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Editor's Note

Dear Collegues

This edition is has a nice cerebral "fuzzy logic" article on making a diagnosis. It's always good to make sure we are not falling into a rut.

Dr Venter has also written an article on pupillary light reflexes and anisocoria, remembering the first step is deciding which pupil is abnormal - the bigger one or the smaller one and that blindness is a function of the eyes as well as the nerves.

I hope you enjoy reading and wishing you all the best for the holiday season and hoping you get a chance to relax and spend time with family and friends. .

Liesel

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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Maximising Staff Performance in a Veterinary Practice

Andrew Christie
BComm (Business Management)

The terms 'motivating' and 'managing performance' are used almost interchangeably when vet business owners are describing the same thing – ensuring that staff are working at their full potential both individually and within their teams. In this, the first of two articles, motivating staff will be explored while the next article will focus on performance management. I am hoping that it is not just practice owners and managers that read this article, but also people that feel distinctly unmotivated. Both sets of people can use the concepts to stimulate a better working environment.

Introduction

Some years ago, I started consulting to a large practice in Gauteng. One of our goals was to increase turnover to reflect the potential of the practice. After some discussion between the partners and myself, we decided that this since the whole team was instrumental in increasing turnover, everyone should benefit and so we introduced a R50,000 incentive for each person, if the goal was achieved.

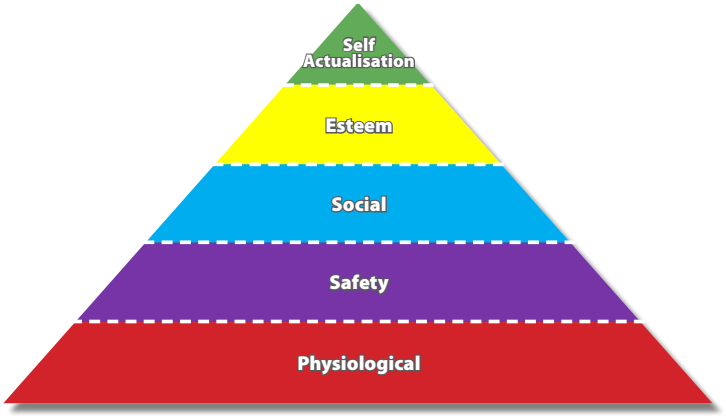
To my amazement, some of the senior vets approached me and asked that they not be included – they felt that they had ethical and professional obligations that meant they should not be trying to increase turnover but rather handling each case to the best of their ability.

Whether they were right or wrong is not something I can comment on, but it certainly reminded me of the golden rule of motivation – money doesn't motivate everyone; in fact, money doesn't motivate many…

Maslow's Hierarchy of Needs

In 1954, Abraham Maslow proposed his 'hierarchy of needs' model, illustrated below:

- Physiological needs include food, water, air and warmth
- Safety needs include physical safety, health, emotional security
- Social needs include acceptance by friends, peers, and family
- Esteem needs include the respect of peers and friends
- Self-actualisation refers to personal growth and includes personal development and spiritual awareness.



The basis of Maslow's model is that higher-level needs won't affect people until the lower-level needs have been met. A simple example – imagine you have no job, and you haven't eaten for three days.

As you walk through a Checkers, you are feeling so hungry that you feel faint. In that position you are being driven by your physiological needs and could well steal a loaf of bread. You are not concerned about being caught and put in jail (safety needs), let alone any of any of the other higher-level needs.

So what does this mean for a veterinary practice?

The example that I use with vet students is that when they get their first private practice job and move out of home, they will be very aware of safety issues when choosing a property that is secure (safety needs). As they settle into that first job, they will be thinking about issues such as job security (safety needs) and once they feel more secure, only then will they want to make friends within the workplace (social needs) and then start earning the ongoing respect of their peers (esteem needs).

But Maslow's model tells us much more than this – his model can be used in considering all members of a practice.

Thinking back to my experience with the senior vets, instead of offering them a flat R50,000 we could have considered sending them to an international congress or facilitated further studies of some sort. This is because clearly their physiological, safety, social and much of their esteem needs were being met – by offering something appealing to their self-actualisation needs, it quite possibly could have been appealing to those vets, and would have ended up costing the practice the same amount.

Motivating different types of staff within a practice

I use Maslow's model as a rough guideline when introducing motivationary strategies at vet practices:

a. Kennel Hands / Grooms / Cleaners are at a low-income level and often have to travel far to get to work. Yes, in this case money would be well received, but in my experience, it can be difficult to explain why sometimes the money is more and sometimes it is less. Knowing that Kennel Hands are motivated by physiological and safety needs, I have found it works well where vouchers for Checkers (or similar), or meat parcels are used.

b. Receptionists / back-office staff can fall into a few categories. For example, there is a trend that retired people in the area to take on a few days work at their vet practice. This can be to get some extra money, of course, but often it is to get out of the house and socialise a bit. Employees such as this are clearly not having their social needs met and might be motivated by practice get-togethers, for example. Younger receptionists may enjoy their social needs being met at a practice, but they may be gaining experience for their future studies (self-actualisation) or needing to pay for rent or food (safety or physiological). The former could be 'rewarded' for good performance by being allowed to observe interesting procedures or surgeries, while the latter might be incentivised by money. However, if times are really tight and the employee is struggling with debt, they may appreciate a Woolies voucher more as this is something that can be used for food or clothing and not merely disappear into debt. Long-serving back-office people may want recognition of their input and value to the practice (esteem). This could be addressed by acknowledging their value through the promotion of the person or, more often, the creation of a senior position for the person. This would be accompanied by an increase in privileges, one of which may be an increase in salary.

c. Vets may be in almost any of the hierarchy. For example, a young vet moving to a new town for the first time may appreciate the practice owner finding suitable accommodation and paying the first 3 months rent on their behalf (safety needs) much more than they would being paid the money since they don't know the first thing about where is safe, where is convenient etc. Younger vets with experience tend to be more motivated at the 'esteem' and 'self-actualisation' level. They already know that they have superior qualifications to most people in the country, but they may find themselves wanting to excel in the eyes of their peers. Paying for vets to attend congresses, while giving them paid time off to attend, might be incentivising for them. Older vets may be motivated at the 'esteem' level but will be increasingly aware of the importance of family and perhaps asking themselves about The Meaning of Life. These 'self-actualisation' needs are particularly difficult to address, but increasing the amount of time they take off and by reducing their involvement in after-hours work will allow them to spend more time with family.

Money…

Most people at this point are still convinced that money is the main motivator. At best they may think that at least money will enable their employees to satisfy their own needs.

However, there are two things to bear in mind:

1. By addressing the need directly with your staff, you are showing that you care about them individually. Additionally, people may have motivated at a 'social' level but feel obliged to put any extra money into the bond or the children's schooling. By paying for a spa day, you ensure that they have been motivated at a social level.
2. Paying staff extra money means they will lose a portion to tax. In extreme cases, they may be paid an amount that pushes them into the next tax bracket, and they end up paying more in tax than the bonus paid to them.

Things to remember

1. **Sometimes levels may be skipped** – some people may satisfy their physiological and safety needs and immediately be motivated by self-actualisation needs such as learning a new skill or taking up a new hobby, while not having much in the way of social or esteem needs.
2. **Maslow is relative** – when I bought my townhouse, I was motivated more by security (safety needs) and accessibility to suitable shopping (physiological needs) than by living closer to my group of friends (social needs). This is very different to someone living in an informal settlement where violence could be a nightly occurrence and obtaining food is a struggle. However, even though the needs are vastly different in nature, in principle they remain the same – we were both motivated by safety.
3. **Maslow is a guideline** – Maslow is a useful tool to examine motivation in different people, but it is a summary, a way of trying to explain something that is very complex. I present the model simply as an alternative to the 'throw money at people' way of motivating.

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NEURO OPHTHALMOLOGY: Anisocoria and visual deficits in a nutshell

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Localizing lesions in patients with anisocoria with or without visual deficits can be challenging. A basic knowledge of the visual pathways is required.

The visual pathways

The visual pathway is a three-neuron pathway. The *first neuron* is the bipolar cell of the retina that receives impulses from the retinal photoreceptor cells namely the rods and cones.

The *second neuron* is the retinal ganglion cell. Bipolar cells and ganglion cells synapse in the inner plexiform layer of the retina.

The axons of the ganglion cells continue in the nerve fibre layer of the retina and come together to form the optic nerve. The optic nerve is in fact a tract of the central nervous system. It is surrounded by a myelin sheath for the rapid conduction of action potentials generated in the ganglion cells.

There are marked differences in the starting point of the myelin sheath between different species. Myelination begins at the level of the optic disk in the dog and more caudally in the cat. This results in the difference in the colour and shape of the optic disk.

The optic nerve leaves the eyeball through the perforations of the lamina cribrosa to form the retro bulbar portion of the optic nerve. The two optic nerves (left and right) enter the skull through the optic canals and join to form the optic chiasm.

In cats approximately 65 % of the fibres cross over and in dogs 75 %. The bundles exiting the optic chiasm are called the optic tracts. The fibres that cross in the optic chiasm originate in the medial (nasal) area of the retina.

In other words, fibres originating from the left medial retina and right lateral retina project to the right cerebral cortex. Decussating fibres do not take a direct route through the optic chiasm.

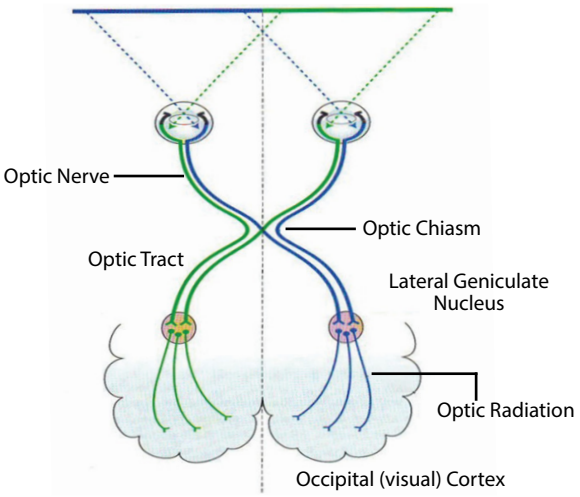


Figure 1: *The visual pathways. Images from the medial aspects of the retina is projected to the contralateral visual cortex.*

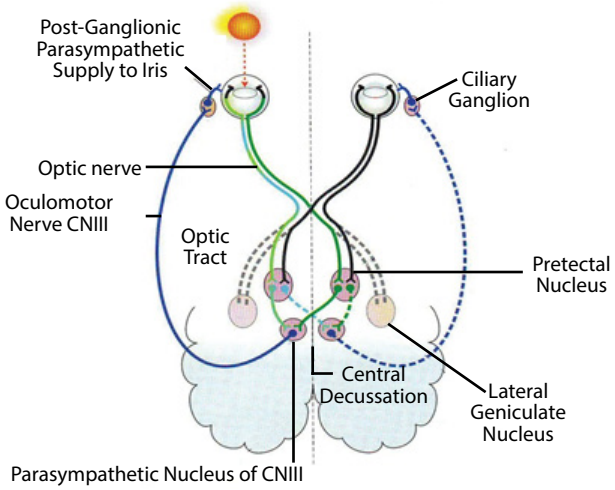


Figure 2: *Pupillary light reflex pathways. Note the 20 % fibres branching before the lateral geniculate nucleus to the pretectal nucleus in other words not contributing to vision but responsible for the PLR.*

The *third neuron* in this three-neuron pathway is the dorsal lateral geniculate nucleus. Approximately 80 % of the ganglion cell axons synapse with the third neuron in the lateral geniculate body. The dorsal lateral geniculate serves as a relay station to transfer information from the optic tract to the visual cortex via the optic radiation.

The other 20 % of the axons do not reach the visual cortex, but synapse in the rostral colliculus.

These 20 % synapse with neurons of three possible pathways:
- Pathways constricting the pupil.
- Pathways co-ordinating eyeball movements.
- Pathways controlling turning of the head and neck.

Lesions in the visual pathways

Lesions in patients with visual and or pupillary light reflex deficits or anisocoria may be divided into one of three categories:
- Blind patients with normal pupillary light reflexes (PLRs)
- Blind patients with abnormal PLRs
- Visual patients with abnormal PLRs

Lesions in blind patients with normal pupillary light reflexes

Based on the anatomy of the PLR pathway, the size of the pupils and their response to light are normal in animals that are blind because of disease processes in central visual pathways, **after** the afferent fibres of the PLR have diverged to the midbrain.

- These patients present with a history of blindness. The menace response is absent, but it is important to note that the dazzle and pupillary light reflexes are present and the eye appear normal on ophthalmoscopy.

Bilateral cerebral lesions that cause blindness include prosencephalic hypoplasia, hydranencephaly cerebral contusion, cerebral oedema, viral encephalitis, thrombotic meningoencephalitis, inflammatory diseases such as granulomatous meningoencephalitis (GME), metabolic disorders (hypoglycaemia, hepatic encephalopathy), poisonings, and nutritional and storage diseases.

The most common causes of a unilateral cerebral lesion with contralateral visual field deficit are neoplasms in small animals and abscesses in large animals. Unilateral lesions are very seldom seen in practice because most patients will not show any obvious clinical signs of unilateral blindness.

Lesions in patients with visual deficits and abnormal pupillary light reflexes

The afferent fibres responsible for the PLR and cortical vision run together from the retina through the optic nerve, optic chiasm, and proximal optic tract. Therefore, lesions in this common pathway cause blindness as well as an abnormal, although not necessarily absent, PLR.

Afferent lesions that interrupt this pathway occur in the retina, optic nerve, or optic chiasm. Bilateral optic tract lesions are very uncommon because the tracts are spread out over a relatively large area.

A single optic tract lesion is rare and may cause no PLR abnormality because of the decussation in optic chiasm and in the pretectal nucleus and oculomotor nucleus.
- A patient with a unilateral lesion in the retina or optic nerve has no menace response in that eye. The pupil on the affected side is mydriatic, but not fully dilated because of the indirect stimulation from the unaffected eye via the consensual pupillary reflex pathway.
- Light directed into the affected eye causes no response in either eye. Light directed into the unaffected eye elicits a bilateral response, though the magnitude of the constriction in the directly stimulated eye will be greater than in the indirectly stimulated eye.
- Another useful diagnostic procedure is the swinging flashlight test that will be positive in these cases.

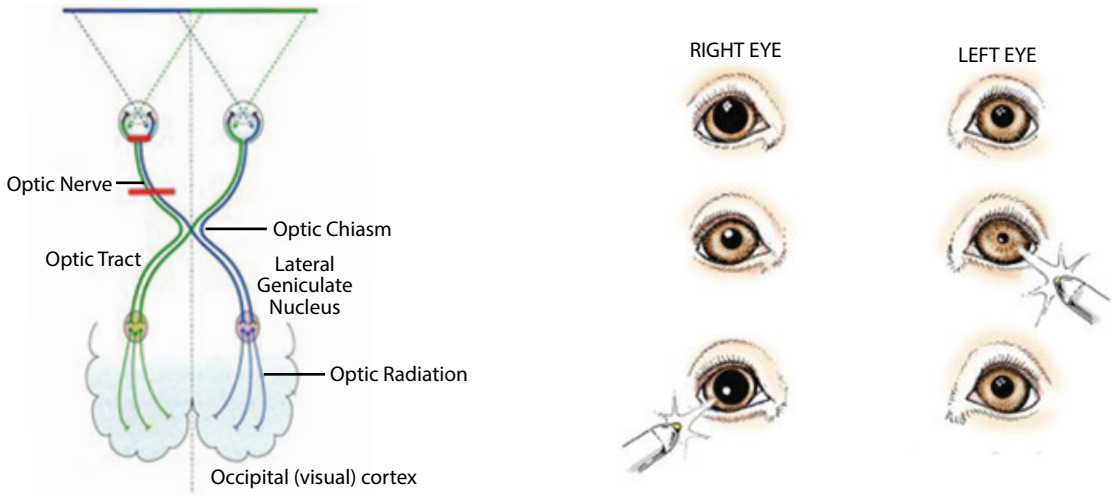


Figure 3: *Pupillary light reflexes in patient with retinal / optic nerve lesion affecting both vision and PLR. Stimulating the unaffected eye leads to both a direct and consensual PLR. Both direct and consensual PLR is absent on stimulating the affected eye. (Image Source Maggs DJ, Miller PE, Ofri R. Slatter's Fundamentals of Veterinary Ophthalmology. 2013.)*

Common ophthalmic causes of unilateral lesions resulting in PLR and visual deficits include retinal detachment, glaucoma, and retrobulbar abscess or neoplasia. Trauma to the optic nerve is another common cause of unilateral lesions. One of the most common causes for traumatic optic nerve injury is proptosis.

Bilateral retinal diseases include retinal detachment, end-stage inherited retinopathies, sudden acquired retinal degeneration (SARD), and glaucoma. The most common bilateral optic nerve disease to affect vision and PLR is optic neuritis.

The optic chiasm may be compressed by extramedullary space-occupying lesions near the hypophyseal fossa. Lesions affecting the entire optic chiasm will lead to total blindness as well as mydriatic nonresponsive pupils.

Due to the cross over of fibres in the optic chiasm lesions affecting only the central aspect of the chiasm will lead to a partial visual deficit and a pupillary light reflex if the lateral aspect of the retina is stimulated. Stimulation of the medial retina will not elicit a PLR. Central optic chiasmal lesions can occur secondary to pituitary neoplasia.

Lesions causing pupillary light reflex abnormalities in visual patients.

Abnormalities in the parasympathetic innervation to the eye leads to a fixed, dilated pupil on the affected side. The indirect PLR to the contralateral eye will be present, but the indirect PLR from the contralateral eye will be absent.

If the lesion is located before the ciliary ganglion only the pupil will be affected [internal ophthalmoplegia]. Post ganglionic lesions will lead to ventrolateral strabismus caused by loss of innervation to the dorsal, medial, and ventral recti and the ventral oblique muscles innervated by the oculomotor nerve as well as an abnormal PLR.

Ptosis of the upper eyelid is also present caused by loss of innervation to the levator palpebral muscle. Vision in these

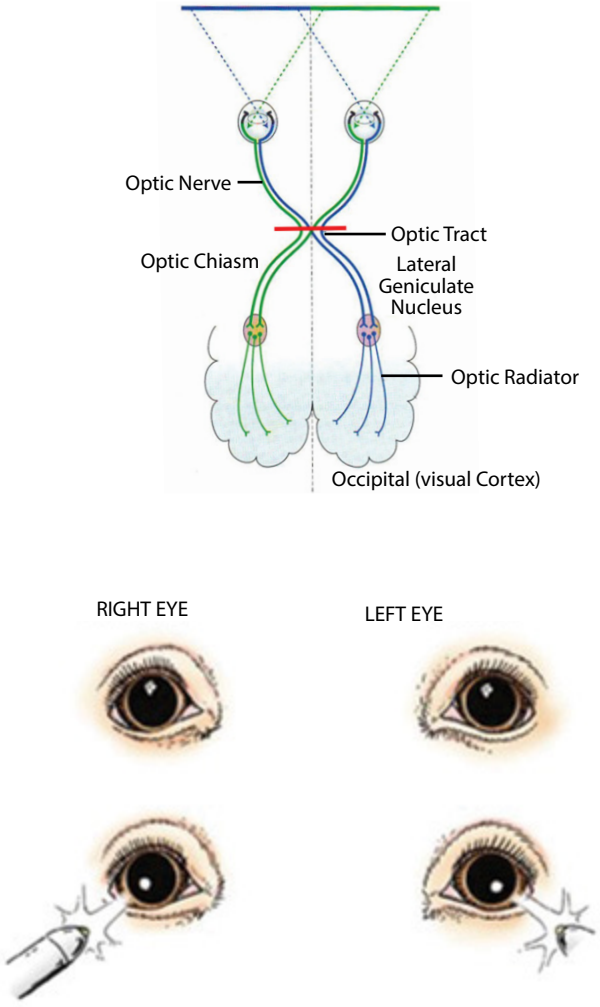
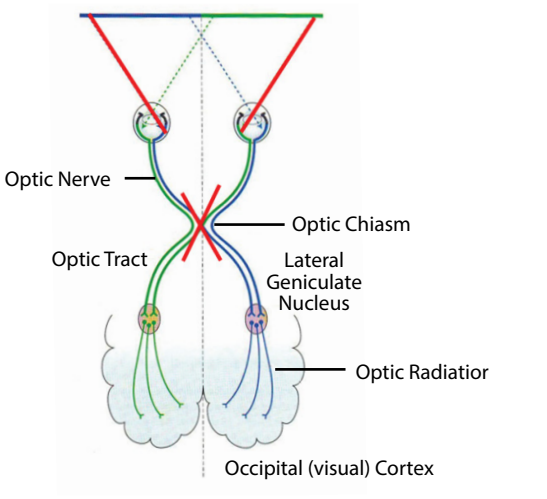


Figure 4: *Pupillary light reflexes in a patient with lesion affecting the entire optic chiasma. Bilateral mydriatic pupils with no direct or consensual PLR as well as blindness. (Image Source Maggs DJ, Miller PE, Ofri R. Slatter's Fundamentals of Veterinary Ophthalmology. 2013.)*



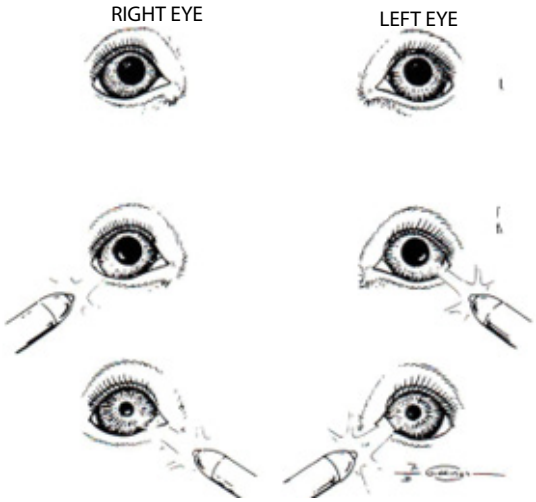


Figure 5: *Pupillary light reflexes in a patient with a lesion affecting the central optic chiasma. Bilateral partially mydriatic pupils with no direct or consensual PLR if the light source is directed onto the medial retina but PLR present when light sourse is directed onto the lateral retina.*

patients is unaffected but they may show signs of photophobia due to increased amounts of light reaching the retina through the dilated pupil.

Non--neurological causes of pupillary light reflex abnormalities

PLR abnormalities and anisocoria may also be caused by several processes that are unrelated to neurologic disease:

Iris degeneration with atrophy causes ipsilateral mydriasis with a variable response to light. This is reasonably common in geriatric patients. The presentation is due to the fact that the iris sphincter muscle is more extensively affected than the iris dilator muscle. Iris hypoplasia is a congenital disorders manifested as lack of iris tissue, and may result in an enlarged pupil.

Anterior uveitis, often secondary to corneal ulcerations causes stimulation and spasm of the iris constrictor and ciliary muscles, resulting in miosis.

Superficial corneal ulcerations lead to reflex spasm of ciliary body muscles and consequent miosis.

Prior treatment with atropine or other parasympatholytic agents can cause a fixed, dilated pupil. Atropine given to a normal eye may cause PLR deficits for up to days. It is very important to rule out this possible cause before extensive further diagnostic testing is performed.

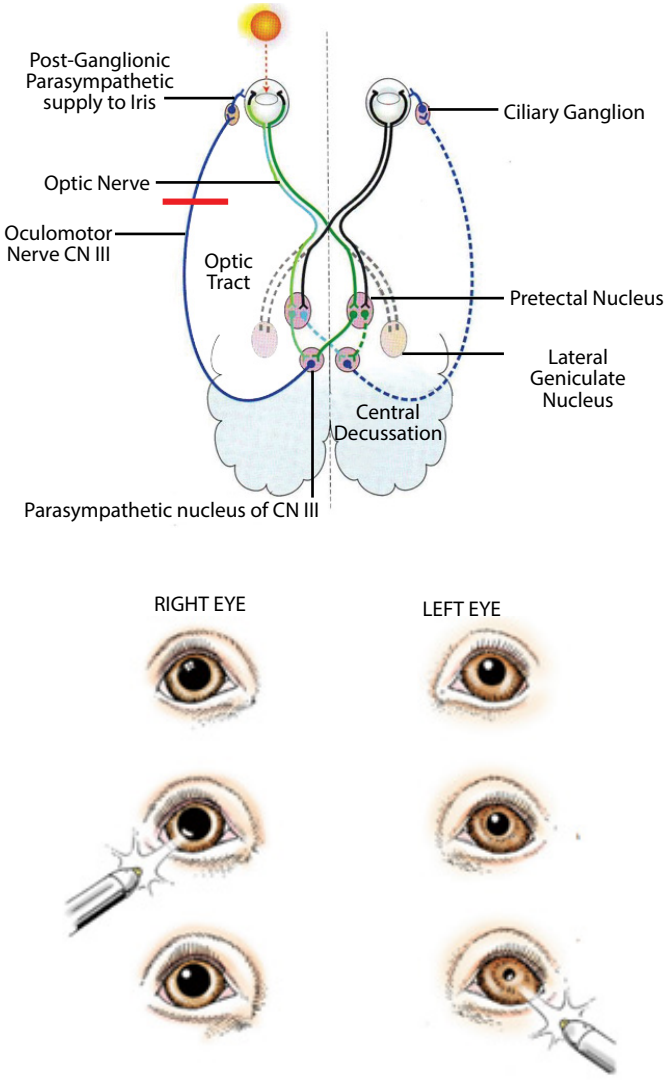


Figure 6: *Pupillary light reflexes in a patient with internal ophthalmoplegia in the right eye.*

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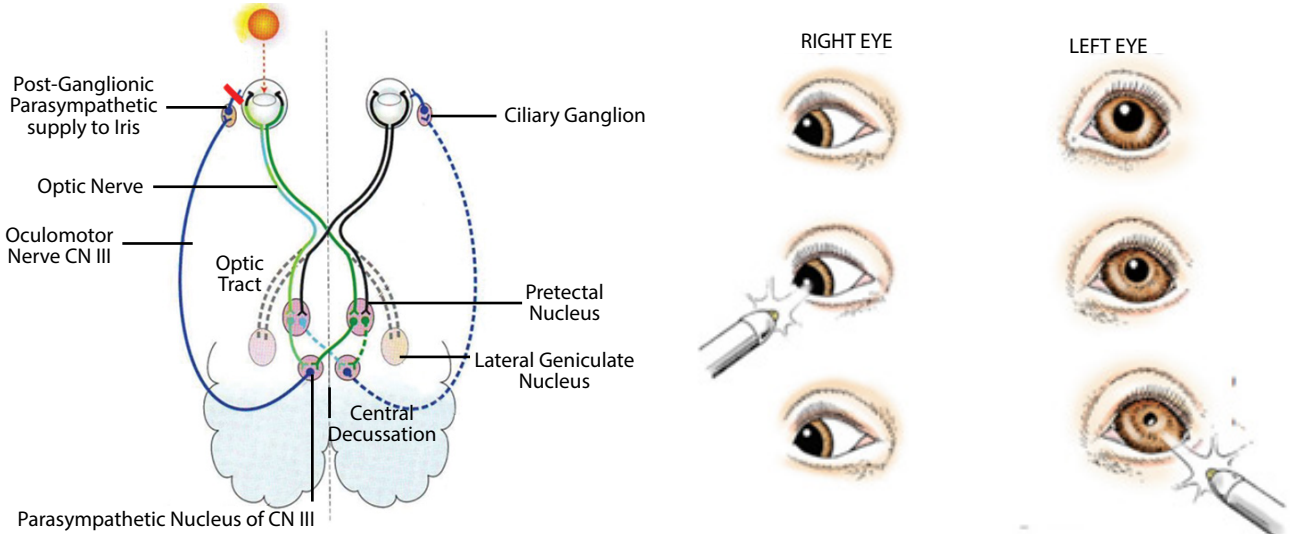

Figure 7: *PLRs in a patient with total ophthalmoplegia. Beside the abnormal PLR there is also ptosis of the upper eyelid caused by denervation of the levator palpebral muscle and ventrolateral strabismus (exotropia) owing to denervation of the dorsal, ventral, and medial recti muscles and the ventral oblique muscle.*

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1 What percentage of optic nerve fibers cross over at the optic chiasm in dogs?

A. 20%
B. 35%
C. 50%
D. 65%
E. 75%

2 Name the first neuron in the three-neuron visual path way.

A. Photoreceptor cells
B. Retinal horizontal cell
C. Retinal bipolar cells
D. Retinal Ganglion cells
E. Lateral geniculate nucleus

3 Which one of the following statements regarding the optic chiasm is correct?

A. The fibres that cross in the optic chiasm originate in the lateral area of the retina.
B. Fibres originating from the left medial retina and right lateral retina project to the left cerebral cortex.
C. Decussating fibres take a direct route through the optic chiasm
D. A lesion affecting the optic chiasm will always lead to complete loss of the pupillary light reflex.
E. Optic chiasmal lesions leads to visual as well as pupillary light reflex deficits.

4 What percentage of retinal ganglion cell axons are responsible for vision?

A. 100 %
B. 90 %
C. 80 %
D. 50 %
E. 20 %

5 Which one of the following statements regarding blindness due to visual cortex lesion is true?

A. It will always lead to anisocoria
B. Unilateral visual cortex lesions will lead to ipsilateral loss of a dazzle reflex
C. Patients present with visual deficits but normal pupillary light reflexes
D. Pupils are mydriatic and non-responsive
E. Unilateral visual cortex lesions will lead to contra lateral loss of a dazzle reflex.

6 What is the typical presentation of a patient with a unilateral lesion in the retina or optic nerve?

A. There will be no menace response in the eye with ipsilateral complete mydriasis
B. There will be no menace response in the eye with ipsilateral partial mydriasis.
C. Light directed into the affected eye causes no direct pupillary reflex but the consensual reflex to the contra lateral eye is normal
D. Light directed into the unaffected eye elicits a unilateral response
E. The swinging flashlight test that will be negative.

7 Regarding unilateral abnormalities in the parasympathetic innervation to the eye which of the following statements are correct?

A. The indirect PLR to the contralateral eye will be present, but the indirect PLR from the contralateral eye will be absent.
B. The pupillary direct pupillary reflex as well as dazzle reflex on the affected eye is abnormal.
C. The patient will have no menace response in the affected eye.
D. The indirect PLR from the contralateral eye will be present.
E. Dorsal strabismus on the affected side is likely.

8 Regarding axons synapsing in the rostral colliculus which one of the following statements are not correct?.

A. They mediate constriction of the pupil.
B. They mediate co-ordination of eyeball movements.
C. They mediate the turning of the head and neck in the direction of the visual stimulus.
D. Loss of innervation to the rostral colliculus will lead to mydriasis
E. Loss of innervation to the rostral colliculus will lead to visual deficits.

9 What is the primary function of the third neuron in the visual pathway?

A. It contributes to the dazzle reflex
B. It contributes to pupillary light reflexes
C. It relays information from the optic tract to the visual cortex
D. It controls eye movements
E. It forms the optic nerve

10. Regarding the afferent fibers responsible for the PLR and cortical vision, which one of the following statements is not true?

A. The fibers run together from the retina through the optic nerve, optic chiasm, and proximal optic tract.
B. Lesions in this common pathway cause blindness as well as an abnormal PLR.
C. Afferent lesions that interrupt this pathway may occur in the retina.
D. Afferent lesions that interrupt this pathway may occur in the optic chiasm.
E. Afferent lesions that interrupt this pathway may occur in the optic radiation.

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Topical Skin Antibacterial

Therapy for Superficial Pyoderma

Liesel van der Merwe
BVSc MMedVet (Med) Small Animals

The increase and increasing incidence of antibiotic resistant infections, especially methicillin resistant staphylococcus (MRS) can be partially blamed on indiscriminate use of antibiotics in human and veterinary medicine. Multiple drug resistant bacteria are resistant to three or more different classes of antibiotics. The ability of a bacteria to develop a biofilm limits the efficacy of antibiotics.

Canine pyoderma, most commonly caused by *Staphylococcus pseudointermedius* is a major reason for systemically administration of antimicrobials in small animal practice. This is counterintuitive, as the large surface are of the skin is and the superficial nature of the infection is uniquely suited to topical application of medicants.

The topical route of medication is recommended for most eye, ear and superficial wound infections. Systemic therapy is only recommended for deeper infections such as cellulitis, otitis media and signs of systemic illness. Pyoderma treatment is dependent on the depth of the infection. Systemic antibiotics, preferably based on culture, is always indicated for deep pyoderma. Topical antibacterial therapy, wherever suitable, is recommended for superficial pyoderma.

Skin infections involving multidrug resistant (MDR) *S pseudointermedius* or Staph aureus (MRSA) have shown response to topical therapy where it is not only effective but has also been shown to reduce zoonotic transmission as well as between other pets, by reducing environmental contamination. The general cleaning effects of topical treatments such as shampoos have become relevant due to their decontaminating effect. Although MDR and MRSA are generally resistant to most or all clinically relevant anti-microbials, this resistance does not extend to topical antibacterials.

Topical treatment should shift from being an adjunctive treatment to becoming the primary treatment in many types of skin infections. They are also effective against bacterial biofilm.

The ideal topical product should:

- Have an active ingredient effective against target organism.
- Be easy to apply.
- Have an effective delivery vehicle.
- Provide adequate contact time.
- Have a residual effect.

In vitro studies show low MICs (minimum inhibitory concentrations) for antibacterial products licensed for topical canine use in pyoderma. Several clinical studies, albeit with small numbers, have shown good efficacy with chlorhexidine and fusidic acid products applied topically to superficial pyoderma. Other active ingredients for which some efficacy has been shown include: acetic acid; benzoyl peroxide, ethyl lactate; medical honey; povodine iodine; salicylic acid; silver sulphadiazine and sodium hypochlorite (bleach). However, there is no strong veterinary evidence to support their use.

Resistance to topical treatments/antiseptic agents is associated with the expression of genes associated with the development of efflux pumps in bacterial cell membranes/ walls. No such genes have been identified in 100 isolates of MDRSP – thus resistance to commonly used antibacterials in veterinary medicine is low.

Classical topical antibacterials microbials include chlorhexidine, benzoyl peroxide and ethyl lactate.

Chlohexidine

Chlorhexidine is the most studied antibacterial. It is clinically effective when used for superficial pyoderma, and shampoos are available in 2-4% concentrations. The mechanism of action is complex and includes disruption of the bacterial cell wall. Active ingredient persists on the skin for four days and may persist on hairs for 7 – 14 days.

In a study comparing the use of chlorhexidine topical therapy against BID use of amoxyxillin clavulanic acid for superficial pyoderma, showed a relatively low MIC for clinical isolates of Staph. *pseudointermedius* and efficacy of the treatment. Efficacy compared to benzoyl peroxide is also published.

Azoles are combined with many formulations containing chlorhexidine, to provide an antifungal component. Miconazole also has some antibacterial activity against Staph. pseudointermedius in vitro, and recently using EM, it was noted that miconazole, disrupted bacterial cell membrane permeability.

Clotrimazole also shows antibacterial to *S pseudointermedius,* regardless of their methicillin resistance status. Additionally clotrimazole was effective in some instances where miconazole failed.

Benzoyl peroxide and ethyl lactate

Benzoyl peroxide breaks down when in contact on the skin and water, to benzoic acid and oxygen which generates oxygen radicals causing oxidative damage to the bacteria, an effect not associated with development of bacterial resistance. Benzoic acid is also comedolytic and keratolytic – inhibiting epidermal proliferation and sebum production, which may cause drying of the skin if used too often. Ethyl lactate is lipid soluble and it penetrates into all skin layers, where it is hydrolysed by bacterial lipases into ethanol which solubilises lipids and lactic acid, which lowers the skin pH, resulting in its antibacterial action. Clinical trials have not proven an antibacterial effect of these products in topical formulations.

Sodium hypchlorite (Bleach)

Sodium hypochlorite is broken down to hypochlorous acid when in contact with water and this reaction generates superoxide radicals which are very effective against bacteria, viruses, spores and fungi. Is not toxic to human skin in concentrations 0.005%-0.01%. In humans it does not disrupt the skin barrier, has anti-inflammatory properties and is able to decreases pruritis. It is a low-cost, effective topical alternative in pets.

Medical honey

Medical honey is prepared with gamma radiation. Its antibacterial effect is due to the hygroscopic effects of honey as well as the high sugar content. Honey also has a low pH and contains glucose oxidase which produces hydrogen peroxide. Its' in vitro efficacy has been documented against staphylococcal species and some literature has shown efficacy against localised canine superficial pyoderma.

Silver compounds

Silver ions cause antibacterial effect by interacting with the thiols of bacteria, damaging the cell wall, affecting nucleic acids and inhibiting cell division. Silver sulphadiazine is used in veterinary medicine for pyoderma caused by Pseudomonas aeruginosa. It is also effective when used in solution with water (dilution 1:30).

Compliance

Perceptions that owners expect antibiotics for their pets may be up of challenge. The general population is much more aware of the development of antibiotic resistance and people are more open to the use of topical therapies. The risk of non-compliance is higher with topical treatment and extra time will be needed to explain the benefits. The frequency of application is very important in determining the efficacy. Application is generally twice-weekly for shampoos, with higher frequency applications of spray and mousse products. Owner compliance is essential to efficacy. Additional benefits of topical treatment includes cleaning which improves coat condition, decreases odours and also reduces environmental contamination. Mechanical disruption of biofilm and reduction of pruritis are additional benefits.

Conversational aides which may assist in this discussion include:

- Comparison to the use of topical treatment in humans for superficial infections
- Safety - only affected tissue will be exposed to the medication, not the healthy animals
- Reduction in the development of drug resistant bacterial in the patient and potentially the household.
- Topical treatment can be more tailor made to owners and patients needs.

So far there is no evidence of the development of resistance to topical antibacterial therapy . In vitro MIC s have remained consistently low. Poor response to treatments often can be ascribed to poor owner compliance, co-morbities or product formulation.

With topical treatment the active ingredient reached the site of infection directly, bypassing metabolism effect by the liver and dilution in the blood. This has been supported in recent trials where topical fusidic acid concentrations in the canine follicle exceeded the highest MICs described for *S pseudointermedius* in vitro.

Conclusions

In an era where antibiotic resistance is extremely common topical antibacterials with low resistance profiles are extremely important in managing superficial pyoderma in dogs. These antibacterials allow for a reduction in the use of antibiotics and decrease environmental contamination. There is, however, a need for more clinical trials evaluating the efficacy of many of these products.

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Retinal Imaging to Assist Diagnosis of Systemic Hypertension

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Introduction

Systemic hypertension is diagnosed where systolic blood pressure (SBP) is elevated and may be classified into four categories on the risk of future target organ damage (TOD). Normotension is defined as SBP <140 mmHg and there is minimal risk of TOD. Cats with SBP 140-159 mmHg are classified as prehypertensive, and there is a low risk of TOD but an increased risk of becoming hypertensive in the future. Where SBP is 160-179 mmHg the risk of TOD is moderate, and the risk of TOD increases to high when SBP is >180 mmHg.

An elevated SBP may be measured as a result of situational stress ('white coat hypertension') or because of pathological hypertension. To differentiate the situational and pathological hypertension, persistence of hypertension or presence of TOD should be observed. In humans most hypertension is defined as primary, however, interestingly around 80% of feline hypertension is associated with an underlying condition[1]. Such conditions include chronic kidney disease, hyperthyroidism and primary hyperaldosteronism[2]. It is therefore important to screen any hypertensive cats for concurrent diseases and also to screen any cats with the above diseases for hypertension.

Epidemiological studies indicate that the risk of hypertension increases with age[3] and hypertension affects up to 40% of cats over 7 years of age[4]. Hypertension can cause TOD to several organs, including the eyes, kidneys, heart and brain[5]. Severe hypertension can result in serious clinical consequences including blindness, chronic kidney disease (CKD) progression, proteinuria, ventricular hypertrophy, and hypertensive encephalopathy.

Fundic examinations can be used to look for evidence of early ocular TOD to assist in the differentiation of situational and pathological hypertension, or to screen cases for cases with ocular TOD [6] and select cases for follow up SBP measurement.

Diagnosis and management of hypertension

Often cats do not show any symptoms until severe target organ damage has occurred. Where hypertension is severe, cats may present blind due to haemorrhage or retinal detachment, with mydriatic pupils (ocular TOD) or with seizures (neurological TOD). As symptoms do not arise until late in the disease process, routine SBP monitoring is required to diagnose hypertension before symptoms arise. SBP can be measured using doppler

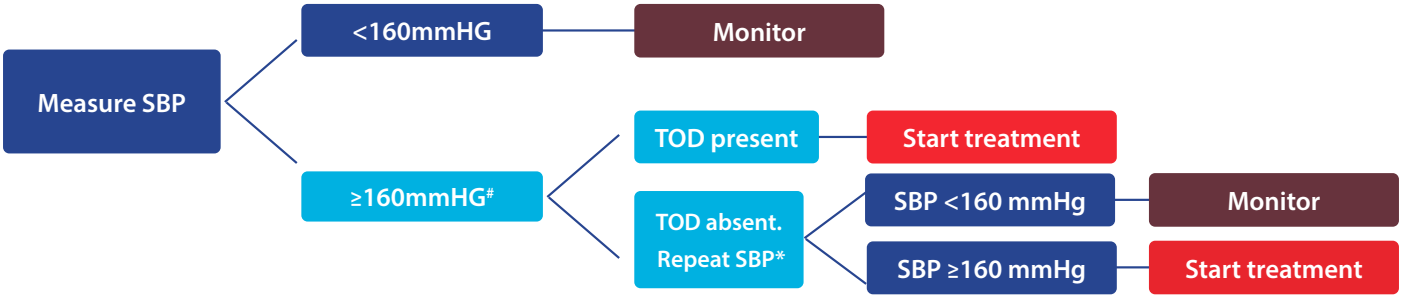

Figure 1: *Treatment flow chart adapted from ACVIM guidelines [7]*
 # Differentiate situational and pathological hypertension by confirming presence of target organ damage (including ocular, renal and neurological)
** Repeat SBP assessment recommended within 2-4 weeks*

sphygmomanometry or high definition oscillometry[7]. Traditional oscillometric devices should be avoided as these are unreliable in conscious patients.

Fundic examinations can be used to check for ocular TOD if SBP is >160 mmHg, and if lesions are present this confirms pathological hypertension and antihypertensive treatment should be initiated[7] (see Figure 1). Antihypertensive medications include the angiotensin receptor blocker, telmisartan (oral solution) or the voltage gate calcium channel blocker, amlodipine (tablet).

Despite increasing awareness that SBP should be measured routinely in practice and many guidelines recommending at least annual SBP measurement[7], it seems there are often barriers to blood pressure assessment in first opinion practices.

This may be a result of lack of equipment, adequately trained staff or due to a lack of time. Fundic imaging may therefore be a useful screening tool to improve detection of cases in which ocular TOD has arisen and SBP measurement is therefore essential[6].

Fundic examination

A dilated pupil is required to obtain a good view of the fundus; this may be possible in a darkened room or where cases have mydriatic pupils. Where additional pupil dilation is required, 1% tropicamide may be used to dilate the pupil and takes around 20 minutes to take effect.

There are several methods to examine the fundus, including direct and indirect ophthalmoscopy. Indirect methods are preferred as they provide a wider field of view than direct ophthalmoscopy. Indirect examinations can be performed with a pen torch and 20 Dioptre condensing lens, with a binocular system or with a fundic imaging camera.

Newer fundic imaging cameras have the benefit of taking images of the fundus quickly, and these images can then be saved in clinical records to aid in lesion monitoring or shared with colleagues to improve confidence in diagnosing lesions. Images can also be shown to pet owners to improve their engagement and understanding.

Owners that are shown lesions within their cat's eyes may better understand that their cat really is hypertensive despite them not

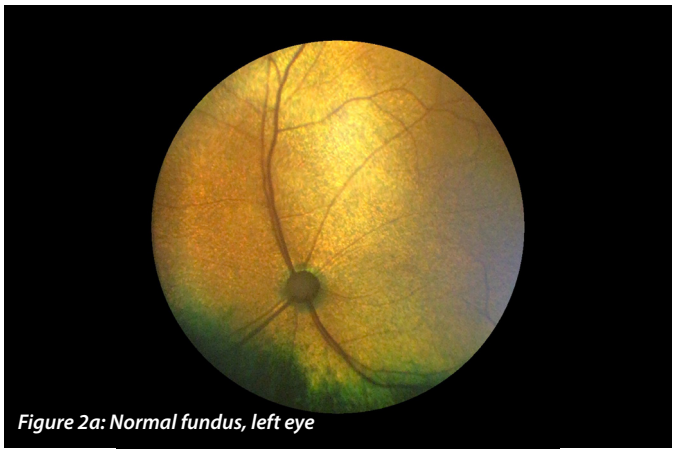

Figure 2a: Normal fundus, left eye

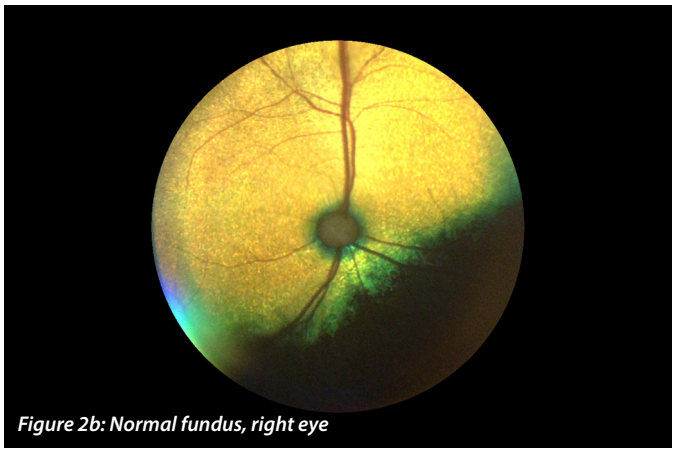

Figure 2b: Normal fundus, right eye

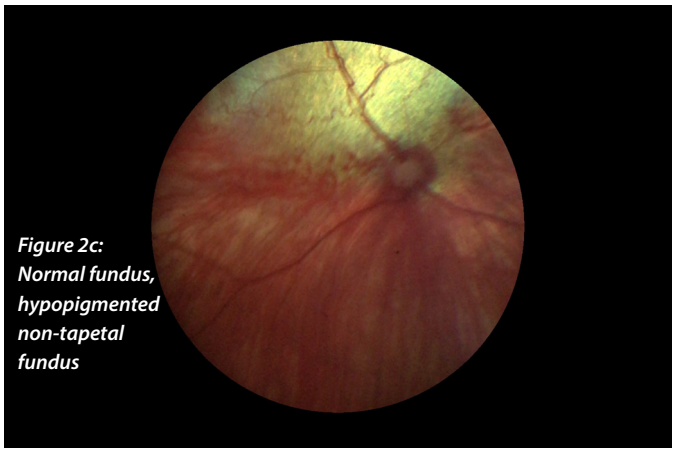

Figure 2c: Normal fundus, hypopigmented non-tapetal fundus

showing clinical signs, therefore potentially improving compliance with medications and recheck appointments.

Normal Fundus

The normal fundus (Figures 2a and 2b) consists of the optic nerve head, retinal vessels, extensive tapetal fundus and smaller non-tapetal fundus. In colourpoint cats the fundus may be non-pigmented making choroidal vessels visible.

An example of a normal Siamese fundus is given in Figure 2c, here the non-tapetal fundus is non-pigmented. Some cases, including albino cats, may have no pigment across the whole fundus. It is important to differentiate this normal variation with fundic haemorrhages.

Hypertensive retinopathy

Both acute intraocular haemorrhage and retinal detachment may be apparent on a physical examination and confirm ocular TOD without the need for a detailed fundic examination. Where a fundic examination is required to detect ocular TOD evidence of bullae, retinal haemorrhages, retinal oedema and tortuous retinal blood vessels should be assessed.

Fundic examinations are required to detect less severe TOD including retinal haemorrhage, bullae, oedema or tortuous retinal vessels. Figures 3a and 3b show examples of cases with retinal bullae indicated with pink arrows and retinal haemorrhage depicted with a blue arrow.

Conclusion

Fundic imaging can be used to diagnose ocular TOD and differentiate pathological from situational hypertension. Fundic imaging may also be used to screen cases quickly and easily to identify patients with TOD that require SBP measurement. Routine screening for hypertension is essential to detect cases early and prevent future problems. This could be implemented within a vaccination appointment, however, as both blood pressure measurement and taking fundic images are non-invasive, there is huge potential to utilise nurses and other trained staff for these procedures. It may be feasible to set up screening assessments by veterinary nurses prior to veterinary appointments in a similar way to the human medical setting, where patients have blood pressure measured by nurses in advance of seeing doctors.

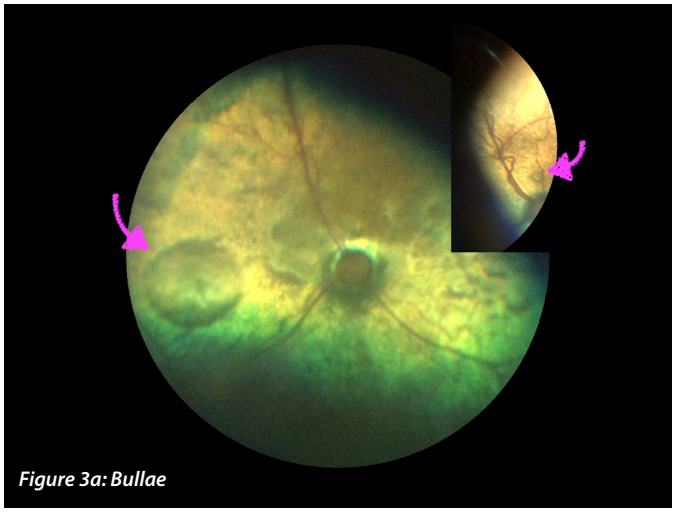

Figure 3a: Bullae

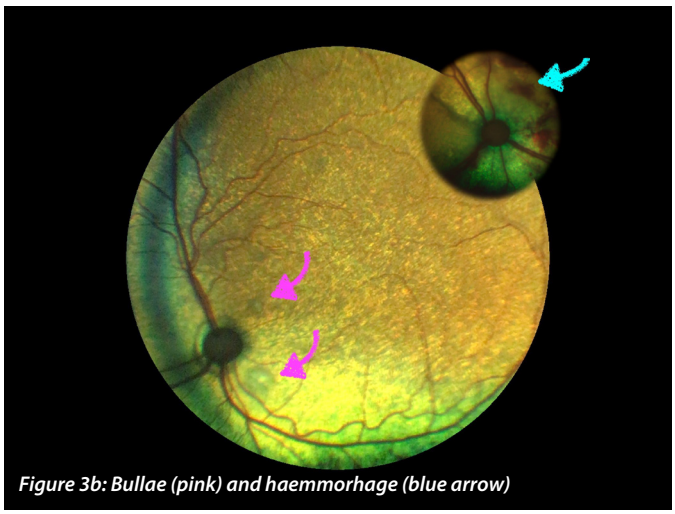

Figure 3b: Bullae (pink) and haemmorhage (blue arrow)

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MAKING A DIAGNOSIS:

How good are we? Can we improve?

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Introduction

For the practicing clinician most daily activities revolve around, or are based upon, deciding what is wrong with your patient (diagnosis) and deciding what to do about it (clinical decisions).

Although, we intuitively know that these diagnostic and clinical decisions are unlikely to be 100% accurate and/or logical, very few of us have ever stopped to ponder the actual degree of inaccuracy.

Furthermore, because we appear to be unaware that there is a problem, or the extent of the problem, we do not actively seek ways of improving our skills, if such methods exist.

In this paper we will examine the literature to discover how good we (biomedical scientists) appear to be at making diagnoses. We will examine what is known about the development of diagnostic ability and how it changes with time.

We will look at different diagnostic methods and examine whether of these are changed with time and increased expertise. We will then address the question as to whether we can improve our diagnostic ability or speed up the changes in diagnostic expertise.

What Is a Diagnosis?

- The diagnostic process is one of identifying disease in the patient by its characteristic signs, symptoms and test findings (laboratory, imaging, electro-diagnostics etc)
- A diagnosis identifies both the disease present (using terminology accepted in the discipline) as well as the agent/process responsible, where possible.
  - o A diagnosis specifies the above in a clear, succinct form.
- Possibly, a diagnosis is best defined by statements that are not diagnoses, but often put forward as diagnoses.

The following are ***NOT*** diagnoses

A presenting complaint	such as	weight loss
A clinical sign/finding	such as	anisocoria
A pathophysiologic process	such as	haemolytic anaemia
A patho-anatomical description	such as	hepatic centrilobular necrosis
A "syndrome"	such as	small intestinal diarrhoea

So, a diagnosis is MORE than any of these

How good are we at making diagnoses?

Pose yourself the question: "*What proportion of the diagnoses that I record on patient cards would stand up to the most intense diagnostic workup including autopsy, or histopathology?*"

Select from

100%	90%	80%	70%	60%	50%	40%

What does the literature say?
Medical literature

As can be seen from the tabulation, below, the error rate is surprisingly high and surprisingly constant over the years. This was also reported by Goldberg et al (2002).

Author	Year	Cases (n)	% Discordance	Comment
Cabot	1912	3000	40	That is understandable. 100 years ago
Fowler	1977	1000	36	No modern technology available
Carvalho	1991	910	36	We're running out of excuses
Kajiwara	1993	997	34 to 40	
Burton	1998	1105	44	
Attems	2004	1594	47.5	We have run out of excuses
18 papers	2004-8	30000	35%	11-54% Class I errors

One may also say, with justification: "*I accept that there are some diagnoses that I'm uncertain of, but there are many that I would stake my life on.*"

That may be an expensive wager, but there is certainly some truth in it as shown in the table, below.

Cases (60 studies)	Certainty assigned by Clinician	% Discordance
9 248	Fairly certain	16
3 694	Probable	33
1 282	Uncertain	50

Shojania K, Burton E, McDonald K, et al. The Autopsy as an Outcome and Performance Measure. Evidence Report/Technology Assessment No. 58 (University of California at San Francisco-Stanford Evidence-based Practice Center) : Agency for Healthcare Research and Quality. October 2002

Then you may say, based on experience,: "*Some organ systems are just much more difficult to arrive at a diagnosis than others.*"

Again, you will be right, but studies of this nature still come up with some frightening surprises.

Examining clinical record against necropsy reports

Disease	Clinical diagnosis	Confirmed at autopsy	% Discordance	Diagnoses missed clinically but found at autopsy,	Missed as % of clinically correct diag.
Pulm TB	15	7	53	7	100
Pulm emboli	79	44	44	99	282
Card infarct	256	198	23	51	26
Cirrhosis	27	22	19	13	59
Ac. GIT/Abd	13	9	31	1	11
Cameron HM, McGoogan E, Clarke J, Wilson BA. Trends in hospital necropsy rates: Scotland 1961-74. BMJ 1977. 1:1577-80					

The two studies analysed, here (above and below), reveal that there is a marked disease/organ-system variation but also, that there is a marked institutional variation. One of the interesting, but possibly puzzling findings in the second study, is that there was a further 32% discordance between the clinical record and the death certificate.

Examining death certificates against necropsy reports

Disease	Clinical diagnosis	Confirmed at autopsy	% Discordance	Diagnoses missed clinically but found at autopsy	Missed as % of clinically correct diag.
Respiratory	177	91	49	89	98
Cardio-Vasc	71	46	36	119	257
Gastro-Intest	49	26	47	33	127
Uro-Genital	34	9	74	9	100
Neurological	70	43	39	5	12
Others	47	25	47	13	52
Singnton JD, Cotterell BJ. Analysis of death certificates in 440 hospital deaths: a comparison with necropsy findings. JCP 2002. 55:499-502 (Aylesbury, UK)					

A few interesting side-issues arise from a look at these studies. One of these is that there has been a marked decline, over the years, in the proportion of cases on which necropsies are performed in medical practice.

One reason advanced was that with the ever-present possibility of litigation, it is better not to expose your clinical diagnosis to the test of necropsy.[Nichols L, Aronica P, Babe C. Are autopsies obsolete? Am. J Med. Sci. 1996; 311: 215–20. ANZ J Surg. 2006 Apr;76(4):205-7. Resuscitating the autopsy in Australian hospitals. Jeganathan VS, Walker SR, Lawrence C.]

And so we conclude, with a fair degree of confidence, and possibly a little "smugness", that our medical colleagues are not doing too well in the diagnosis stakes. From these data, it seems a fairly sound practice to ask for a second and possibly a third opinion before we accept the diagnosis proclaimed by our doctors. We veterinarians might even develop a sense of superiority.

So, the question arises, how well does veterinary diagnosis-making compare with medical diagnosis-making?

The first startling finding is that the veterinary profession has either hardly ever asked the question and has certainly hardly ever tried to answer the question. Where as the medical literature will produce a plethora of studies (The recent AHRQ {Agency for healthcare, research and quality} survey {2002} found 225 English-language and 34 foreign-language studies published), there are only **TWO** articles in the electronic veterinary database (i.e. published since 1968).

Fortunately, one article, published in 2004, was produced by one of the Ivy-League universities (from a veterinary perspective), namely, Davis campus of the University of California, and probably represents current "best practice". After all, if some "third-rate" institution had produced it, we could have claimed exemption.

Section of Vet Hospital	Cases (n)	% Discordance
Emergency Critical Care	139	45
Internal Medicine	272	44
Neurology	100	35
Surgery	33	27
Cardiology	19	21
Oncology	41	15
TOTAL	**604**	**39**
Kent MS et al. Concurrence between clinical and pathological diagnoses in a veterinary medical teaching hospital (623 cases) 1989 and 1999. JAVMA 2004, 224, 3, 403-406. U Ca Davis.		

The second study, from the Netherlands, is not much more encouraging: [Vos JH et al. *Comparison of Clinical and Pathological Diagnoses in Dogs (Prospective study – Netherlands) . Vet Quarterly 2005, 27 March (1), 2-10*]. 145 dogs (36 died, 109 euthanased) of which 119 dogs had both a Clin & Path Diag Total agreement in 61 cases (51%), Disagreement in 31 cases (26%), Partial agreement in 27 cases (23%)

Is There a Problem with the Accuracy of Clinical Diagnoses?

From the above data the obvious answer is, patently yes. The problem is not just limited to the medical fraternity but, most certainly, exists in veterinary medicine too. The problem differs somewhat between sub-disciplines but appears to apply, to a greater or lesser extent, across the board.

So, the questions that we should address next are:

- "Do we just live with it?"

Or

- "Is it possible to improve/make progress?"

At this juncture it is probably appropriate to quote George Bernard Shaw

"The reasonable man adapts himself to the world; the unreasonable one persists in trying to adapt the world to himself. Therefore, all progress depends on the unreasonable man."
(Man & Superman III, 1903).

Do these data apply to everyone?

If not, then is it possible that there are "expert" experts? And, is it possible to improve your expertise? To address these questions we should have are brief look at the field of research that is owned by biomedical science, psychology and education. Namely, the study of how diagnoses are made or alternately biomedical problem solving.

The Diagnostic Process

Components of the diagnostic process

There are probably many classifications but it would appear that there is consensus that there are three principal components.

1. **Data gathering** involves
   a. Non-clinical/physical information
      i. Taking of a history and the
      ii. Collection of information pertinent to the case that may be environmental and/or circumstantial.
   b. The accumulation of information about the patient's clinical status (signs and symptoms).
      i. Subjective data (those that are not strictly measured)
      ii. Objective data (measured or using classification systems) including clinical, laboratory and other diagnostic aids.

2. **Data interpretation** appears to be clearly divided into two activities, namely.
   a. Understanding the data. This implies epidemiological, clinical and medical knowledge and
   b. Understanding disease. This implies knowledge of pathophysiology as well as disease-causing organisms.

3. **Establishing the diagnosis** is then a process of using the gathered and interpreted data to
   a. Propose a provisional diagnostic hypothesis (and there appear to be a few ways of skinning that cat {pardon the pun})
   b. Ruling out alternative hypotheses.

Sources of error

Using this diagnostic process model, investigators have accumulated erroneous diagnoses and analysed the component/sub-component of the process where the error appeared to have its origin, with the following results.

1. Although the gathering of non-clinical (historical etc) data is considered by most diagnosticians (from 60 to 80% of the success of the diagnostic process is attributed to this phase) to be the single-most important component, it is really surprising that ***neither the time spent*** on this process (I imagine within reasonable limitations – allowing that it happens at all) ***nor the volume of data gathered*** in this process, appear to bear any significant relationship to the incidence of errors. However, ***the omission of diagnostically relevant data***, pertinent to that specific case, (and therein lies the trick) does promote diagnostic failure.
2. Lack of understanding the data (both medical as well as pathophysiological) leads to diagnostic failure but apparently, more importantly, ***ignoring diagnostically relevant data*** (i.e. having gathered it but now not using it) leads to "**premature closure**" and consequent diagnostic failure.
3. In the generation of diagnostic hypotheses, the generation of inappropriate hypotheses for the available data (logic failure) leads to diagnostic errors but ***early elimination (discarding) of reasonable diagnostic hypotheses*** (either as principal or alternate), another form of **premature closure**, leads to diagnostic failure. Another term for such an error is "ideolepsis", making up your mind what the diagnosis is and thereafter ignoring evidence to the contrary.

Premature Closure

The concept of **premature closure** implies making a diagnosis (establishing a diagnostic hypothesis) when reasonable alternate hypotheses are potentially available but have not been allowed to see the light of day, either because some critical data are missing OR because the clinician is ignoring {possibly not recognising} available critical data. Of all the diagnostic errors that the investigators detected, **premature closure**, as a concept, was the single most common, explaining fully 91% of all the incorrect diagnoses.

Does our diagnostic ability change with time?

Various investigators have pronounced on the different diagnostic strategies or emphasis employed by

1. Novice diagnosticians (undergraduate students),
2. Fully-schooled diagnosticians (interns or post-graduate students) and
3. Experienced diagnosticians (variously including qualified

physicians or specialists).

There appears to be a general consensus (although there are dissenting voices) that the experienced diagnosticians

- Generate fewer diagnostic hypotheses.
- Are more likely to include the correct diagnostic hypothesis in their list (so can fall back to a list of alternates that is likely to be successful)
- Select the most appropriate diagnostic procedures (laboratory, imaging, electrodiagnostics etc) to rule-in/out competing hypotheses.

It appears that these "indicators of expertise" are related to the degree of diagnostic success as well as the speed at which the correct diagnosis is reached.

The extent to which these "indicators of expertise" are displayed, appears to improve with the student's progression through the medical training programme.

Is there something special about medical diagnostics – or is it all simply problem solving?

Again, the literature is not unanimous (and it is voluminous).

The evidence on one point, however, is fairly clear. The expertise involved in making a diagnosis is NOT the possession of a generic problem solving skill. Training in problem solving *per-se* does not translate into competence as a medical diagnostician.

Furthermore, it appears that problem solving skills are not exportable from one discipline to another. Even within medicine, experts in one discipline may struggle to outperform novices (and certainly interns) in an unfamiliar medical discipline.

Are there different diagnostic methods (within medicine)?

Most authors recognise two basic groups of methods:

1. Backward reasoning (deductive) which is also referred to as the "Hypothetico-deductive" method. It appears that novices tend to use this method principally. The process is assumed to work as follows:

a. Look at the mass of data

b. Think of as many differential diagnoses as the data allows and then go back to the data time-and-again to try to accommodate everything.

c. This tends to drown the novice in data and competing hypotheses.

   i. This system is often considered to be the "core" of the Problem-Oriented Medical Record System, originally proposed by Lawrence Weed and adopted into veterinary teaching by Osborne.

2. Forward reasoning (inductive): is considered the "province" of the experts who integrate the data to form a few rational hypotheses and then employ a little backward reasoning only to consider inconsistencies and alternatives.

As I read the literature, these concepts are beginning to wear thin and the evidence is fairly clear that these models are an outmoded oversimplification. Apparently, the forward *vs* backward issue is still central to computer-based/artificial-intelligence approaches.

In the audience to whom this paper is addressed, the issue is probably not of how to get from novice to intermediate, where these concepts remain central to the teaching paradigm, but how to get from intermediate to expert and possibly from expert to super-expert. That being the case, we need to examine what the expert appears to do. Because if we can discover that, then we might be able to suggest ways of "thinking" or "training" to climb the ladder of expertise.

What is it that allows experts to succeed at clinical reasoning?

1. Experts are able to recognise non-diagnostic (non-pertinent) data (not always simply "normal" data) almost instantaneously and appear to be able to ignore these data almost subconsciously. When quizzed or interviewed about a case that they have solved they are often unable to recall the non-pertinent data. Novices, on the other hand will usually be able to outperform the expert on the recall of such data. This means that the expert has less data (but pertinent data) to "juggle" around.
2. Experts devote time and attention on inconsistencies (recognising them as such) and this appears to allow them to identify good rational alternative hypotheses. By contrast, novices try to shoehorn inconsistencies into their favoured hypothesis.
3. Experts are able to integrate historical, environmental and circumstantial data (some of which can be described as "enabling" data) with the clinical picture and thereby reduce the number of possible hypotheses.
4. Experts are presentation-sequence driven. They seem to be able to use the presentation course and sequence to rapidly access the more likely diagnostic hypotheses that would fit such a pattern.

a. It is of particular interest that when case data are randomised experts tend to lose much of their diagnostic advantage over non-experts.

5. Experts appear to use "illness scripts" in solving clinical diagnostic problems. There is some controversy about this issue but, currently, the consensus is that there is such a thing as an illness script (more about that, below). The difference of opinion seems to involve the issue of whether these are "experience-based" ready-made scripts in memory and the expert simply picks the appropriate one after a quick intra-cranial search or whether there are generic script models, in memory, and the expert "fleshes" these out with data from the case.

a. These scripts allow the expert to reach a diagnosis (or select the most appropriate diagnostic hypothesis) extremely rapidly without having to go through a large amount of mental gymnastics.

The illness script concept

History

While the forward and backward reasoning and the Hypothetico-deductive models were being used to create insight (and some confusion) into diagnostic thinking, and guide the developments in biomedical teaching as well as artificial intelligence, there were some "*unreasonable men*", to use Shaw's phrase, developing an alternate approach to both teaching (PBL) as well as understanding medical expertise (Scripts).

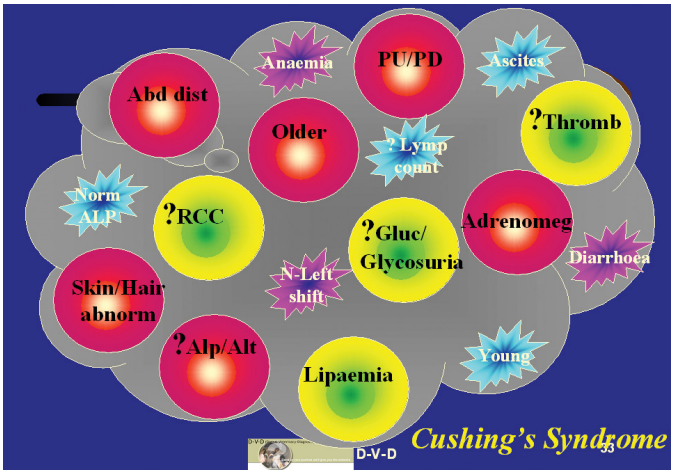
Howard Barrows, at the Southern Illinois University School of Medicine in Springfield, Illinois led a group of "alternate thinkers" including Paul Feltovich (Urbana, Illinois), Henk Boshuizen at the University of Maastricht in the Netherlands, Geoff Norman at McMaster University, Ontario, Canada (later joined by Henk Schmidt, now at Maastricht) and the late David Maddison at the University of Newcastle, Australia.

In a series of studies and publications Barrows, Feltovich, Schmidt and Boshuizen, particularly, developed the concept of "illness scripts" and how these appear to facilitate medical problem solving by experts.

Schematic representation

The illness script can be represented as a short tabulation of typical/commonly encountered findings in a particular disease, arranged in a logical (possibly temporal) order with default values/scores for the most important criteria.

An imaginary picture of an imaginary "Illness Script"



Pink/Red = Essential
Yellow = "Nice to Have"
Lavender = Inconsistent but "no big deal"
Blue = Inconsistencies that make you have to possibly reconsider.

The script reads, generally, from left to right.

Script formation

The script appears to exist as a knowledge network in the expert's memory (long term). It is thought to be created when a clinician experiences a patient with a particular disease (possibly initially based on things that struck the clinician). It is possible that as more cases of this disease are seen, the script becomes more and more sophisticated (esp. in terms of the default values and the attribute chronology).

Script implementation

- The clinician experiences the patient in terms of the presenting data. This usually includes the presenting complaint (which may even have been transmitted telephonically when the patient was scheduled) as one of the first issues as well as the history.
- By the time the clinician has got half-way (if that far) through the examination, some of the script attributes will have "triggered" a sub-conscious script search and certain candidate scripts seem to be brought from deep memory into a sort of "Random Access" (to use a computer term) memory.
- It is possible that several scripts, with similar early (from a case evaluation point of view) script attributes, are mobilised simultaneously and then those that fail to give further expected attributes (or attributes inconsistent with the script) and acceptable default values will be dropped.
- The more "hits" (script attributes present), the more certain the diagnosis.
- Script items that do not conform to default values alert the clinician to the possibility of the script's inapplicability and if there are a number of non-default values, the script is rejected.
- There appears to be a "when these are present, that is sufficient" logic that operates and this may explain why scripts can allow very rapid diagnostic conclusions – with a possible control process that checks if any of the defaults are badly violated.
- How much of this is sub-conscious and how much conscious problem solving does not appear to have been clearly identified. It can be seen from the above that this is very similar to the hypothetico-deductive problem solving strategy that the script concept seeks to depose. The more sub-conscious, the less hypothetico-deductive and the more "forward reasoning".

Acquiring expert clinical problem solving skills

The Problem Oriented Medical Record (POMR) system, that has been the hallmark of biomedical education for many decades now, was developed and introduced into medical teaching by Lawrence Weed, who simply extended a neat hospital record keeping system into a teaching tool.

In veterinary education, Osborne was the advocate of its use in teaching. It appears that the POMR system is not suited to the creation of expertise.

Whether it is essential to the creation of a post-novice status is a point that will be hotly debated as most biomedical curricula are designed around the system and changing that would be very disruptive. If the illness script is the tool that experts use, then it makes sense to find ways and means of acquiring good script networks.

Features of scripts that have implication for their acquisition

- Currently, script development is something that "just happens" as clinicians are exposed to patients.
- It is likely that scripts, as such, cannot be transmitted from one expert's mind into another's by some form of rote learning.
    - They have to be constructed (possibly unique in their detail for each individual) by the individual.

- However, there is every reason to believe that the process of script building (and possibly script implementation) can be facilitated by creating a "script-positive" learning environment.
- Scripts represent elaborated and organised knowledge
- It should be possible to construct efficient and well-structured knowledge bases.
- Scripts appear to be constructed onto/from pre-existing knowledge networks in memory
- Scripts may develop through a process of becoming aware of explicit disease features when exposed to a case with such a disease (i.e. they need some explication of the case)
- This explication, and thus the possibility of building good scripts, may be enhanced by discussing cases with one's peers or instructors.
    - This would suggest that the "apprentice system" would promote script formation.
- Studies have shown that when experts discuss cases with their peers they very often render the case in the form of a script – but often do not mention all the script attributes, only the outstanding (interesting) features, apparently "knowing" that the listeners already have most of the necessary script information in their own heads. Consequently, this would have to be a conscious, deliberate process.

Proposal for improving, accelerating and/or acquiring good scripts

- With each case that the clinician experiences, a script tabulation/image (similar to the one described, above) should be constructed and filed with the records.
- From time to time (and preferably on a regular basis) clinicians should discuss their more interesting cases with peers or instructors with an emphasis on
- Rendering the case in script format
- Explicating the reasons for the script attributes
- Pontificating on the default values

Conclusion

It is hoped that by highlighting the very fallible nature of clinical diagnoses and outlining some of the studies on expert clinical reasoning, the readers have been encouraged to seek ways and means of improving their own clinical problem solving skills.

The illness script concept appears to present an answer to those who seek to improve their skills.

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Metabolic Malfunction: Gangliosidosis in Cats

Gangliosidosis is a genetically inherited metabolic disease found in several breeds of cat.

There are two forms of gangliosidosis (GM1 and GM2), which both result in a defect of the lysosomal function. Lysosomes are found in the cell membrane of almost every cell in the body and are fluid filled sacs that contain enzymes which help with waste disposal from the cells. The enzymes help to break down large molecules in the cell, such as lipids, carbohydrates and proteins, which are then passed on to other parts of the cell for recycling. Several causative mutations have been identified for gangliosidosis in cats:

Table 1: *Known causative mutations for gangliosidosis in cats. Table adapted from the OMIA database.*

Type	Region	Affected gene	Mutation	Mode of inheritance	Breeds affected
GM1	Chr2	Galactosidase, beta 1 (GLB1)	c.1448 G>C (R483P)	Autosomal recessive	• Korat • Siamese
GM2, type II (Sandhoff or variant 0)	ChrA1	Hexosaminidase B (HEXB)	• c.39del • c.1467_1491inv • c.667 C>T • c.1244-8_1250del	Autosomal recessive	• Korat • Domestic • Burmese
GM2A (Tay-Sachs disease)	ChrA1	GM2 ganglioside activator (GM2A)	c.516_519del	Autosomal recessive	
GM2AB (AB variant)	-	Hexosaminidase A (HEXA)	Key variant not known	Unknown	Not specified

The amino acid change in GM1 gangliosidosis leads to the deficiency (approximately only 18% of normal activity) of the beta-galactosidase enzyme, which causes gangliosides (type of carbohydrate) to accumulate in the cells instead of being broken down. Instead, the cells store these carbohydrates in membrane bound vacuoles, which over time, reduces the available space within the cell in which to perform its cell functions.

This eventually leads to cell death, which damages the central and peripheral nervous system and other organs. The second type of gangliosidosis can further be broken down into 3 subtypes, namely Tay-Sachs (GM2A), Sandoff or 0 variant (GM2B) and AB variant (GM2AB). The mutation leading to AB variant disrupts the function of the GM2 activator, which is involved in the normal functioning of beta hexosaminidase A. Both GM2A and GM2B are characterised by a deficiency in the beta hexosaminidases A and B. All mutations lead to the inability to break down ganglioside lipids.

Affected cats show no signs of the disease at birth, but over time, gangliosides will accumulate in the neuronal cells and cause damage to the nervous system.

Clinical signs start to present at 1-5 months of age and may include the following:

- Facial dysmorphism (head is flat and broad with a small skull)
- Tremors
- Dysmetria (high-stepping gait)
- Ataxia (uncoordinated gait)
- Reduced postural reflexes
- Stiffened gait
- Weakness in front and hind limbs
- Depression
- Nystagmus (involuntary side to side movement of eyes)
- Enlarged liver
- Clouding of the cornea leading to visual impairment
- Neutrophils and lymphocytes (white blood cells) may contain cytoplasmic granules
- Seizures
- Affected kittens may be stunted

The progressive accumulation of the gangliosides within the cells and the resulting damage to the central nervous system will ultimately prove fatal for affected cats by a young age, typically between 8 – 10 months of age.

Diagnosis can be confirmed with bloodwork and genetic testing. Storage products and enzymes can be tested for through biochemical identification as affected animals will have low beta-galactosidase enzyme activity in their liver and increased levels of Gm-1 ganglioside in the brain. Genetic testing can be performed for known causative variants to determine whether the kitten has mutated copies of the genes. As the disease is autosomal recessive, kittens will require two copies of the mutated gene to be affected. Cats with a single copy of the mutated gene will be silent carriers and will run the risk of passing on the mutation to their offspring. As most affected cats will die before reaching sexual maturity, the greatest risk of passing on the disease, comes from breeding silent carriers to each other. As there is currently no known cure for gangliosidosis, the only way to prevent the spread of the disease is to breed with confirmed clear animals.

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Ten Years of Dextrose Prolotherapy

Steve Wimberley ((BVSc) Hons
Westille Veterinary Clinic

I was introduced to the concept of prolotherapy ten years ago by a very good friend and colleague. She knew I had a surgical bias but felt that I might find prolotherapy a useful option in cases where surgery is not really an option such as those with severe financial constraints, or older animals with co-morbidities where surgery is very risky or where post-operative confinement would be very challenging.

The underlying principle of prolotherapy is well accepted in human and veterinary sports and rehabilitation medicine. To use the words of Dr Lowri Davies[1] specialist veterinarian in canine sports medicine and rehabilitation – "We heal by inflammation"

Simply put when it comes to injured tendons, ligaments, and joints it is the inflammation that drives fibroblast activity, collagen deposition, remodeling and healing.[2] Consequently if we take an injured ligament, tendon or joint which is already inflamed and trying to heal and infiltrate it with an irritant solution like 25% dextrose we create even more inflammation and thereby accelerate healing significantly.

Modern prolotherapy was pioneered by a medical doctor and surgeon from Ohio Dr George Hackett in the 1930s.

Dr Hackett coined the term prolotherapy because his original work demonstrated that the tissue laid down during the healing process was new healthy tissue not scar tissue. This distinguishes prolotherapy from a related technique called sclerotherapy.

I was inspired to try prolotherapy after reading articles on the success they were experiencing with it by veterinarians like Dr Carvel Tieckert[3] from the USA and Dr Sagiv Ben-Yakir[4] from Israel. What also influenced me was a mindset change I experienced. As a young graduate my mindset was very much that of a surgeon i.e. "A chance to cut is a chance to cure". After twenty years of doing surgery and enjoying lots of success but also living though some horrendous failures, where I wished I had never operated, my mindset started to change to one of "First do no harm".

The first few cases I treated were small dogs with cruciate injuries. They all responded very well but I remained skeptical as we

know that small dogs in particular can heal their cruciate injuries conservatively. However when I started to experience similar success with larger dog cruciate injuries I realised that there was more than just luck and self-healing happening. I started to see cruciate injuries that may have healed conservatively in 9 to 12 months improved and heal in 3 to 6 months reaffirming to me that prolotherapy really does accelerate healing.

As I grew in confidence with the technique I started to apply it to other conditions and experienced similar success. Currently I have enjoyed success in treating the following conditions:

- Cruciate injuries in cats and all size dogs
- Rescuing extra-capsular cruciate surgery where the patient has overdone things post operatively and stretched or loosened the implant
- Collateral ligament injuries of carpal and hock joints
- Carpal hyperextension injuries in cats and dogs
- Tarsal-metatarsal subluxations
- Partial gastrocnemius tendon injuries
- Grade 2 medial patella luxations
- Medial scapulo-humeral ligament injuries
- Hip sub luxations
- Young dog with very lax and painful hips

In all cases I advise regular physiotherapy as well. I have found the combination of prolotherapy and physiotherapy very beneficial.

Having said that many owners have not been able to follow through with physiotherapy yet their animals have still responded well to prolotherapy.

For those wanting more information on the mixture I use and my techniques for the above conditions you are welcome to join the Prolotherapy Interest Group I started on Facebook. It is a closed group for veterinarians and veterinary rehabilitationists only.

On there you will find the webinar I gave on prolotherapy to the JHB branch of the SAVA in 2020, video clips of me treating various conditions and lots of case reports. You will also be able to read the articles by Drs Tieckert and Ben-Yakir mentioned above and watch some webinars by an Australian veterinarian called Dr Ian Bidstrup who has been doing prolotherapy on horses and small animals for over 30 years. It has been very heartwarming to receive regular messages from veterinarians who have started doing prolotherapy and are experiencing similar successes as I have. They are very grateful to have another option to offer their clients.

In summary after treating over 2000 injured joints and ligaments with prolotherapy over the past ten years my experience is that it is a very safe, economical and easy to perform treatment that can restore many injured animals to function.

It should be of particular use to veterinarians in welfare organisations, poorer socio-economic areas, very remote areas as well as our community service colleagues and colleagues with no particular interest in orthopaedic surgery but who would still like to help their patients.

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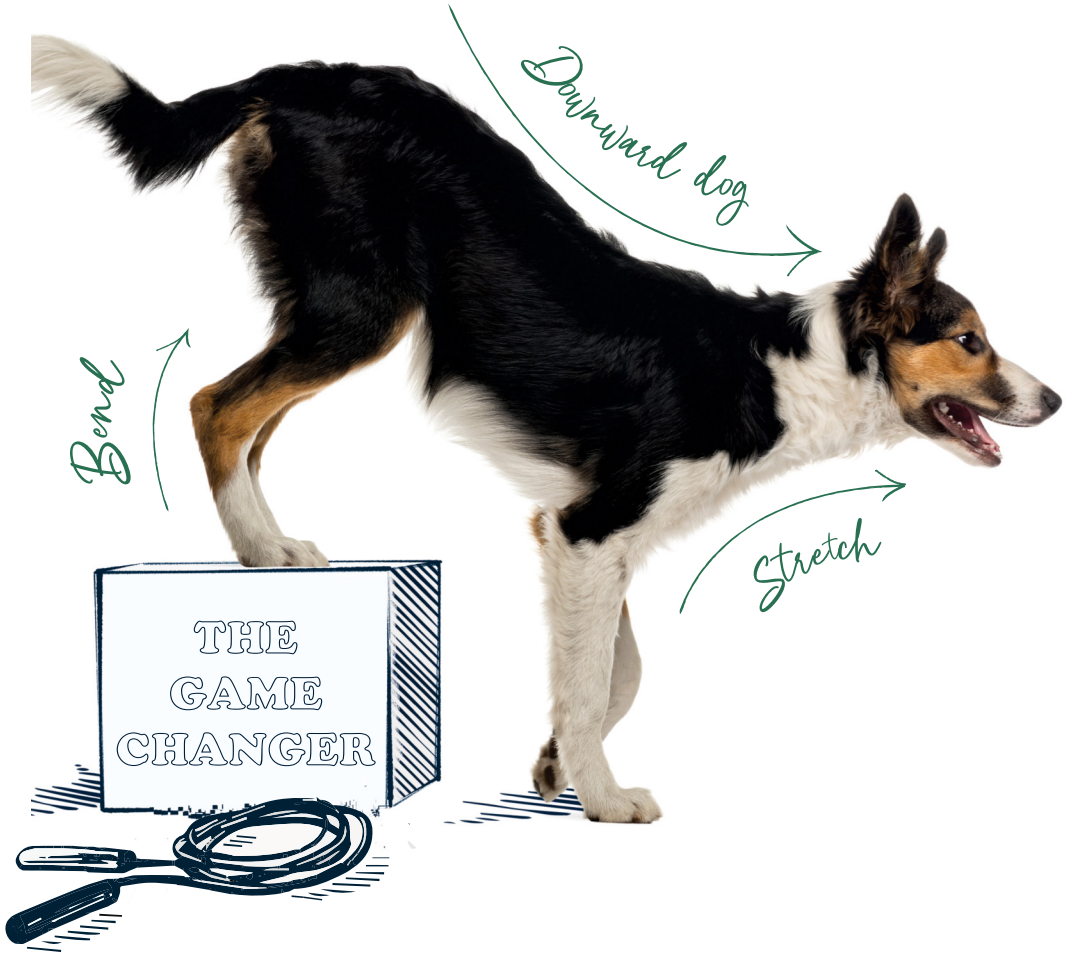

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