee and communicate to your team exactly why they were fired, reinforcing how seriously you take your values.

The best solution: don't hire toxic people in the first place

With the right hiring process, you can avoid these issues altogether. Here's how to avoid hiring toxic people:

1. Have your mission, values, and culture clearly defined, and ask questions that check how the interviewee aligns with your culture.
2. Listen carefully for red flags. These are best uncovered through dialogue. My hospital manager is a real expert at this. Specifically, she listens for complaints about former employers or negativity about work, clients, or family.
3. Prioritize will and culture fit over skill and experience. It's tempting to hire someone with experience. But it's much easier to train someone on the skills they'll need than to train them to change already-ingrained behavioral dysfunctions.

By taking these steps, you'll be well on your way to a practice free of toxic people, where you and your employees will thrive, and your clients and patients will benefit greatly.



Gastrointestinal Foreign Body Decision-Making

Part 1: Radiography

By Amy Haylock, BSc(Hons) BVSc(Hons) MRCVS
IMV imaging Clinical Manager

A wide range of gastrointestinal foreign bodies (FBs), of varying materials and radio-opacities, are commonly ingested by our patients; usually necessitating emergency intervention. The question in the clinician's mind when performing diagnostic imaging is usually; if there is a FB in this abdomen, is it detectable on radiography and/or ultrasound, and if so, how will it appear? This is particularly pertinent if there might be multiple FBs in one patient, and the owner is unaware of FB ingestion; i.e. a stone and a sock.

Ultrasonography has been shown to have a higher sensitivity and specificity than radiography in the diagnosis of intestinal obstruction^[1,2], with the sensitivity of radiography being as low as 56% for the detection of FBs in comparison with ultrasonography^[1]. Conversely, radiography has several advantages; being quicker to perform, more readily available, less expensive, and less operator skill-dependent^[3].

However, radiography and ultrasonography provide complementary information for the gastrointestinal tract and, as such, many clinicians utilise both methods of

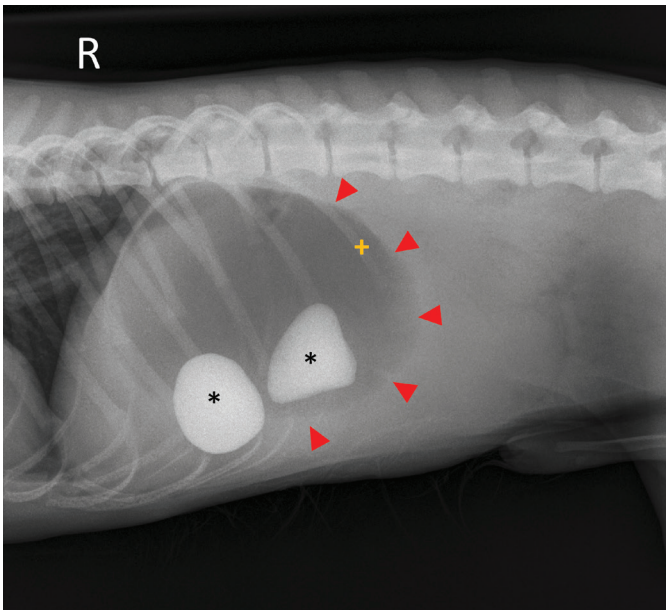


Figure 1: Right lateral abdominal radiograph of an 11 month old ME springer spaniel with two radio-opaque stone FBs within the descending duodenum (black asterisks). The obstruction is proximal to the jejunum, resulting in gastric dilation only (red arrowheads). The degree of gastric dilation is seen to extend past the last rib (yellow cross), so can be graded as severe^[6].

Note the poor serosal detail within the abdominal cavity. The dog was lean, with relatively little intra-abdominal fat, and at surgery, a small volume peritoneal effusion was identified.

Image source: author's own.

imaging for the same patient. This article will examine the radiographic findings in cases of gastrointestinal FBs.

Ascertaining whether the gastrointestinal FB has caused obstruction is of paramount importance. Surgery which follows a delay in the diagnosis of gastrointestinal obstruction is associated with an increased morbidity owing to dehydration, electrolyte imbalances, devitalisation of the bowel, bowel perforation, adhesion formation, bacterial translocation, and sepsis^[4,5].

The diagnosis of gastrointestinal FBs via radiography can be more accurately viewed as the diagnosis of mechanical obstruction, or mechanical ileus. The temptation for the clinician is to look only for the presence of a radio-opaque FB first and foremost (see Figure 1), and feel frustrated when one is not immediately apparent.

Gastrointestinal FBs are often radio-lucent, and may be obscured by superimposed gas shadows, abdominal effusion, or poor serosal detail in very young animals and

animals with a low body condition score. It is important to remember that obstructive FBs are associated with a range of secondary effects, many of which can be detected on radiography.

The most common radiographic signs of mechanical obstruction include^[1,6]:

- A visible FB or mass (see Figure 1)
- Segmental dilation of the bowel, either with gas or fluid, proximal to the lesion (see Figures 2, 5 and 6)
- Abnormal position and/or path of the bowel (see Figure 3)
- Persistent focal accumulation of small mineral fragments within the gastrointestinal tract, also known as the "gravel sign" (see Figure 6)
- Loss of serosal detail (see Figure 1)
- Horizontal fluid lines within loops of bowel

A consensus cutoff value for the severity of small intestinal (SI) dilation, which strongly suggests mechanical

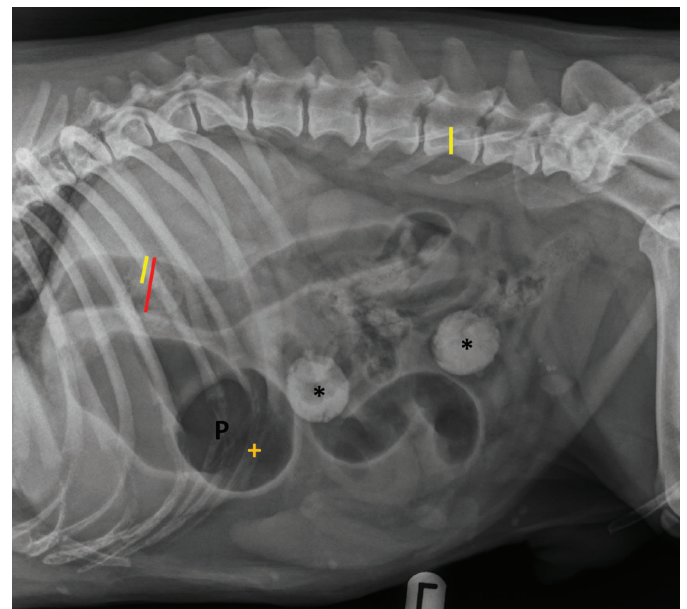


Figure 2: Left lateral abdominal radiograph of an 8 year old MN English Bull Terrier with two mid-jejunal obstructive rubber bone toy FBs (black asterisks). The height of the middle of L5 has been marked by a yellow line, and compared alongside the height of the maximally dilated SI (red line). The SI/L5 ratio in this case is approximately 2.0. The pattern of intestinal dilation here is segmental; i.e. <25% of the SI is affected^[3].

Note the gastric distension can be classed as severe, since the pylorus (P) extends past the line of the costal arch (yellow cross)^[3].

Image source: author's own.



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obstruction, has not been reached. A ratio of the width of the most dilated loop of SI to the height of mid-L5 (SI/L5) is often utilised^[3,7,8], and readily applicable in first opinion practice (see Figure 2).

In one study, 86% of unobstructed dogs had a maximal SI/L5 ratio of <1.6 , whereas a group of 44 animals with intestinal obstruction had mean SI/L5 ratios of 2.3, with a range of 1.5–4.8^[7]. Notably, in that study, maximal SI/L5 diameters in un-obstructed dogs were as high as 2.5. For cases of apparent functional ileus such as these, the pattern of SI dilation has been shown to be strongly linked with the likelihood of an acute obstruction (see image 2)^[3].

Segmental dilation ($<25\%$ of SI loops affected) is very suggestive of obstruction, particularly if accompanied by $SI/L5 \geq 2.4$ ^[3]. This is in comparison to regional dilation (25–75% of the intestine affected), and diffuse dilation ($>75\%$ of intestine affected). A more diffuse pattern of SI dilation might be expected with an inflammatory gastrointestinal condition, or a more chronic, distal intestinal obstruction.

Interestingly, another study demonstrated a sensitivity and specificity of 66% when using a cut-off SI/L5 ratio of ≤ 1.7 , with no increase in the accuracy of diagnosis of mechanical obstruction when this objective cut-off was used, as opposed to an initial, subjective visual assessment by six observers^[8]. An SI/L5 of 1.95 represented a 77% probability of obstruction, and an SI/L5 of 2.07 represented an 85% probability of obstruction^[8].

Linear FBs present unique diagnostic and surgical challenges. Although rarely visible as radio-opaque entities, they are associated with high morbidity, owing to the potential for a delayed diagnosis, and damage caused to the mesenteric border of the gastrointestinal tract.

Deviations in the normal path and gas distribution of the SI are suggestive of a linear FB. Proximal anchoring of a linear FB within the pylorus, or under the tongue, together with peristalsis, cause the SI to gather and bunch around the linear FB. Eccentric tapered gas bubbles can be seen in multiple locations within the SI (see Figure 3).

While initially not an obstructive lesion, linear FBs can lead to the development of an intussusception, and therefore, mechanical obstruction.

In cases of equivocal mechanical obstruction, or when surgery is not an immediate option, performing serial abdominal radiographs can be helpful (see Figure 4). Continuing obstruction will usually result in progressive SI dilation (see Figure 5), +/- abdominal effusion, gravel sign and pneumoperitoneum in the case of gastrointestinal rupture. However, linear FBs are often associated with little or no SI dilation, and serial radiographs may not identify progressive dilation over time, even though the damage to the intestine may be worsening.

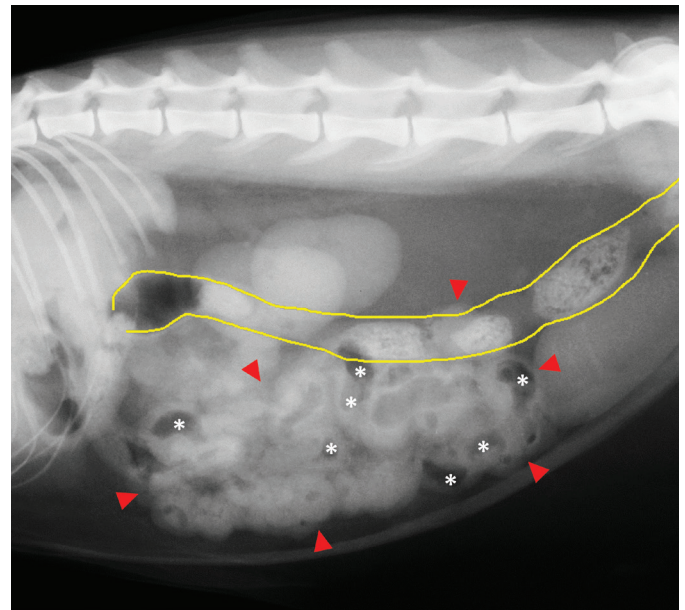


Figure 3: Lateral abdominal radiograph of a cat with a linear gastrointestinal FB. Note the bunching of the SI in the ventral abdomen (red arrowheads) and the small, eccentric comma-shaped gas bubbles spaced around the area of the SI (white asterisks). The descending colon (delineated in yellow) has been displaced ventrally by the tension in the SI.

Image courtesy of Nic Hayward, BVM&S DVR DipECVDI MRCVS

In a retrospective study of patients receiving medical treatment following the radiographic diagnosis of gastrointestinal obstruction or the presence of foreign material, serial radiographs demonstrated that radiographic resolution was dependent on the location of gastrointestinal dilation. 37.5% of gastric dilation cases resolved, 17.1% of SI dilation cases resolved, and 11.4% of concurrent gastric and SI dilation cases resolved^[6].

Of the 153 animals which underwent one repeat radiograph within 36h, 41 (26.8%) resolved. Of the 26 animals which underwent a second repeat radiograph after 36h, 9 (34.6%) resolved. Of the 6 animals which underwent a third repeat radiograph after 36h, 3 (50%) resolved. Overall, 34.6% of radiographically diagnosed obstructions resolved without surgery.

Radiographs performed after 36h did not improve the chances of resolution. Only 2 of the 153 animals (1.3%) experienced gastrointestinal tract rupture, both of which occurred after 3 repeat radiographs. This finding supports surgical intervention if radiographic resolution is not detected within the first 36h.

Another prospective study of equivocal cases of gastrointestinal obstruction performed follow-up radiographs between 7 and 36 hours after the initial radiographs, and employed abdominal ultrasonography as the gold standard reference test for the presence or absence of obstruction.

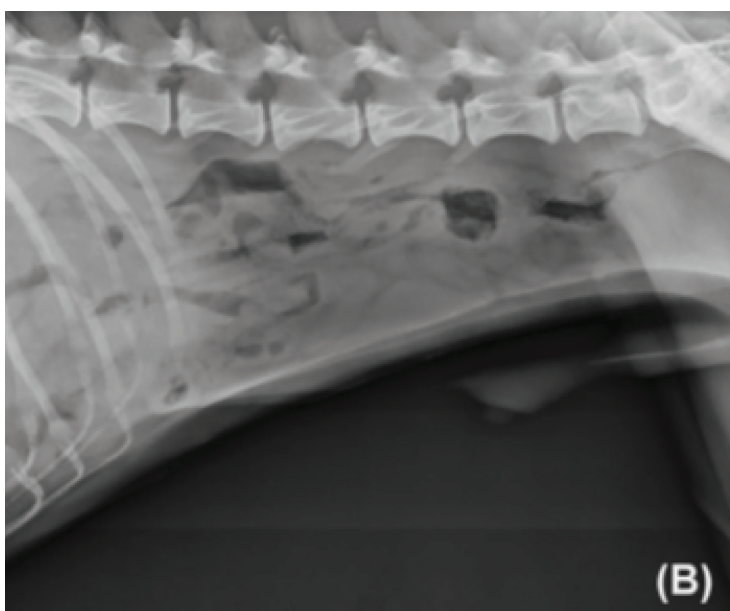
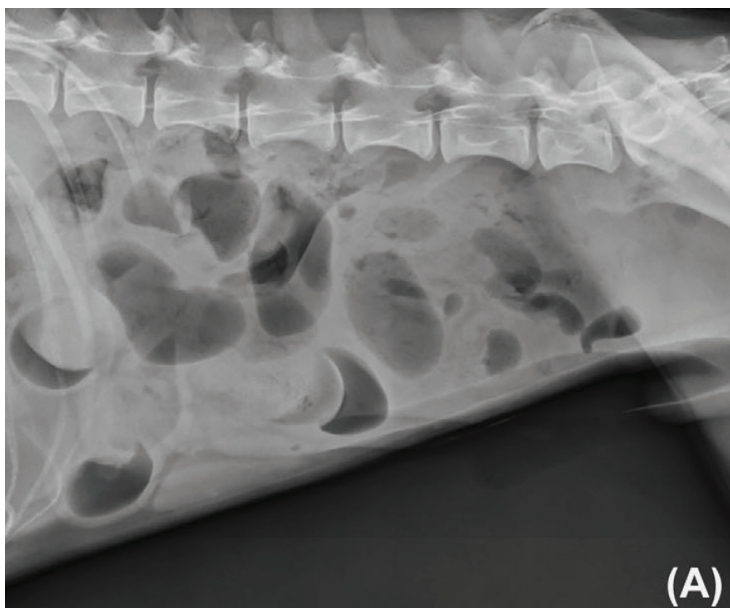


Figure 4: Lateral radiograph of a 3 year old MN Labrador at time = 0h (A) and time = 19h (B). In image A, there is mild gastric dilation, and severe SI regional dilation (25 -75% SI affected). In image B, the radiographic signs of dilation have resolved.

Image source: Miles et al, 2021^[6]

The results of this study found that there was no increase in the accuracy of the diagnosis of obstruction, across all four reviewers, between viewing the first and the follow-up radiographs^[9]. Ingested mineralised material within the gastrointestinal tract can provide useful information in the case of partial obstruction.

While softer, less heavy materials may pass through a reduced lumen diameter or an altered lumen course, gravel-type material will accumulate in discrete areas. This is known as the gravel sign (see Figure 6).



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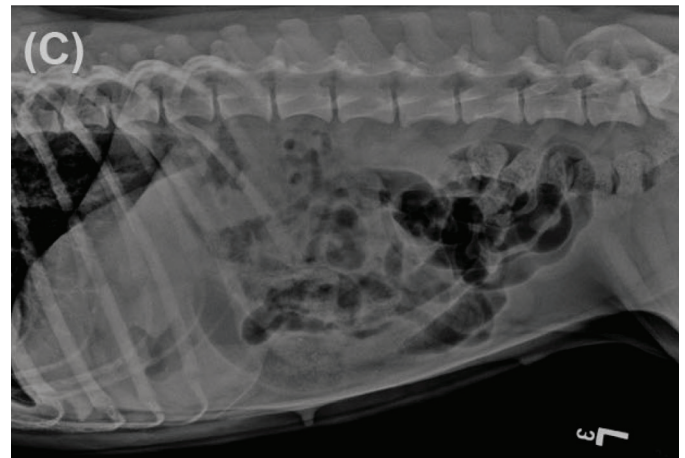


Figure 5: Left lateral initial radiograph (A) and follow up left lateral radiograph (C) of a dog with a jejunal FB causing mechanical obstruction. The four reviewers all provided an initial false negative diagnosis of obstruction when viewing the initial radiograph (A). Three out of four reviewers changed their assessment to obstructed when viewing the follow-up radiograph (C). Note the worsening SI dilation in (C), both in terms of level of dilation and amount of SI affected.

Image source: Elser et al, 2020^[9]

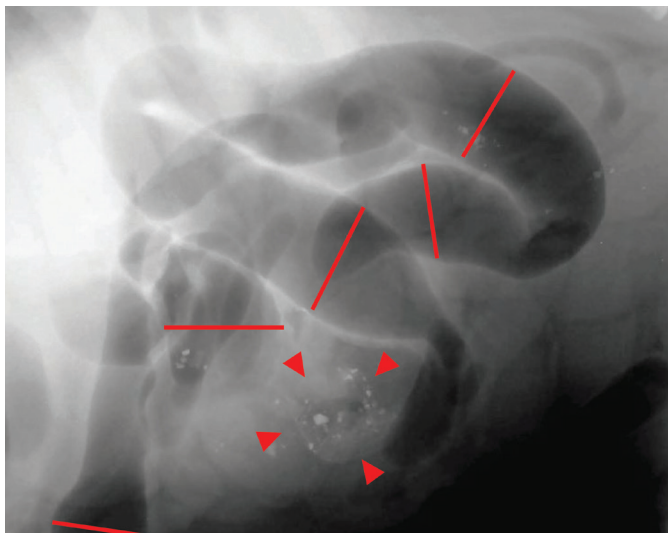


Figure 6: Left lateral abdominal radiograph of a mature dog with a chronic history of vomiting. Note the presence of multiple loops of dilated SI (marked by red lines) and the presence of the "gravel sign" (red arrowheads).

Image source: Parry et al, 2019 [10]

In conclusion, although less sensitive than ultrasonography for the diagnosis of gastrointestinal FBs, particularly radio-lucent and linear FBs, there are a wide range of characteristic signs of intestinal obstruction which can be detected on plain films.

The two modalities are complementary, and radiography remains a useful and important first line diagnostic modality for gastrointestinal FBs. As with all imaging modalities, the experience of the clinician is key to interpretation, as is correlating the imaging findings alongside the clinical signs and timing of image acquisition. Please look out for part two of this article series, which will focus on ultrasonography's role in abdominal foreign body detection.

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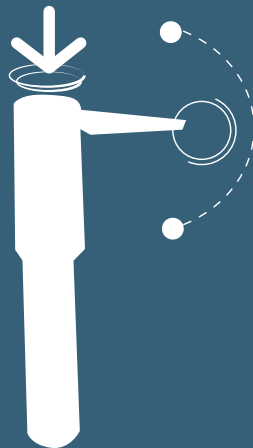
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Performing Open Surgical Liver Biopsy

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A guide for general practitioners in performing hepatic guillotine and punch biopsy techniques.

Liver biopsy is indicated for a number of hepatic disorders. While laparoscopic surgery is an excellent means of obtaining biopsy samples because of its reduced surgical morbidity, the instrumentation and training required to perform the procedure can limit its usefulness in general practice. Patients that require liver biopsy can be referred to specialists, especially patients at high risk or when a minimally invasive approach is desired. However, becoming familiar with techniques for open liver biopsy allows the general practitioner to perform this procedure when indicated.

Open surgical liver biopsy has many advantages:

- The surgeon can visually inspect the entire liver, sample focal lesions or areas of interest, and easily control hemorrhage.
- Samples can be collected from multiple liver lobes, increasing the likelihood of reaching a diagnosis.
- The samples collected during open surgical liver biopsy are large enough for histopathologic examination or mineral analysis, which is required for diagnosis of some disease conditions.

Overall, histopathology is more accurate than cytology and is usually considered the gold standard for hepatic evaluation. In a study designed to determine the accuracy of diagnosis between hepatic cytology and histology, the two methods were found to be in agreement in only 15 of 56 canine patients and 21 of 41 feline patients.¹

Guillotine technique

The guillotine technique is performed when attempting to sample the outer margin of a liver lobe.

Isolate an easily accessible margin of liver lobe and, using monofilament absorbable suture, place a circumferential ligature around the tissue margin. A surgeon's throw or friction knot is recommended for the initial throw.

Pull tightly on the loop of suture, crushing the hepatic parenchyma; both blood vessels and bile ducts will be

occluded as the soft tissue is crushed. Then tie the suture with an appropriate number of additional throws to complete the knot (Figure 1).

Holding the liver margin in one hand, sharply incise the tissue several millimeters distal to your ligature. Ensure there is adequate space between your suture and the tissue margin so the ligature does not slip off and increase the risk for hemorrhage.

Do not handle the biopsy sample with forceps or other surgical instruments, as these can damage the tissue and cause artifacts during histopathologic examination. Handle the tissue as minimally as possible before placing it in formalin.

Check the biopsy site for hemorrhage. An absorbable gelatin sponge and gentle pressure can be used to control minor hemorrhage.

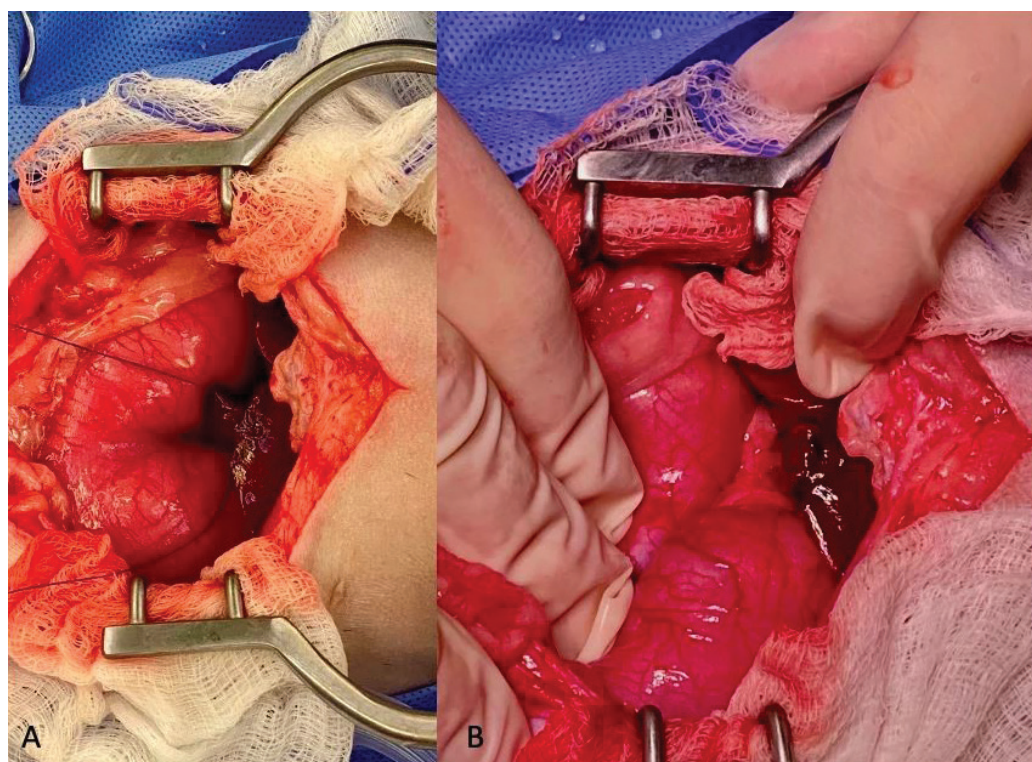


Figure 1: Guillotine technique: The suture has been tightened around a peripheral area of the liver (A). After completing the knot and transection distal to the ligature, the site is inspected for evidence of hemorrhage (B).

Punch biopsy technique

The punch biopsy technique is best used when sampling centrally located areas of hepatic parenchyma and focal lesions. Keep in mind when using this method that six to eight portal triads at minimum are recommended for accurate results; this can be achieved using a 6-mm Baker's biopsy punch.²

1. Isolate a focal lesion or centrally located area for biopsy sampling.
2. Prior to sample collection, prepare a piece of absorbable gelatin sponge roughly the same size of the

sample you plan to collect. This will be packed in the site after removal of the sample. Alternatively, use the same biopsy punch to cut a piece of gelatin sponge.

3. Press the biopsy punch against the parenchyma and advance in a rotating clockwise and counterclockwise motion (Figure 2). Ensure that you advance less than half the thickness of the lobe to avoid the large hepatic veins located on the dorsal surface of the liver.

4. Using Metzenbaum scissors, cut the deep margin of the sample from its attachment within the parenchyma.
5. Prepare for some hemorrhaging from the biopsy site after removal of the sample. Insert the previously prepared gelatin sponge into the hole to promote hemostasis.

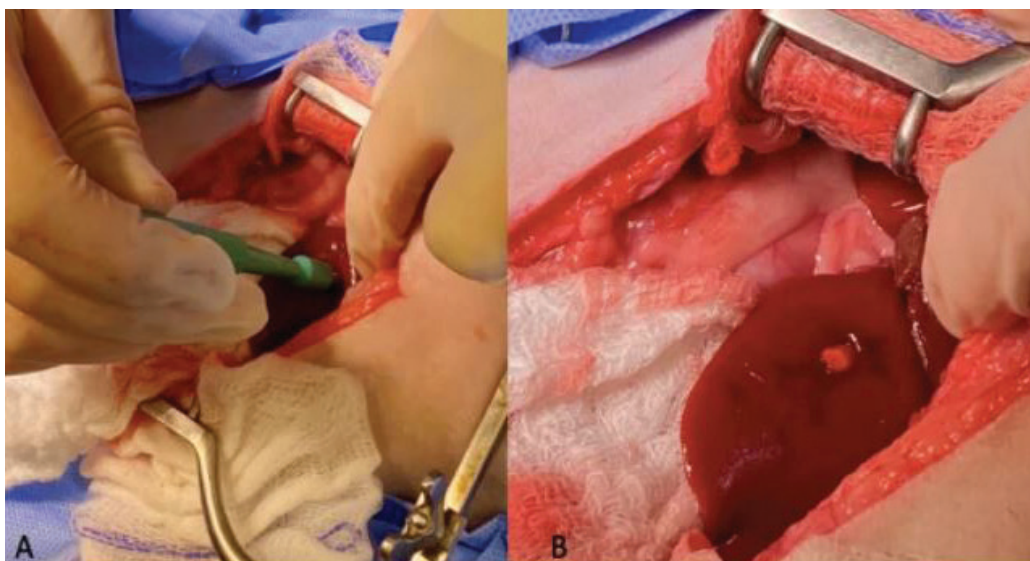


Figure 2: Punch biopsy technique: A punch biopsy instrument is used to sample a central area of the liver (A). Following tissue removal, a piece of absorbable gelatin sponge is placed in the defect (B) to attain hemostasis.

General considerations

To improve access to the liver, be sure to extend the laparotomy incision to the xiphoid process of the sternum. Sterile laparotomy pads can also be placed gently between the diaphragm and the liver to improve visualization. Using radio-opaque gauze markers and counting the number of surgical sponges prior to closure of the abdomen are essential.

Before closure, it is important to ensure there is no evidence of hemorrhage. While the techniques described here should produce minimal bleeding, there is always a risk for serious complications. Furthermore, patients with significant hepatic dysfunction should always be evaluated for coagulation abnormalities prior to surgery.

Conclusion

Performing open surgical liver biopsy using a guillotine or punch biopsy technique is a valuable skill any general

practitioner can learn. The diagnostic information obtained can ultimately improve the level of care provided to patients.

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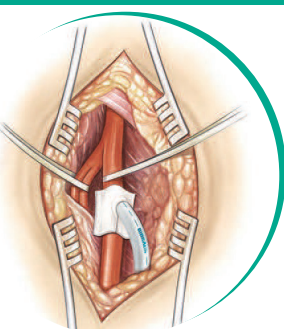
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1. Which of the following are advantages of open surgical biopsy over fine needle aspirate?

- a. Large enough sample for histopathology, not only cytology as with FNA
- b. Better able to visualise areas of pathology and get a biopsy of an affected site
- c. Easier to control haemorrhage, should it occur
- d. None of the above – minimally invasive FNA is always better when possible
- e. A, B and C

2. Which of the following statements is NOT correct, regarding the choice between laparoscopic and open surgical liver biopsies?

- a. Both can be used to obtain samples for histopathology, the gold-standard for hepatic evaluation
- b. Both can be used to evaluate the liver visually and select visibly affected areas for biopsy
- c. Laparoscopy is preferred for high-risk cases due to lower surgical morbidity associated with this technique
- d. Both are considered minimally invasive surgical techniques
- e. Both can be carried out by a general practitioner, providing they have had appropriate training in handling the laparoscopic equipment

3. Select the statement which is correct for the guillotine technique of liver biopsy.

- a. It is used to resect an entire liver lobe at a time
- b. It is used to obtain a sample from the edge of a liver lobe
- c. It can only be used when there is a pedunculated mass on the liver
- d. It can only be used in focal disease and is not appropriate when conditions diffusely affecting the liver parenchyma are suspected
- e. It cannot be used in cats, since their liver is not sufficiently lobulated

4. Select the correct method to handle the biopsy sample area from the options below

- a. Place a stay suture through the edge before removing it and use it to transfer the sample to formalin. Do not touch the sample at all.
- b. Take the free edge gently in your hand before excising it and transfer it to formalin. Do not use instruments.
- c. Place a single pair of artery forceps distal to the ligature and cut between the forceps and the ligature.
- d. Place two pairs of artery forceps, parallel, distal to the ligature and cut between them. Transfer the sample to formalin using the attached pair of forceps
- e. Due to the size of the sample, particularly careful handling is not required since there is sufficient tissue.

5. Which is the correct sequence of actions when taking a guillotine biopsy?

- a. Sharp dissection from the edge of the lobe, place a ligature with surgeon's or friction knot around any large vessels uncovered, remove sample, tighten ligature
- b. Place curved forceps of a sufficient size to crush the desired ligature zone over the edge of the lobe, place ligature, use sharp dissection to remove sample

- c. Select easily accessible margin, place ligature suture with a surgeon's or friction knot, tighten and secure with additional throws, use sharp dissection to remove sample
- d. Select easily accessible margin, use cutting cautery to remove the desired sample, use ligature sutures only to control large bleeding vessels (the guillotine name is only historic)
- e. Any of the above would be acceptable

6. In which case would a punch biopsy be preferred over a guillotine biopsy?

- a. When the animal has a bleeding tendency
- b. When liver function is compromised and it is preferred not to resect a whole section of liver
- c. When disease is focal and lesions are visibly smaller than the size of the biopsy punch
- d. When biopsies need to be taken from the centre of a liver lobe
- e. When trying to avoid cutting a bile duct

7. What is the minimum structure required for accurate histopathology?

- a. 1 portal triad (preferably one per sample over several samples)
- b. 2 – 4 portal triads
- c. 6 – 8 portal triads
- d. 15 – 20 portal triads
- e. 20 – 30 portal triads and a minor bile duct

8. What is the smallest size biopsy punch recommended to guarantee the required number of portal triads will be included in a single punch biopsy sample?

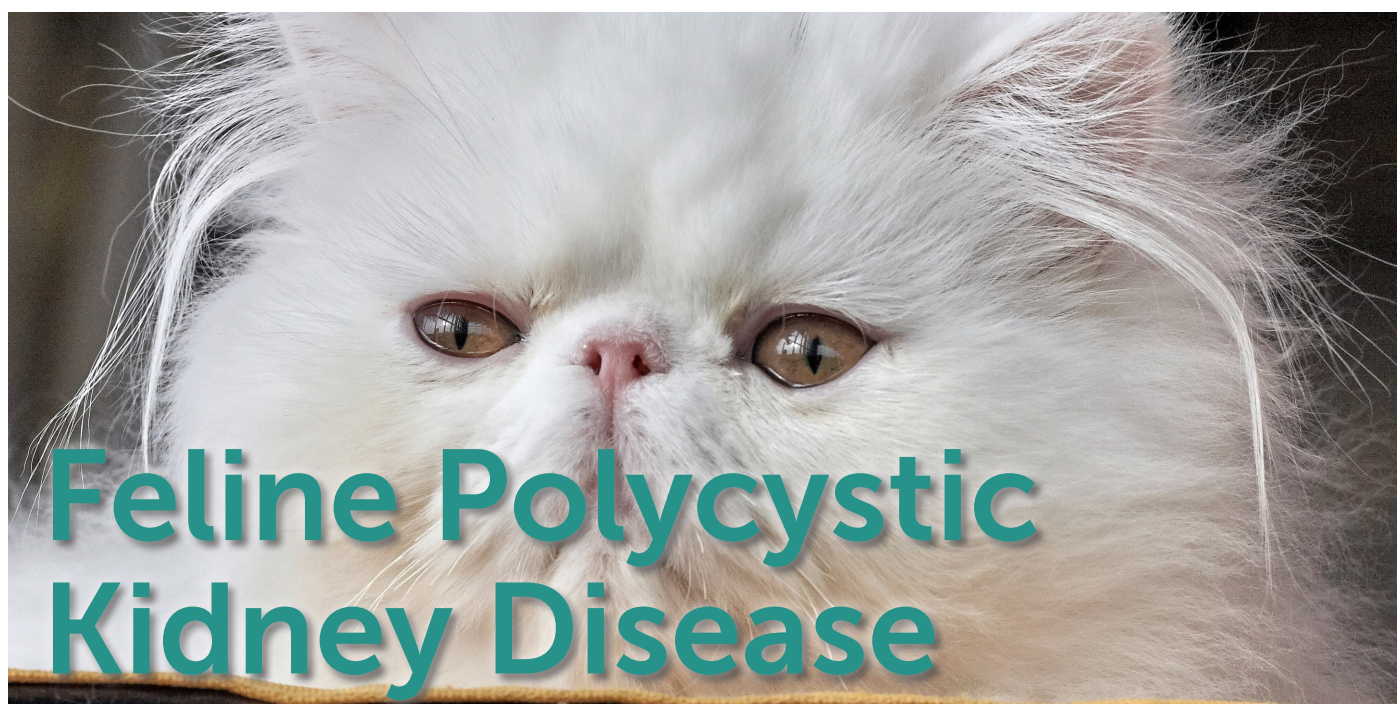
- a. 3mm
- b. 4mm
- c. 5mm
- d. 6mm
- e. 7mm

9. Which of the following is incorrect, when describing the process of taking a punch liver biopsy?

- a. The ideal is to take a full-thickness punch, incorporating both dorsal and ventral surfaces of the liver
- b. Absorbable gelatin sponge can be packed into the hole left by the biopsy punch to control bleeding
- c. Some bleeding is to be expected from punch biopsy lesions
- d. The gelatin sponge can be cut to required size using the same punch that the biopsy was taken with
- e. The biopsy punch should be advanced slowly into the parenchyma, using clockwise-anticlockwise twisting motions

10. Which of the following techniques can be used to improve visualisation of the liver during open surgical liver biopsy?

- a. Extending the laparotomy incision cranial to the xiphoid process
- b. Extending the laparotomy incision cranial to the umbilicus
- c. Inserting sterile swabs between the diaphragm and the liver
- d. A and C
- e. B and C



PDK affects 6% of the total feline population around the world.

Polycystic kidney disease (PKD) is a genetically inherited disease most commonly found in Persian cats. This condition causes the formation of multiple cysts in the kidneys (in the renal parenchyma), which are present at birth and grow larger as the cat ages, eventually disrupting the normal functioning of the kidneys. Although most abundantly found in the Persian breed (around 30%), the disease also affects breeds that have been developed using Persian bloodlines, such as Exotic Shorthair, American Shorthair, British Shorthair, Himalayan, Burmilla, Ragdoll, Neva Masquerade, Maine Coon, Chartreux and Chinchillas.

All cats who have the mutation for PKD will have cysts in their kidneys, but the number of cysts and the rate at which they grow will differ between cats. In most cases, these cysts will slowly enlarge, with the affected cat showing very few signs of the disease until later in life. There are unfortunately no predictive markers for how rapidly the disease will progress, but the average onset of symptoms starts at around age seven. The cysts can range in sizes from less than a millimeter to larger than a centimeter in diameter.

PKD is caused by an autosomal dominant mutation with complete penetrance within the *PKD1* gene. The variation is characterized by the substitution of a cytosine with an adenine at position 3 284 of exon 29 (c.10063 C>A), resulting in a premature stop codon in the messenger RNA. This generates a mutated protein by causing a 25% loss of the C-terminal in the formation of the polycystin-1 protein. To date, no affected cats have been found to carry homozygous copies of the mutation, which strengthens the idea that the mutation is lethal *in utero* when two copies of the mutation are present. The mutation seems to play a significant role in

the differentiation and proliferation of the tubular epithelium. Cysts seem to appear when the balance between tubular activation, degeneration, apoptosis, and necrosis is disturbed. The pathogenesis of the disease could therefore be related to the induction of cell death in the affected cells.

The clinical signs for PKD are not pathognomonic and are typically the same as those associated with other kidney diseases: polydipsia, polyuria, vomiting, nausea, general dehydration, pale mucous membranes, decreased appetite, anorexia, weight loss, lethargy, blood in urine, high blood pressure, bad appearance of coat, gastrointestinal disorders. Once disease has progressed, bosselated kidneys may be detected.

Diagnosing PKD should not be based on clinical features alone. Breed, clinical presentation, medical history, genetic testing, and ultrasound screenings should also be taken into consideration before making a diagnosis. Genetic testing can confirm the presence of the causative mutation, but it cannot give an indication of how far the disease has progressed or how large the cysts have grown. Although intravenous urography and radiography can be successfully used to diagnose more advanced cases, ultrasound imaging remains the method of choice, as it allows the veterinarian to see the severity and progression of the disease. While performing the imaging, it should also be taken into consideration that cysts might be more difficult to detect in the medulla than in the cortex. This is due to the echogenicity of the medulla, and that the hypoechoic nature of the medulla can lead to false positives. Estimates of Total Kidney Volume (TKV), Total Cyst Volume (TCV) and Fractional Cyst Volume (FCV) can be used in conjunction with imaging to assess the disease

progression. X-rays can also be used to determine overall kidney function in advanced stages.

Table 1: Diagnosis criteria of feline PKD according to the age of the cat.
Data obtained from Guerra et al 2018.

Age (Months)	Diagnostic criteria for Kidney Ultrasound
<15	≥ 1 cyst
16-32	≥2 cysts
33-49	≥3 cysts
50-66	≥4 cysts

There is unfortunately no cure for PKD, but symptoms can be managed as they progress. Treatment strategies typically include those used in other chronic kidney diseases such as adapting their diets, providing them with appetite stimulants, fluid therapy, administering medications to reduce vomiting and nausea, treatment of pain and discomfort as well as to block phosphorus absorption. Although in theory, cysts could be drained of their fluid, it would be a losing battle. There are typically too many cysts to drain, and if the drainage was successful, they would fill up with fluid again over time. Affected cats should be screened every two to six months to monitor progression and symptoms. The main goal with management is to keep the kidneys healthy and working

for as long as possible. If bacterial infections and associated sepsis can be avoided, the short-term prognosis is favourable.

As PKD will eventually progress to kidney failure, the only way to prevent the occurrence of it, is to breed unaffected animals. Especially in breeds with ties to Persian bloodlines, breeding cats should be genetically screened for the disease before being mated. As this is a dominant mutation, a cat will require only a single mutated copy to develop the disease. It is therefore highly important that carriers are identified within a stud and removed from further mating to reduce the prevalence of this fatal disease.

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