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A chance to cure- surgical oncology in the veterinary practice

General principles of surgical oncology

There are steps that must be followed in every surgical cancer patients to avoid missing critical factors.

Tumour identification:

This is the most important step. Accurate diagnosis allows the rest of the treatment cascade to take place. Most mistakes in surgical oncology are made because of a lack of tumour diagnosis prior to treatment. This often results in inadequate surgical dosing resulting in residual tumour after surgery and in missing obvious metastatic lesions, not pursued because the clinician failed to suspect their presence. There are progressively more invasive techniques used to obtain a diagnosis. These are usually followed in a sequential manner until a diagnosis is made.

Tumour diagnosis should be made prior to treatment when either the type or extent of treatment may be affected by diagnosis or if the prognosis would affect the owner's willingness to treat.

Example: Subcutaneous mass on lateral thorax of a dog. Distinction between lipomas and hemangiopericytomas is important because the extent of surgery required to achieve a cure is vastly different.

Example: 2 cm mass on the rostral mandible of a dog. Distinction between a melanoma and an acanthomatous epulis is important because melanoma often requires radical mandibulectomy while epulides can often be cured using radiation therapy.

Example: 6 cm cranial mediastinal mass: Distinction between lymphoma and thymoma is important because lymphoma is not a surgical disease and is more appropriately treated with chemotherapy, while thymoma is a surgical disease.

Example: 3 cm mass on mandible of a dog. Distinction between fibrosarcoma and melanoma is important because the prognosis with melanoma is poor for long term survival, even with a regional cure, while the prognosis with fibrosarcoma is quite good.

Example: 2 cm mast cell tumour on digit of forelimb of a dog (diagnosed on aspirate). Determination of grade is important because with a high grade tumour, metastasis is likely and long term survival is poor.

Biopsy prior to surgical excision should not be performed if the biopsy procedure has greater risk than the definitive surgery, especially if the surgical excision is the same regardless of histologic diagnosis. Examples include solitary lung and splenic masses.

The first step is usually a fine needle aspirate, where a small (usually 22 to 25 gauge) needle is passed through the tumour multiple times to obtain a sample of cells which are subsequently transferred to a slide (needle technique). This technique is preferable to attaching the needle to a syringe and forcibly aspirating on the needle (syringe technique) because when using the syringe technique, the cells can be distorted and often more blood will be aspirated than tumour cells. Advantages of needle aspirate are that it is safe, simple, cheap, requires no special equipment, some may be read in-house, and has almost no contraindications. Disadvantages are that needle aspiration is relatively selective, but not

particularly sensitive (few false positives, but many diagnoses are missed). It never allows determination of tumour grade or invasiveness. Needle aspiration is most applicable to mast cell tumours, carcinomas, some sarcomas, lipomas.

The next most aggressive technique is a needle core biopsy (Trucut). A special instrument is passed through tumour, and a 1-2 mm cylinder of tissue is submitted. Advantages are that it is safe, simple, cheap, requires little special equipment, allows determination of histologic type and grade in many cases. Disadvantages are that a relatively small amount of tissue is submitted, so tumour heterogeneity may result in incomplete or inaccurate diagnosis, it may be difficult to assess invasiveness into surrounding tissues and biopsy tracts must be positioned so they can be excised during definitive diagnosis.

Following needle core biopsy, incisional biopsy is most aggressive, wherein a wedge or plug of tissue is submitted. It is best to submit a sample from the centre of the tumour and from the periphery so that the degree of invasiveness into normal tissue can be assessed. Advantages include that a large of amount of tissue is submitted, the junction between normal and abnormal tissue can be submitted to allow more accurate determination of invasiveness, no specialized equipment is required. The disadvantages are that general anesthesia is usually required, poorly placed biopsy incisions can really negatively affect future attempts at surgical excision and it is usually associated with higher cost to the client (is this a disadvantage to the vet?)

When all other attempts to diagnose the tumour before excision, reluctantly, an excisional biopsy can be performed wherein the entire tumour is excised before a diagnosis is made. The whole sample is the submitted for biopsy. The theoretical advantage is that one can possibly diagnose and treat tumour at same time and there is only one anaesthetic episode. The disadvantage is that it is very difficult to choose the appropriate surgical dose if the surgeon does not know what he or she is treating. If further surgery is required because incomplete margins were achieved, disruption of normal anatomy can make that surgery difficult or impossible. Excisional biopsies are overused. These are the most common "clean-up" procedures I do. This should only be performed in anatomic regions where either the widest surgical margins (2-3 cm) are achieved in the first place, or where a second, more aggressive surgery can easily be performed if margins are found to be inadequate. This should never be done on the extremities or head.

General principles of biopsies include: no surgical drains are placed because all drain tracts are contaminated; good hemostasis and closure of dead space takes the place of surgical drains. Excessive use of electrocautery should be avoided. Specimens should be placed in 10% formalin. One should use a pathologist that will respond to phone calls for questions. When interpreting the results, the following issues should be considered: is the diagnosis tumour or some other process, what is the degree of malignancy, what is the histologic type, what is the histologic grade, if clinically relevant. Histologic grade (or subtype) has been shown to be prognostic in soft tissue sarcomas, mast cell tumours, chondrosarcomas, haemangiosarcomas, and melanomas. Completeness of surgical margins should be assessed as well if there was curative intent.

Literature Review: The next most important step is the gathering of information about the tumour (literature review). There is a vast quantity of information which is increasing almost daily about the diagnosis and treatment of cancer and it is nearly impossible to keep up. For this reason, primary care practitioners should make use of resources that summarise then information concisely. These include textbooks, specialist and Veterinary Information

Network (www.vin.com). If you are using printed resources, make sure they are recent. Older literature often paints a fairly pessimistic picture regarding the treatment of cancer in animals. Many recent advances have allowed cures in many patients and substantial improvement in quantity and quality of life with other patients. Only when relevant information has been gathered can an accurate picture be painted for the owner with informed recommendation about staging, treatment and prognosis.

When gathering information, the following questions should be answered in every patient:

1. Potential local and systemic extent of tumour.
2. What is the likely organ or structure of origin?
3. How far are tumour cells likely to exist from the primary tumour?
4. Where are metastatic lesions likely to occur?
5. Common paraneoplastic syndromes which may affect outcome or complicate treatment?
   - Hypercalcemia with anal sac adenocarcinoma
   - Thrombocytopenia with splenic hemangiosarcoma.
6. Likelihood that surgery will be curative for primary tumour.
   - Many solid tumours can have a regional cure of the primary tumour with surgery.
   - Some tumours (lymphoma) are more appropriately treated with chemotherapy.
   - Some tumour (epulides) are very effectively treated with radiation therapy.
7. Dose of surgery required to achieve a primary tumour cure.
   - This is based on the distance that tumour cells are likely to exist from the primary tumour.
   - There is great variation for different tumour types.
   - With mast cell tumours, one tumour diameter as a surgical margins will achieve 95% clean margins.
8. Gender, breed and species variations in tumour behavior.
9. Benefit of adjuvant therapy.
10. Has survival advantage been demonstrated with chemotherapy?
11. Is radiation therapy likely to be effective if residual tumour is left behind after surgery?
12. Is radiation therapy likely to be more effective if it performed before surgery?
13. Median survival times for each treatment path. This allows clinicians and owners to balance the morbidity associated with a treatment path with the expected outcome or range of outcomes.

Determination of regional and systemic extent of the tumour (Staging)

Staging of the primary tumour refers to the visible, palpable or microscopic extent of the tumour mass. Other imaging may be required (Computed tomography, MRI, regional radiographs, nuclear scans). When assessing the tumour, the extent of the tumour should be assessed including proximity to important structures which are at risk if curative surgery is anticipated (don't cut anything you know that name of). The degree of functional impairment likely to be encountered with curative surgery should be considered. This is an opportunity to consider reconstructive techniques which may be required to close the defect.

Staging for Systemic metastasis refers to surveillance for spread of the tumour to other regions. Literature review should guide search, but remember to look for exceptions to the rules. Microscopic metastasis may not be detectable and is a common cause of treatment failures. These metastatic lesions are not obvious at the time of diagnosis. Just because they are not obvious doesn't mean they aren't there. Gross evidence of metastasis almost always imparts a grave prognosis. General health should be assessed, considering common paraneoplastic syndromes.

Surgery for curative intent.

The objective is for tumour and patient to "part ways" in the operating room. The surgeon must be resolute in accomplishing this objective. First, one must decide on the surgical dose most likely to cure the regional disease, without causing unnecessary morbidity.

Cytoreductive surgery (debulking): usually contraindicated. Leaves macroscopic (visible) tumour behind. Improving success of adjuvant therapy is more theoretic than realistic.

Marginal surgery (shelling out): usually contraindicated, unless benign lesion. Leaves microscopic tumour behind. If malignant tumours are treated in this fashion, MUST follow with radiation therapy, or malignant cancer will recur.

Wide local excision: distance required from tumour depends on histologic diagnosis. These distances must be maintained in all directions. Deep margin can be muscle or fascial plane.

1. 1 cm and one fascial plane:
2. 2 cm and one fascial plane:
3. 3 cm and one to two fascial planes:
4. 4-5 cm and two fascial planes:
5. Compartmental or radical excision: entire compartment is removed (radical mastectomy, amputation).

Palliative surgery: Palliative surgery is over done and the benefits are overestimated. Theoretical benefits include pain relief in the face of terminal disease and increased benefit of other therapies like radiotherapy and chemotherapy. Examples would include amputation for osteosarcomas with gross evidence of metastasis, resection of inflammatory mammary gland adenocarcinoma and resection of anal sac adenocarcinoma and associated lymph nodes.

Human surgical oncology groups often include two teams: the resection team and the reconstruction team. The resection team excises everything they feel they need to cure the patient. The resection team walks out and reconstruction team walks in and tries to make sense of the mess that was created by the resection team. These two teams hate each other. They do not "high five" each other on the way past. In veterinary medicine, obviously economics dictate that two team are not practical. So, when we are performing cancer surgery, the surgeon must conceptually divide his or her brain into resection and reconstruction teams. One cannot temper extent of resection for fear of closure (or inability to close). Once surgery has started is NOT the time to chicken out. It is better to leave a hole than to leave a tumour. Draw your margin, and start cutting. Take no prisoners.

Mark and evaluate surgical margins: Black ink from office supply store is used to mark surgical margins. The object is to paint the cut surface of the tumour to indicate to the pathologist where surgical margins were. If the pathologist sees tumour cells touching ink, tumour was left behind. If margins are dirty ANYWHERE, entire incision is considered contaminated, and must be excised or irradiated.

Radiation therapy or second surgery for dirty margins: When treating patient with incomplete surgical margins, the entire incision is considered contaminated. When performing a second surgery, the proposed surgical excision is marked using 2-3 cm in all directions as a guideline. The surgeons should cut deep and wide taking appropriate surgical planes. Obviously, this can get out of hand easily. This justifies making the first surgery count.

Radiation therapy: Radiation therapy does not prevent secondary metastasis. It only treats the local disease and prevents local recurrence. It is effective for treating microscopic residual cells of many tumour types. (Soft tissue sarcoma, melanoma, mast cell tumour, ceruminous gland adenocarcinoma, many other sarcomas and carcinomas- to discussed in more detail later. It is associated with side effects which in many cases are well tolerated, and can be mitigated. These are discussed in greater detail as well.

Adjuvant chemotherapy: In general, chemotherapy has no effect on residual tumour. It only prevents systemic metastasis. Relatively few tumours have shown definitive response to chemotherapy. These include: osteosarcoma, lymphoma, multiple myeloma, haemangiosarcoma, possibly mast cell tumours, transmissible venereal tumour, and possibly melanoma. Chemotherapy is well tolerated in most patients, and should be considered even in patients with malignancies not proven to be responsive to chemotherapy.

Incomplete excision:

Situations where the surgeon recognises that complete excision cannot be achieved include before the surgery, during the surgery, when biopsy results return and when the tumour recurs. In general, an active, preventative role with planning is recommended, in preference to a "whoops, what do I do now" approach.

The intent of cancer surgery is cure. If the surgeon does not anticipate that a cure will be achieved with an intended surgery (that means 2-3 cm IN ALL DIRECTIONS and one fascial plane deep to the tumour), this is the best opportunity to change gears and offer other options that have a better chance of being curative. The most important step is to discuss options with owners. They need to be informed that the intended surgery is unlikely to be curative and they need to be given alternative plans with better success rates. The obvious first choice is to offer a more aggressive surgery (including the possibility of amputation) which is more likely to be curative. If the primary care practitioner is unable (or unwilling) to perform the surgery needed for a cure, then he or she is obligated to offer referral to a specialist who may be able to perform a more aggressive surgery. A marginal surgery followed by radiotherapy is also an excellent choice for many tumour types with cure rates of 85% commonly reported.

DO NOT TRY TO SHELL-OUT MALIGNANT TUMOURS. During cancer surgery, the objective is for the surgeon to never "see" tumour tissue. In 99% of my cancer surgeries, I never actually make contact with the tumour. I only make contact with normal tissue 3 cm away from the tumour. This kind of rules out the "shelling-out" cancer surgery. If I see tumour at any time during the surgery (except what can be seen from the outside), I have failed. Studies have shown that human cancer surgeons are very accurate in predicting if margins are going to be clean or dirty. If you encounter tumour tissue during the surgery, STOP RIGHT THERE. A new game plan must be selected. The first step is to contact the owners and discuss options. If a marginal excision is performed, the tumour will recur. If the tumour is "shelled

out" it will recur. If you cut into tumour during the surgery it will recur. If you take the tumour out in "bits" it will recur. Make sure the owners recognise this and are happy for you to proceed. There are only two acceptable options that will prevent recurrence. The first is to close the incision, reprep, redrape, change instruments and start over, taking wider and deeper margins (2-3 cm wider and deeper). Amputation is sometimes an option. The second is to complete the surgery, taking out all visible tumour and planning on radiation therapy. All other options will almost certainly result in recurrence.

When biopsy results return, try to get an impression from the pathologist about not only whether the margins are clean or dirty, but how far tumour cells exist from the surgical margin. Determination of surgical margins by a pathologist is not an exact science. In human medicine, the entire specimen is sliced in small increments to determine whether tumour cell breached the surgical specimen. In veterinary medicine, economics dictate that only a few areas are evaluated. This means that if margins are "close but clean" a few tentacles could have slipped by in between areas evaluated by the pathologist. If I get a reading that says that margins are clean by 2 cm, I am much more comfortable than if they are clean by 2 mm. Marking the margins is very helpful for the pathologist to determine what is and is not a surgical margin.

If margins are very close or overtly dirty, again, one must have a discussion with owners about options. The tumour will likely recur unless decisive action is taken. Options include wider local excision (2-3 cm margins and one fascial plane deep around the entire surgical scar), amputation (if possible, less appropriate for tumours on the head, unless the patient is a Labrador), or radiation therapy of the wound bed. Radiation therapy is very effective for "cleaning up" dirty cuts with cure rates of 85% with many tumour types.

The last situation when the surgeons recognises that incomplete excision was achieved is when the tumour recurs. It is important that the patient be restaged- chest radiographs and/or lymph node aspirate because we don't want to jump straight into surgery if gross systemic disease is present. CT or MRI can be helpful for staging local and systemic disease. A second surgery can be performed and 2-3 cm margins should be taken on the entire recurrence and PREVIOUS SURGICAL SCAR. A second surgery may require amputation. Radiation of the recurrence can be performed but is much more likely to be successful if a second surgery is performed first to remove all visible tumour. Again, success rates of 85% can be expected if the tumour can be reduced to microscopic disease.

Radiation therapy

General principles: Radiation uses x-rays or gamma rays to damage cellular DNA. Cells death occurs during replication. Therefore, rapidly dividing cells are preferentially killed. Normal tissues with a high mitotic rate are also damaged if they are within the field. Cellular damage is a random event. The more radiation, the more likely lethal damage will occur. Normal tissues have better reparative mechanisms and are therefore more resistant to radiation damage. Radiation protocols have been honed to maximise tumour death with acceptable tissue toxicity.

There are characteristics of radiation sources that describe the way the beam interacts with matter. The energy of a particular beam describes the average velocity of the photons which make up that beam. Depth-dose curves for different energies relate to the percent of the dose which is administered at different depths. For example, low energy beams drop off

very quickly when they strike tissue and deliver most of the energy within the first few millimeters. High energy beams can penetrate several centimeters. DMax is the depth where the maximum dose is delivered. A 300 Kvp orthovoltage beam has a DMax at the skin surface and loses 50% of the dose at about 5-6 cm. This means that to deliver a lethal dose to a tumour 5-6 cm deep, twice that dose of radiation is delivered to the skin. This limits the application of orthovoltage to more superficial tumours. Generally, orthovoltage is appropriate to tumours which are less than 3 cm from the surface of the skin.

Another issue with orthovoltage is that it is selectively absorbed by bone. Therefore, bone which is within the field is going to get a greater dose than the surrounding soft tissue. Bone gets more side effects, and the deeper tissues are shielded by bone and get a lower dose. Orthovoltage is most appropriate for superficial tumours and dirty margins with little bone within the field.

Cobalt has an energy of 1.25 megavolts (Mv) and a DMax at 0.5-1.0 cm below the surface. This means that the skin can be "spared" some of the side-effects of radiation. Some linear accelerators have a 10-20 Mv beam with a Dmax of 4-5 cm and only 50% of the total dose delivered at the skin and a penetration of several centimeters.

Side effects of radiation therapy.

Side effects of radiation therapy relate to the undesired effects to normal tissue when curative doses of radiation are used. They can be decreased through appropriate patient and protocol selection. Radiation only affects tissue directly within the radiation beam. Side effects are catagorised as acute and late. Acute effects occur during or shortly after radiation therapy. They affect rapidly proliferating tissues like oral mucosa, intestinal epithelium, eye and skin. They are generally self-limiting and recover rapidly. They can be annoying but should not limit the dose of radiation. Owners need to be warned that acute side effects will occur if curative doses of radiation are administered.

Mucous membranes within the field will become inflamed. They can develop mucositis. Patients may become reluctant to eat and drink and may become dehydrated or malnourished. Low-salt foods are more palatable and less irritating. Hand feeding by owners may be helpful. Administration of fluids may be required. Feeding tube placement may be necessary. Management is directed at nutrition, hydration, pain relief and prevention of self-mutilation. Mucositis starts at the beginning of radiation therapy and peaks within 2-3 weeks. It is more severe when there is preexisting dental disease. Dental prophylaxis is recommended prior to radiation therapy if noticeable dental disease exists.

Skin is commonly affected by acute effects. These effects usually start within 2-3 weeks and last 2-3 weeks beyond the end of radiation. The severity is dose-related. Epilation and alopecia occurs commonly and may be permanent. Depigmentation of skin and hair may occur. Dark-haired fur may grow back white and visa-versa. Moist desquamation is when the skin becomes weepy and inflamed. It can be itchy and painful. Prevention of self-mutilation and good general hygiene are of paramount importance. If radiation is being performed in conjunction with surgery, wound healing is an issue. Radiation should be delayed for at least 2 weeks after surgery. Surgery on irradiated tissue should be delayed for at least 2 months following completion of radiation therapy.

If eyes are within the field side effects will occur. Tungsten contact lenses can reduce ocular side effects. Ocular side effects include KCS, conjunctivitis, cataracts and retinal damage.

Late effects tend to affect slowly proliferating tissues like bone, lung, heart, kidneys and spinal cord. Late effects tend to occur 12-18 months after radiation therapy. These are typically very serious and result in necrosis of affected tissue. The onset of late effects should be considered a treatment failure. The only solution is resection of the tissue. With orthovoltage, bone and muscle are the most likely sites of late effects. Other tissues are too deep to be affected by orthovolage. Late effects can be reduced by decreasing dose per fraction and increasing frequency of treatment.

Indications for radiotherapy

Radiotherapy can be described as having curative intent or palliative intent. Radiotherapy is particularly effective for cleaning-up of dirty margins following mast cell tumour and soft tissue sarcoma removal (5 year local control of 90%).This is the real strength of radiation therapy and particularly orthovoltage, because in general, incisions are superficial and are well-within reach of the relatively low-energy orthovoltage beam. Side effects can be mitigated through the use of intraoperative radiation therapy wherein vital organs are retracted out of the way while a single large dose of radiation is delivered at the time of tumour resection. After that, and abbreviated course of external beam radiation is administered with reduction in skin effects.

Radiation of oral epulides are associated with up to 90% local tumour control when used as a sole method of treatment and is curative if it is used with marginal surgery. Radiation of canine oral squamous cell carcinomas carries up to 75% local control at one year. Treatment of nasal tumours is associated with up to 23 month median survival time when combined with surgery. This is in contrast with 12 months with megavoltage radiotherapy alone, 7 months with chemotherapy and 2 months with no treatment. Localised mycosis fungoides can be effectively treated. Ceruminous gland adenocarcinoma has a 60% local cure rate after incomplete surgical excision with a median survival time of 3.5years. 72% of patients with incompletely excised thyroid carcinoma treated with radiation therapy will be alive after 3 years. Incompletely excised ceruminous gland adenocarcinoma carry a median survival time of 3.5 years when irradiated postoperatively. Feline cutaneous squamous cell carcinoma is challenging with any treatment modality. It carries a 50-60% one year survival rate with radiation n therapy. Brain tumours, using orthovoltage have a median survival time of 345 days and mean of 489 days, compared with median of 30 and mean of 81 days without treatment. We use a combination of intraoperative radiotherapy of brain tumours with the added benefit of the fact that we make a bone window through which external beam radiotherapy can be administered.

Palliative intent radiation therapy results in an improvement in quality of life and a modest increase in survival time. Only a few large doses of radiation are administered with very few side effects. Examples include treatment of osteosarcoma (median survival time of 4 months) and oral melanoma (median survival time of 8 months with only 3 treatments). The injection of contrast material into the tumour before palliative radiotherapy may increase its effectiveness.

In summary, incomplete surgical excision almost always results in local recurrence of malignant tumours. Radiation therapy is often very effective at preventing recurrence following incomplete surgical excision.

Patella Luxation in Dogs

Patellar luxation is a common orthopaedic condition that is seen in both dogs and cats. Large and small breed dogs can be affected with medial luxation being much more common than lateral. Like many conditions there is a spectrum of severity and as such a grading system has been developed to characterise the severity. Dogs are typically graded from 1-4 based on the tendency of the patella to luxate out of the trochlear groove. Ultimately the problem is that there is malalignment between the quadriceps muscles, the patella, the patellar tendon, the tibial crest and the underlying trochlear groove. There are a number of theories pertaining to the eitiology of this condition however there is no one consensus about the cause. In reality there are likely multiple contributing factors. Aside from the medial location of the patella, a number of other musculoskeletal abnormalities may be present. These include distal femoral varus, internal rotation of the tibia and a shallow trochlear groove. These changes are a result of altered biomechanics caused by medial malalignment of the quadriceps and the patella.
These changes become more pronounced as the animal grows and higher grade luxations are often associated with more severe secondary changes. Treatment of this condition is somewhat controversial with various surgical procedures described and no absolute consensus relating to timing of surgery or grade of luxation for which surgery is recommended. Similar to most orthopaedic conditions treatment is grouped broadly into conservative (medical) management and surgical management. Regardless of whether surgery is performed or not, these joints are not normal joints and so conservative management for osteoarthritis (weight reduction, exercise modification, EPA rich diet, pentosan injections, and NSAID's as required) is an important part of overall management.

In my opinion surgical correction of patellar luxation aims to achieve 3 goals:
1. Improve/resolve the clinical signs: The "lameness" associated with patellar luxation may be due to 3 factors
a. Altered biomechamics in the stifle when the patella is located medially
b. Pain associated with the secondary development of osteoarthritis
c. Pain due to instability created by secondary rupture/ damage to the anterior cruciate ligament. Reducing the patella luxation through realigment of the quadriceps, patella and the tibial crest with the trochlear groove restores the biomechanics and helps to reduces the wear on the articular cartilage.
2. Slow the progression of arthritis: A recent publication indicated that around 60% of dogs with medial patella luxation had evidence of cartilage erosions on the underside of the patella. Larger dogs and those with grade 4 luxations were more severely affected. 2 The cartilage on the medial trochlear ridge is also damaged as the patella repeatedly luxates and reduces. This damage to the cartilage will perpetuate osteoarthritis and is likely to progress if the patella is unstable.
3. Reduce the likelihood of cranial cruciate ligament rupture: The quadriceps is an important secondary stabilizer preventing cranial translation of the tibia (tibial thrust). When the patella is luxated there is more stress placed upon the cruciate ligament and therefore a greater chance of damage to the ligament. In one study 41% of dogs had a concurrent cranial cruciate ligament rupture. The likelihood of rupture was associated with increasing grade and with grade 4 luxations. 3 Based on these aims I recommend surgery for all animals with a

grade 2 or above luxation. I also feel that it is important for patients with lower grade luxations to be operated on earlier while there is less secondary degenerative change present. In general the surgery that we perform at Southpaws is a tibial crest transposition and lateral fascial imbrication. In a small number of dogs a medial fascial release is required to allow the patella to track in the trochlear groove. I do not routinely perform or recommend trochleoplasties of any type. At Southpaws our recurrence rate for luxation, using only a tibial crest transposition, is around 10% and is in line with the reported incidence of recurrence. 4 The concern with performing a trochleoplasty as a matter of routine is that you are creating potentially unnecessary trauma to the articular cartilage that can perpetuate the progression of
osteoarthritis. It is important to remember that stifles with a patella luxation are not normal joints and that there will likely be progression of osteoarthritis even when surgery is performed. The hope is that surgical intervention will slow the progression of the arthritis more than if surgery was not performed. Having said this it is always important to match the owners expectations with the treatment that is prescribed and to weigh up the benefits vs the risks/complications of performing the surgery. In some cases surgical intervention may not be the best course of action.

Cranial Cruciate Ligament Rupture in Dogs

Cruciate ligament rupture is the most common orthopaedic disease seen in dogs. Traditionally, it was thought of as a specific injury resulting from acute stress on the knee causing an acute ligament tear. More recently, theories of an abnormal slope to the tibial plateau have been developed which probably more accurately address the inciting cause.

When the tibial plateau is tilted backwards, the femur tends to slide "downhill" putting stress on the cruciate ligament. Repeated stress ultimately results in failure. Studies have shown that in dogs weighing less than 15 Kg, conservative (non-surgical) management is often appropriate. This involves weight-loss, pain relief, cartilage protectants and exercise modification (physiotherapy). In dogs weighing over 15 Kg, non-surgical management is only associated with a 40% success rate. Surgical management results in an 85-95% success rate depending on issues like body weight and proportions, rehabilitation and surgical technique used.

I regularly perform three different techniques for cruciate ligament repair. There are advantages and disadvantages to each. The traditional technique for cruciate ligament repair is extracapsular repair. It involves replacement of the ligament with a piece of nylon. Advantages of this technique include technical simplicity, infrequency of complications and cost. These are most commonly done at primary care veterinary clinics with good success. The primary disadvantage is a much higher incidence of severe arthritis when compared with other procedures. It is most appropriate for small to medium sized dogs with a moderate level of activity.

Step-by-Step for Extracapsular repair

1. Clip and sterile preparation from the level of the hip to the level of the hock.
2. Hang the leg for a sterile orthopaedic prep.
3. Make an incision on the lateral side of the stifle from a level about 2 cm above the patella to a level of the distal end of the tibial crest.
4. The fascia lata is incised to a level of about 2 cm above the patella.
5. The patella is luxated medially and retracted using Gelpi retractors.
6. The cruciate ligament is examined and debrided.
7. A Hohman retractor is placed behind the tibial plateau and is used to push the femur out of the way. This can be difficult if the cruciate ligament is only partially torn.
8. The menisci are examined. If there is a partial tear, the portion that is torn is debrided.
9. The joint is flushed using sterile saline.
10. The joint capsule is closed using simple continuous suture, but the fascia lata is not closed at this point.
11. The fascia lata is dissected and retracted caudally to allow exposure of the lateral fabella.
12. A 14 gauge needle is advanced medially from a point just distal to the femoro-fabella ligament.
13. A right angle forcep is used to grasp the end of the 14 gauge needle.
14. The grip on the needle is loosened slightly and the nylon suture is advanced through the needle with several centimeters protruding past the end of the needle.

15. The needle is retracted until the jaws of the forcep are only grasping the suture (tightly).
16. The needle is retracted completely and the right angle forceps are pulled out with the suture in the jaws.
17. The suture is passed under the patellar tendon from lateral to medial.
18. A hole is drilled in the tibial crest as far caudal and proximal asa is possible using the 14 gauge needle.
19. The suture is passed through the needle from medial to lateral. The bone plug may have to be cleared from the needle first.
20. The two ends of the suture are then tied tightly or crimped.
21. The fascia lata is closed in a simple continuous pattern.
22. The subQ and skin are closed in routine fashion.

The second commonly performed technique is called the TTA or tibial tuberosity advancement procedure. It is a fairly new procedure which has had very favourable results. In this procedure, the tibial crest is advanced forward so that the patellar tendon acts like the cruciate ligament. The advantages include very rapid recovery, when compared with any other repair technique, favourable outcome, and cost when compared with a tibial plateau leveling osteotomy. The disadvantages include relatively short track-record and potential for meniscal injury. The meniscus is a shock-absorber between the femur and the tibia. A meniscal release procedure can prevent this damage in the future. Results to date are very encouraging and we have been particularly impressed with the speed of recovery.

The third procedure is the TPLO or tibial plateau leveling osteotomy. This procedure has been around for around 20 years and the results are as good or better than anything else published to date. This procedure is based on the fact that the tibial plateau in cruciate-deficient knees is tilted backward, causing excessive tension on the cruciate ligament. The top of the tibia is cut and rotated to make the cruciate ligament obsolete. The advantage of this procedure is the excellent outcome. The disadvantage is cost when compared with the other procedures.

Perioperative care, wound healing, patient warming and prevention of surgical infections

Surgical infections

Prevention of surgical infections involves rigorous adherence to principles of sterile technique. Many other factors have been identified which also are associated with the development of surgical infections. Some of the factors appear to be counterintuitive and even blasphemous. All of the factors presented in these notes have been documented through peer-reviewed veterinary and human literature.

Patient warming

One of the most important factors is the prevention of hypothermia before, during and after surgery. Appropriate patient warming provides patient comfort, owner satisfaction, infection control, improved wound healing, more rapid anaesthetic recovery, improved tissue profusion, improved kidney function and improved survival. Inappropriate patient warming can result in severe and even life-threatening burns.

Factors known to cause excessive patient cooling include recovery on a cold surface, wet fur, the excessive use of vasodilating drugs (like ACP), irrigation of the abdomen with room-temperature fluids, urine-soaked fur, excessive pain (causing vasoconstriction), and excessively long anaesthetic episodes. I have identified methods of patient warming which are more likely to cause skin burns. Some of these burns are severe, full thickness and, again, may be life-threatening. Debilitated patients may not be able to get away from dangerous heat sources. I feel very strongly that anything that has been placed in the microwave should not be used in contact with patient skin unless the temperature can be verified. Nothing should be in contact with patient skin that is over 43 C. This includes fluid bags,hot water bottles, oat bags, rice bags, etc. The interposition of towels will not prevent s akin burns. Non-medical electric heating pads should not be used. Heat lamps should also not be used unless very closely monitored. My stepmother fell asleep with an electrical heating pad on her neck. She suffered severe full-thickness burns which required numerous reconstructive surgeries to resolve.

Patient warming is a very gratifying part of veterinary practice. It gives a great deal of satisfaction to see a patient sitting comfortably in its cage following surgery who is toasty warm with appropriate pain relief. Nursing staff should be encouraged to take a very active role in assuring that patients are warm after surgery. An obvious first-step in maintaining patient temperature is measuring patient temperature. Rectal, oral or ear temperatures should be taken every 5 minutes during an anaesthetic procedure. Many new anesthetic monitors include temperature which can be displayed constantly and trends can be recorded. I strongly recommend keeping patients dry. Anaesthetics should be as short as reasonably possible using low doses of vasodilating drugs. I also recommend wrapping patients in blankets, cushions, bubble wrap during and after surgery.

Any device which is in contact with the patient with the intent of maintaining temperature should optimally be 42-43 C. Probably the best source of warmth is a medical warm air device which delivers air at a selected temperature through disposable or reusable blankets. They offer the added advantage of drying wet fur, as well. Circulating warm water blankets work as well but may be punctured.

I have purchased, second-hand, a large incubator from a microbiology lab which is the size of a small refrigerator. It is set to 43 C and we keep fluids, hot water bottles and blankets for use in patients. It guarantees that the temperature will not be any higher than 43 degrees. Even medical grade electric heating pads can have hot-spots. I recommend the use of warmed abdominal lavage fluids, ideally from an incubator. If they are warmed in the microwave, the temperature should be tested using a thermometer before using them in a patient.

Regarding prophylactic antibiotic use, I recommend a single dose of intravenous ampicillin or cefazolin at induction and every 90 minutes through the surgical period. With clean surgical wounds, antibiotics given after 24 hours after surgery INCREASE the surgical infection rate due to selection for resistant strains of bacteria. This has been definitively demonstrated in multiple studies. Again, do not use antibiotics prophylactically after 24 hours after surgery with clean surgical wounds (cruciates, closed fractures, tumour removals, etc).

Other factors which have been shown to prevent surgical infections include avoiding the use of drains. That's right- surgical drains definitively increase the infection rate. Instead, use delicate tissue handling, closing of dead space and excellent haemostasis. If drains must be used, I prefer the use of a single closed suction drain which exits very close to the primary incision. If penrose drains must be used, they should be bandaged because ascending infections can travel up the drain as easily as fluid can drain out.

Further measures to prevent surgical infection include patient hygeine, prevention of self mutilation, and keeping the surgical time as short as reasonably possible. I also recommend the use of disposable drapes, which can be purchased for as little at 17.00 per patient for utility drapes, patient drape, instrument table drape and two surgical gowns. Obviously, not scrubbing, not wearing surgical gloves, and not wearing surgical gowns went out of styles early in the last century and are inexcusable.

Wound healing and the selection of suture materials

Wound healing and bandage changes

One of the major tenets of cancer surgery is to remove the tumour aggressively, with no concern for inability to close. Dogs have a remarkable ability to heal. Healing takes place by granulation, contraction and reepithelialisation. We often have very large wounds completely heal by second-intention healing.

Key in supporting wound healing is placement of a good bandage. I always use stirrups to keep the bandage from falling off. The contact layer I prefer is a dry non-adherent (Melolin, Telfa, Adaptic). I then use several rolls of cotton cast padding. This provides stability and helps prevent "benching" of the bandage. I often use 3-4 rolls of cotton. Cling is applied next. The stirrups are separated and applied to the bandage. Vetwrap is then place. On a granulating wound, I do bandage changes every 3-5 days. It is critical that the bandage not get wet and that the owners not modify a bandage because it has slipped, has gotten wet, or appears to be causing swelling of the toes. If any of the above occurs, the owners immediately need to get back to the vet. If they cannot, I prefer that they completely remove the bandage and leave it off than attempt to revise it themselves.

1. Phases of wound healing

a. Acute phase
    i. Vasoconstriction (5-10 minutes) to reduce blood loss.
    ii. Vasodilation, increased vascular permeability and leukocyte infiltration (chemotactic agents-diapedesis)
        1. Pain, redness, swelling, dysfunction, heat
    iii. Platelets aggregate
    iv. Coagulation cascade
        1. Exposure of subendothelial collagen
        2. Object is to stabilize platelet plug
    v. Fibrin clots to control haemorrhage- Provides some strength to wound on first postoperative day, but most wound strength comes from sutures.

2. Lag Phase (inflammation and oedema) (usually 3-4 days)
    i. Lag phase continues until wound is clean
        1. Can accelerate this phase if contaminated wound is debrided surgically or with wet-to dry bandages
    ii. Fibrinolysis occurs due to plasminogen activation to plasmin
    iii. In visceral wounds, epithelial regeneration begins immediately, but epithelium provides little strength. Sutures and fibrin must be present to prevent leakage.
    iv. Most critical in visceral wounds because most leakage occurs during this time period.
    v. Important cells
        1. Neutrophils
        2. Macrophages (most important)
    vi. Delays in wound healing usually occur in this phase
        1. Infection
            a. Causes
                i. Foreign bodies
                    1. The greater the inflammatory response of suture material the more likely the infection
                    2. Braided, nonabsorbable suture material
                ii. Dead space
                iii. Disruption of blood supply
                iv. Tension on tissues
                v. Poor haemostasis
                vi. Length of surgery
                vii. Lack of prophylactic antibiotics
                viii. Damage to tissues via rough handling

b. Proliferative phase or logarithmic phase (day 3 or 4 to day 14)
    i. Again, can only occur once wound is clean
    ii. Rapid fibroblast proliferation
    iii. Fibroblasts produce immature collagen resulting in rapid wound strengthening.
    iv. Collagenolysis occurs due to collagenase activity (not a major factor in the intestine, stomach and urinary bladder.)

v. At the end of 14 days, gastric and small intestinal wound bursting strength is 75% of normal. Urinary bladder is even faster with 100% bursting strength by 14-21 days.
vi. Colon is slower with only 50% at 14 days, with marked collagenase activity at the wound edge.
1. Factors like traumatic suturing, foecal contamination, bacterial contamination and infection increase collagenase activity at the wound edge.

c. Maturation phase (day 14 – 180 g.i. tract and 14-90 in urinary bladder)
i. Reorganisation and cross-linking of collagen.
ii. Size and thickness of scar decreases without decrease in wound strength.
iii. Contraction of wound.
1. Fibroblasts take on contractile properties (actually develop actin and myosin filaments)
2. Can use corticosteroids and colchicine to reduce contraction

3. Wound healing always involves a race between implant failure and tissue healing
a. Every implant will eventually fail be it suture, intramedullary pin or dynamic compression plate
b. Object is to choose implant that will maintain strength until tissue can take over
c. Different tissues have different rates of collagen deposition
d. Different suture materials have different rates of degradation
i. Suture material should be selected based on expected rate of wound healing and absorption profile of suture material
e. Different patients have different rates of wound healing

Biography:

Charles graduated from the University of Florida in 1990. He then did an internship at the Animal Medical Center in New York City. He completed a residency and Master's degree in surgery at Virginia Tech in 1994 and achieved specialty board certification in surgery in 1996. He did a one year fellowship in cardiovascular research and surgery. He completed a fellowship in surgical oncology at Colorado State University, and is one of 20 people world wide to have received this training. He was then a professor of Orthopedic Surgery at Colorado State University before he left for Northern Virginia where he started a surgical referral practice which was among the busiest in the Washington DC area. Charles moved to Australia 4 years ago and is the director of SouthPaws Specialty Surgery for Animals in Melbourne.

Charles has published many scientific articles, summary articles, abstracts, proceedings and book chapters on topics of surgical oncology and general and orthopaedic surgery. He was the chairperson of the oncology section of the National Meeting of the American College of Veterinary Surgeons. He is the section editor of the oncology section of the current edition of Slatter's Textbook of Small Animal Surgery. He was the surgical expert on panel discussions of feline vaccine associated soft tissue sarcomas at recent meetings of the American College of Veterinary Surgeons and the American College of Veterinary Internal Medicine. He was asked to write a chapter for a human surgical oncology textbook in bone cancer because of his reputation and expertise in cancer surgery. He started and currently runs Australia's first

deep radiation therapy unit for animals. He has 5 United States patents for devices used in the treatment of diseases in animals. He has personally operated on over 5,000 patients with cancer with local cure rates of over 95%. Charles has been seen on "Talk to the Animals", "Animal ER", "A Current Affair", "National Nine News", "The Today Show" as well as numerous appearances on ABC radio. He receives referrals from all over Australia and consultations by phone and email world-wide.

Functional anatomy of the canine stifle

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Stifle joint disorders are one of the major causes of hind limb lameness in the Dog. Knowledge of the anatomy and function of joint components is required for establishing an accurate diagnosis and performing an effective treatment.

The stifle is a composite joint with two functionally distinct parts. Weightbearing occurs primarily through the **femorotibial joint** that units the femoral and tibial condyles. The **femoropatellar joint** greatly increases the mechanical efficiency of the quadriceps muscle in stifle extension (1).
In the dog, the stifle also includes the proximal tibiofibular joint as well as the joints between the femur and paired sesamoids in the origins of the gastrocnemius (2). All these articulations share a common synovial cavity.

I – Femorotibial joint

A. Menisci

The femorotibial joint includes two fibrocartilaginous **menisci** interposed between the **femoral and tibial condyles**. The menisci, which compensate for the incongruence of the articular surfaces, are each semilunar in plan and wedge-shaped in section. They have concave proximal and flattened distal surfaces; their peripheral border is thick and convex, with attachments to the joint capsule, whereas the central border is thin and concave.
Each meniscus is attached to the proximal tibia (tibial plateau) by cranial and caudal ligaments. The lateral meniscus has an additional ligament to the intercondylar fossa of the femur, the meniscofemoral ligament.
The cranial tibial ligaments of the menisci extend from the cranial part of each meniscus to the lateral and medial cranial intercondyloid area of the tibia.
The caudal tibial ligaments of the menisci extend, for the lateral ligament, from the caudal angle of the lateral meniscus to the popliteal notch of the tibia, and for the medial ligament, from the caudal angle of the medial meniscus to the caudal intercondylar area of the tibia.
The femoral ligament of the lateral meniscus (meniscofemoral ligament) passes from the caudal angle of the lateral meniscus to the inside of the medial femoral condyle.
The small transverse or intermeniscal ligament connects the cranial angles of the two menisci.
It is an important landmark as it overlies the tibial attachment of the cranial cruciate ligament and may be used to anchor grafts used for cranial cruciate ligament reconstruction (1).

The menisci decrease the incongruity of the joint, increase the stability of the stifle and act as shock absorbers.
The medial meniscus is attached to the medial collateral ligament and joint capsule, and moves only slightly when the stifle is flexed. The lateral meniscus is separated from the lateral collateral ligament by the tendon of origin of the popliteus muscle, and is attached to the femur, which pulls it caudally as the lateral femoral condyle moves caudally over the tibia. During flexion of the stifle, both menisci move but the lateral one is free to move farther.

The menisci are prone to traumatic injury. Injury to the medial meniscus is more commonly associated with tearing of the cranial cruciate ligament and excessive medial (internal) tibial rotation (3). After a cranial cruciate ligament rupture, the tibia glides cranially during weight bearing and the medial meniscus is drawn cranially with the tibia so that its caudal part moves under the femoral condyle, where it can be crushed.

B. Femorotibial ligaments

The primary ligamentous support of the stifle joint is provided by the **femorotibial ligaments**, which are the medial and lateral collateral ligaments and the cranial and caudal cruciate ligaments. The collateral ligaments, extra-articular, unite with and provide some support to the joint capsule. The cruciate ligaments invaginate the joint capsule from the caudal aspect of the joint and are covered by a layer of synovial membrane; thus, they are intra-articular but extra-synovial (1).

1. Collateral ligaments

The **medial collateral ligament** passes between the femoral epicondyle and the proximal part of the tibia, toward the caudal part of the joint. This ligament blends with and forms a strong attachment to the joint capsule and medial meniscus. It extends distally across the medial tibial condyle. A fluid-filled bursa is generally present between the medial collateral ligament and the tibia, suggesting translational movement between the ligament and bone surfaces.
The cranial border of the medial collateral ligament remains taut throughout a normal range of motion; however, the caudal portion becomes lax in flexion.

The **lateral collateral ligament** has a similar disposition but attaches to the fibular head (and adjacent lateral condyle of the tibia). It arises from the lateral epicondyle of the femur just proximal to the origin of the popliteal muscle and passes superficial to the popliteal tendon. Only loose connective tissue joins the lateral collateral ligament to the joint capsule, and there are no attachments to the lateral meniscus.
The entire lateral collateral ligament is lax in flexion, becoming taut during extension.

The collateral ligaments are primarily responsible for **limiting varus** (lateral collateral ligament) **and valgus** (medial collateral ligament) motion of the tibia. Their effect is most pronounced in **extension**; as the stifle joint flexes, the cruciate ligaments become stressed by varus or valgus loads and are increasingly important in limiting varus and valgus motion (1).

2. Cruciate ligaments

The cruciate ligaments pass between the intercondylar areas of the femur and tibia and limit craniocaudal motion of these bones. The ligaments cross each other near their attachments in the intercondylar fossa of the femur. The **cranial and caudal cruciate ligaments** are named for their respective tibial insertion sites.

The **cranial** (lateral) **cruciate ligament** arises from the caudomedial part of the lateral condyle of the femur within the intercondylar fossa and extends diagonally, craniodistally, to attach on the central (and cranial) intercondylar area of the tibia, just behind the cranial attachment of the medial meniscus (4).
The cranial cruciate ligament keeps the tibia from sliding cranially beneath the femur when the limb bears weight (the cranial translation of the tibia relative to the femur that is

observed when the cranial cruciate ligament is ruptured is called "**cranial drawer motion")**. It also limits medial rotation of the tibia when the stifle is flexed.
This ligament is composed of numerous bundles of collagen fibers, grouped into fascicles of various sizes. The entire ligament spirals laterally about 90° between attachment sites. The twist in the ligament results in the gross appearance of two fairly distinct « bands », especially in flexion. If the ligament is untwisted, it is more uniform in appearance. In this context two functional components, a craniomedial band and a larger caudolateral portion have been described. Differences in the mechanical properties of each subunit have been reported, with the craniomedial band providing the most resistance to cranial drawer motion at 30° and 60° of flexion. The craniomedial band is taut throughout the range of motion, and the caudolateral portion is taut in extension but lax in flexion (1).

The **caudal** (medial) **cruciate ligament** runs approximately at right angles to the cranial one. It is attached to the medial femoral condyle within the intercondylar fossa, and is directed caudodistally and ends at the medial edge of the popliteal notch of the tibia, caudally to the caudal attachment of the medial meniscus.
The caudal cruciate ligament is also separated into two functional components. The relatively larger cranial portion is taut in flexion and lax in extension, and the caudal portion is taut in extention and lax in flexion.
This ligament prevents caudal movement of the tibia beneath the femur when the limb bears weight (the caudal translation of the tibia relative to the femur that is observed when the caudal cruciate ligament is ruptured is called "**caudal drawer motion**").

The cruciate ligaments also provide rotational stability to the stifle, by twisting with each other. This is however primarily a function of the cranial ligament, which limits medial (internal) rotation of the tibia on the femur when the stifle is flexed. Medial rotation of the tibia is ample when the stifle is flexed, but with rupture of the cranial cruciate ligament, this is significantly increased (3). The cranial cruciate ligament also limits stifle hyperextension.
Extreme and unexpected internal rotation of the tibia on the femur, and overextension of the stifle, are common circumstances of cranial cruciate ligament injuries.

The primary blood supply to the cruciate ligaments arises from the synovial tissues that ensheaths the ligaments rather than from sources arising at osseous attachment sites. The infrapatellar fat body and soft tissues caudal to the joint are important sources of vessels. The central core of the midsection of each ligament is less vascularized (1).

II – Femoropatellar joint

The **femoropatellar joint** is formed between the femoral trochlea and the patella, extended by its **parapatellar fibrocartilages**. Relatively weak **lateral and medial femoropatellar ligaments** run between these cartilages and the femur. Distally the patella is joined to the tibial tuberosity by a single **patellar ligament** in the dog, which represents the "insertion tendon" of the quadriceps femoris (2).

The medial and lateral femoropatellar ligaments are narrow bands of loose fibers that partially blend with the overlying femoral fascia. They extend from the patella to the respective sesamoid bones of the gastrocnemius muscle, medially and laterally. The lateral ligament is usually visible, whereas the medial ligament often blends with femoral periosteum and is not easily discernible. These ligaments combine with the more substantial femoral fascia to support the patella in the femoral trochlea.

The stifle joint is flexed in the standing posture. Though it is more fully extended in certain phases of locomotion, the femur and tibia are never brought into line, and in dogs the caudal angle of the joint does not open beyond 150 degrees or so (considerably greater extension is permitted to cats) (2). Some lateral or medial angulation of the joint may often be observed when the limb is viewed from in front or behind. In the « bowlegged » version common in certain toy breeds, the pull of the quadriceps does not coincide with the axis of the femoral trochlea, and there is a tendency to medial **luxation of the patella**.

III - Synovial membrane

The synovial membrane attaches around the peripheries of the articular surfaces and the menisci. It covers the cruciate ligaments and here forms a partition, but not complete in the dog, between the medial and lateral femorotibial joints. The femoropatellar portion of the cavity extends proximally between the femur and the quadriceps. Diverticula of the capsule embrace the lesser joints with the fibula and the sesamoid bones and extend along the tendons of origin of the long digital extensor and popliteus muscles.
Distal to the patella, the synovial and fibrous layers of the joint capsule are separated by the **infrapatellar fat body**.

IV - Mouvements

Despite its complexity, the stifle functions as a hinge joint with free movement restricted to flexion and extension. The femoral condyles roll on the menisci, and these in turn slide over the tibial plateau-cranially on extension, caudally on flexion. The travel between the femur and menisci is about three times that between the menisci and the tibia. The spiral configuration of the femoral condyles - when viewed from the side - tightens the ligaments and slows the movements when the joint moves toward the extended position. The stability of the articulation depends much on the **cruciate ligaments**.
The cruciate ligaments assist the collateral ligaments in opposing rotation and medial or lateral deviation of the leg. They are most susceptible to injury when tautened. The cranial cruciate ligament is therefore at highest risk when strained in overextension of the joint; its rupture allows abnormally free forward displacement of the tibia in relation to the femur (the **« cranial drawer » sign**). The caudal cruciate ligament is at greatest risk in the flexed position of the joint, and its rupture allows excessive caudal displacement of the tibia (the **« caudal drawer » sign**). Various surgical techniques for the restoration or replacement of these ligaments use fascial or artificial substitutes.

Flexion and extension occur in the sagittal plane, with the normal range of motion being about 140°. Because of ligamentous constraint and the complex geometry of the articulations involving the femoral and tibial condyles and the menisci, in particular the irregular contours of the femoral condyles, simple uniplanar rotation about a stationary axis does not occur. With flexion, the lateral collateral ligament relaxes and allows the lateral femoral condyle to displace caudally, resulting in internal rotation of the tibia. Conversely, during extension, the lateral collateral ligament tightens and causes the lateral femoral condyle to move cranially, resulting in external rotation of the tibia (1). A small amount of craniocaudal motion also occurs in the sagittal plane as a result of the cam shape of the femoral condyles. The femoral condyles roll caudally with flexion and cranially with extension, relative to the tibial plateau.

Slight varus (medial) and valgus (lateral) movement of the tibia occurs in the transverse plane. The collateral ligaments are responsible for limiting this motion in the extended joint; with flexion, the cruciate ligaments also contribue to the control of varus and valgus motion. Excessive joint motion is prevented not only by the ligamentous constraints of the stifle joint but also by a complex system of reflex arcs that involves the major muscle groups around the stifle.

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CURRENT CONCEPTS IN VETERINARY OPHTHALMOLOGY

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EXAMINATION OF THE EYE

Vision evaluation

The animal should be observed walking into the examination room with the client, or in its own environment. A blind animal may exhibit pricked ears or a tentative gate as he walks into the exam room. He may collide into objects, have a stare-like expression, or reluctant to move in a strange environment. The owner's impression that the animal "sees" well at home must be interpreted cautiously. Animals can "memorize" their own environment and often can adjust very well to the loss of sight. Once the animal is in the exam room, the animal is permitted a few minutes to adjust to the room and observed as the history is obtained.

The patient's vision can be further evaluated by noting the response to hand movements, bright lights or to cotton balls tossed into the visual field. The menace response and the visual placement reaction can also be performed to evaluate the vision. In certain circumstances, each eye should be evaluated separately by covering one eye with your hand.

Examination for evaluation of vision should be performed in normal light and then in dim light. If you can see the cotton balls or the obstacles of the maze test, the dog or the cat should be able to see them better than you since their night vision is more developed than ours. Cats generally do not menace well, but respond well to bright light stimulation (such a **laser pointer**) and cotton ball testing.

Ocular examination

The ophthalmic examination is conducted in a quiet, slightly darkened room. The patient is placed on the examination table and restrained by the owner or an assistant. Minimal but firm restraint under most circumstances is sufficient to permit a thorough ocular examination. Tranquilization and rarely general anesthesia may be necessary for certain diagnostic procedures. Also, these drugs may produce artifacts such as the lowering of intraocular pressure which may lead to erroneous conclusions.

Following an evaluation of vision the need for special diagnostic tests is determined. An orderly sequence of diagnostic tests must be followed based on the special requirements of each test. Evaluation of the tear film (Schirmer tear test) must be done before the eye is manipulated or any drugs are instilled. Cultures of the external ocular structures must be done before extensive cleaning is done and before drugs are instilled. The use of mydriatics is necessary for examination of the lens and posterior segment, but should not be given prior to measuring the intraocular pressure (IOP). The intraocular pressure evaluation requires topical anesthetic and must be recorded before excessive manipulation or before the patient becomes restless and excited.

Periocular Exam:

Examination of anatomic structures should begin with the orbit and other periocular tissues.

1. Orbit: Evaluated for symmetry, eye-orbit relationship, deformities or enlargements. Because of marked variations in eye position of different breeds, one should be acquainted with the various breed characteristics.
2. Globe Position: The presence or absence of **strabismus** and **nystagmus** is noted. **Esotropia (crossed-eyes)** is inherited in Siamese cats but in dogs may represent severe intraocular or neurological disease. Nystagmus occurs frequently in Siamese, apparently not always associated with clinically detectable vision defects, but in dogs may result from congenital intraocular diseases, or acquired vestibular or cerebellar diseases.
3. Eyelid Position: may be helpful in determining relative globe size. Looking from over the top of the animal's head helps to estimate globe position. Additional evaluation of the orbit consists of examination of the mouth (floor of the orbit), palpation of orbital rim, retropulsion of the globe, and evaluation of nasal patency, if necessary.
4. Extraocular Muscles: the function of the extraocular muscles is tested by the **" tonic eye reflexes**." As the head is moved right and left, up and down, an optokinetic nystagmus occurs. The animal's ability to follow a moving object (light, hand, etc.) without moving the head can also help in evaluating the function of the extraocular muscles. The integrity of the 3rd, 4th and 6th cranial nerves and their respective muscles is determined through this technique.

Adnexa: Eyelids
The eyelids are examined for abnormalities of position, function and structure such as lagophthalmos, ptosis, trichiasis, ectropion, entropion, blepharitis, lid neoplasms, etc. The lids are evaluated without topical or general anesthesia, sedatives and tranquilizers. How the head is restrained may also alter your evaluation of eyelid position and function. Adequate illumination and some magnification may be necessary. The otoscope with speculum removed, penlight and various hand held or head mounted magnifiers, and including the slit lamp biomicroscopy may be used.

The **blink reflex** should be evaluated. The efferent limb of this reflex requires the integrity of the facial nerve (CN VII) and the orbicularis oculi muscle. The afferent limb may be a menace (CN II), corneal sensation (CN V) or touch sensation to the periorbital skin (CN V). Rapidity and completeness of the blink should be evaluated.

The lower and upper eyelids should touch the globe. Lower lid-globe contact is important to prevent accumulation of tears and debris. The lower "lacrimal lake" may be grossly distorted by anesthetics and tranquilizers. Cilia or eyelashes occur mainly on the dog's upper lid in three irregular rows. The lower eyelids of dogs and both eyelids of cats are usually void of cilia. The eyelid contours are regular and gently curved, partially exposing the openings of the tarsal or **Meibomian glands** (gray line). The duct orifices are frequently raised and nonpigmented. Aberrant cilia (distichia) may emerge from the spaces among the Meibomian gland ducts, or the actual duct orifices. **Ectopic cilia** emerge from the within the palpebral conjunctiva and are frequently the same color as the dog's hair coat. They can escape detection without careful examination.

Conjunctiva and third eyelid
The **palpebral conjunctiva** is examined by manual eversion of the upper and lower eyelids. Although the lower eyelid everts rather easily, the upper palpebral conjunctiva, to be adequately examined, may need topical anesthesia and eversion of the lids by digital pressure, chalazion forceps, Q-tips or wooden tongue depressors. Excessive lymphoid

follicles, increased vascularity, foreign bodies, ectopic cilia, obstructed tarsal glands, hemorrhage, lacerations, abnormal growths and edema (chemosis) may be abnormalities observed. Coloration of the conjunctiva can be used to assess the presence of anemia and icterus. Because the palpebral conjunctiva is transparent, chalazia or impacted Meibomian glands appear as slightly raised yellow masses.

Examination of the palpebral (outer) and bulbar (inner) surfaces of the nictitans is important for diagnosis of several common external ocular conditions. Frequent abnormalities are eversion of the cartilage of the nictitans, prolapse of the gland (cherry eye), foreign bodies, follicular conjunctivitis, and enlargement of the secretory gland, foreign bodies, follicular conjunctivitis, and enlargement of the bulbar lymphoid tissue.

Examination of the palpebral conjunctiva is usually accomplished by digital manipulation; distorting the palpebral fissure and retropulsing the globe causes protrusion of the nictitans. Examination of the bulbar aspect requires topical anesthesia, thumb forceps (without rat teeth) and restraint of the head. Occasionally, with intractable individuals, sedation or general anesthesia is necessary. The leading margin of the nictitans is gently grasped and pulled medially, allowing visualization of the bulbar surface and fornix.

Sclera

The **sclera** is examined from the limbus to near the equator. By moving the patient's head in different directions, the more posterior aspects are inspected. The sclera should be scrutinized for change in color, abnormal masses, and tears or lacerations. Small vessels in the episclera are usually visible and occasionally a large vortex vein (especially the dorsolateral vein) can be seen. Enlargement and congestion of the episcleral veins occur commonly with glaucoma. This venous enlargement remains even after the glaucoma is "controlled". Hyperemia of the episcleral vessels occurs in association with inflammatory conditions. The "ciliary flush" or limbal hyperemia from iridocyclitis is usually less affected by topical phenylephrine while that associated with the conjunctivitis will usually blanch. The perilimbal scleral vessels are small straight and immovable vs larger mobile and branching conjunctival vessels.

Cornea

Examination of the cornea is accomplished with a focal light source and magnification (head loupe or hand held lens) or with the slit lamp biomicroscope. Measurement of the cornea using calipers may provide approximation of globe size, except in cases of micro and megalocornea.

Corneal sensitivity (corneal reflex) is tested by a small wisp of cotton gently touched to the cornea. (This must be done prior to topical anesthetic instillation). If the animal sees the stimulation, you will get a false positive.

The cornea is normally transparent, avascular, moist, and unpigmented with a smooth, even contour. It should be carefully examined for loss of transparency (edema or infiltrates), opacity, vascularization, pigmentation, dryness, growths, foreign bodies, lacerations, changes of contour, and ulceration.

Two types of vascularization occur in the cornea: superficial and deep. Superficial vessels occur in the anterior one-half of the corneal stroma, are usually continuous with visible conjunctival vessels, are "tree-like", and associated with external corneal diseases. Deep vessels appear as small, fine vessels in the corneal stroma that extend from the anterior sclera

or deeper limbal vessels (paint brush border), and are associated with intraocular inflammation.

Slit lamp biomicroscopy permits the most accurate localization of corneal lesions. The layers can be differentiated into epithelium, stroma, and Descemet's membrane - endothelium.

Examination of the cornea is incomplete without utilization of topical ophthalmic stains. **Fluorescein is used to demonstrate the presence or absence of corneal ulcers.** For topical use, fluorescein impregnated paper strips are preferred to fluorescein solution to insure sterility.

Because the water-soluble fluorescein stains the preocular film, a **faint** green may occur on the corneal surface. The corneal epithelium is lipid-selective and prevents any appreciable corneal penetration by fluorescein. In the presence of a corneal epithelial defect, the dye rapidly diffuses into the corneal stroma. An area of fluorescein retention by corneal stroma is indicative of an epithelial defect (a corneal ulcer/erosion).

Rose Bengal is a valuable stain in the evaluation of the health of the corneal and conjunctival epithelium. It produces a brilliant red coloration of any dead or degenerating cells, and indicates defects in the mucin layer of the tear film. Rose Bengal is retained by the cornea and conjunctiva in early fungal keratitis, keratoconjunctivitis sicca, pigmentary keratitis, exposure keratitis, viral keratitis, and certain other corneal ulcers.

Anterior chamber and iris

The anterior chamber is evaluated from the anterior, lateral and dorsal aspects using magnification and a focused beam of light. The slit lamp biomicroscope provides additional magnification. The anterior chamber can be evaluated for elevated protein levels (flare), blood, iris cysts, depth, parasites, cellular contents, and foreign bodies. Increased protein in the aqueous humor, when viewed with a focal light source, gives the appearance of a light beam passing through smoke. This is known clinically as "**aqueous flare**" and its appearance results from the optical Tyndall phenomenon. Examination for flare can be performed easily by using the small dot of the ophthalmoscope and shining the light from the front while looking from the side. Focus the dot on the cornea (usually <1 cm from the cornea). There is a space (the anterior chamber) between the small dot in the cornea and the long dot which represents the lens. A Tyndall effect as smoke in a movie theater beam indicates aqueous flare. **Aqueous flare means there is uveitis**. When checking for flare compare the depth of the anterior chamber between eyes and if you suspect a lens luxation there may be variation in chamber depth within an eye.

The iris is examined with a focused beam of light and magnification for color, shape, pupil size, surface, and movement. Iridal color in dogs varies from dark brown to blue, and generally 3 "zones" of color are evident (**pupillary margin, iris collarette and the iris base**). Light brown irides occur in many breeds, such as the Brittany Spaniels, German Short Hair Pointers and other breeds. Iridal heterochromia is not uncommon in white cats, St. Bernard's, Great Danes, Beagles, merle Collies, Australian Shepherds, Old English Sheepdogs, Dalmatians and the merle Sheltie. Iris color in cats varies from blue to yellow-green to brown. In acute iritis, the iris may appear congested and swollen with loss of detail, and it may become darker in appearance with chronicity. Transillumination and retroillumination is used for anterior segment diseases to differentiate hollow (i.e. cysts) from solid masses (possible tumors). The transilluminator is positioned in the limbal area. Light penetrates the sclera to highlight structures in the eye. Retroillumination utilizes the fundus reflex in the same manner.

Lens
The entire lens can only be fully evaluated with drug-induced mydriasis or in cases in which the pupil is already dilated. Many small focal cataracts can occur outside the central pupillary axis of the lens (at the equator) and will escape detection unless mydriasis is employed. The lens is examined by direct and oblique illumination with some magnification, direct ophthalmoscopy (set at +8 to +12 diopters), indirect ophthalmoscopy, slit lamp biomicroscopy, and retroillumination.

The lens, which is normally a transparent avascular structure, should be examined for opacities (cataracts), position, presence, and size. Focal cataracts should be localized within the various parts of the lens as prognosis and etiology may be suggested by location. Nuclear cataracts are usually stationary while those affecting the equator or posterior cortex are often progressive. By slit lamp biomicroscopy, the canine lens may contain focal imperfections that are not "cataractous." Early cataract formation, evidenced usually as focal crystallization, vacuoles and water clefts, can be detected long before visual disturbances occur. Localization of focal cataracts can be performed using the tapetal reflex to highlight the opacity and then observing which direction it moves as the animal's eye moves. For practical purposes, in the dog and cat the center of axis of rotation of the eye is the center of the lens. Thus if a cataract is in front of the lens it will move with the eye movement. If a cataract is in the back of the lens it will move in the opposite direction of the eye movement. Location of a cataract may give clues about its cause i.e. inherited or associated with PRA.

Nuclear sclerosis of the lens begins to develop in dogs around 6 years. Biomicroscopic examinations can detect refractive changes between the lens nucleus and cortex as early as three years of age in dogs. Advanced nuclear sclerosis is clinically evident as a blue zone limited to the lens nucleus that does not impair ophthalmoscopic visualization of the fundus and does not impair vision. This is frequently mistaken for cataract formation in older animals by owners and veterinarians.

Vitreous
The vitreous humor is normally a clear gel. The anterior portion can be examined using focal illumination and some magnification. The posterior aspect of the vitreous is examined by ophthalmoscopy or the slit lamp biomicroscope with added lenses. Frequently seen vitreous abnormalities include vitreous strands, asteroid hyalosis, hemorrhage and infiltration with inflammatory cells. Small remnants of the hyaloid vasculature (seen as white strands) are frequently encountered behind the central posterior lens capsule in the vitreous immediately posterior to the lens. Liquefaction of the vitreous is called syneresis, and opacities that occur in the liquefied state are called "synchysis scintillans". These opacities often rise and fall in the vitreous as the eye moves.

Differentiation of lens and vitreous opacities may pose a problem for the clinician. Localization of intraocular opacities can be achieved by noting direction of movement in relation to the center of the globe, or by slit lamp biomicroscopy. The first procedure is convenient and assumes the center of rotation of the eye is the posterior aspect of the lens nucleus in the dog. Opacities which are anterior will move with eye movement; for example, an anterior cortical cataract will move left when the eye turns left. Opacities posterior to the center of rotation will move in the opposite direction. In the horse the optical center of the eye is the posterior pole of the lens. The stability of the opacity may also help to differentiate lens from vitreous. Lens opacities are fixed and remain stationary when the eye stops moving. Vitreous opacities tend to move slightly or oscillate within the gel vitreous after eye movement ceases.

Fundus
The ocular fundus is examined last and requires direct and/or indirect ophthalmoscopy. Although the fundus can be viewed without drug-induced mydriasis, dilation of the pupil greatly facilitates examination of the complete ocular fundus. The ocular fundus is examined for changes in the normal appearance, detachment of the retina, chorioretinal hypoplasia or dysplasia, vascular patterns, attenuation, congestion, hemorrhage, colobomas, scars, alteration in coloration, changes in pigmentation and foci of inflammation. The optic disc should also be examined for size, shape, color, masses, and pits or colobomas. Swelling and inflammation of the optic disc occurs with optic neuritis, which is characterized by blindness. Myelination of the disk must be differentiated from swelling of the disk.

Pupillary light reflexes (PLRS)
The size of the pupils is evaluated and the direct and consensual pupillary light reflexes (indirect reflex) are tested. This should be done with a **bright** light (halogen) in a dimly lit room. A penlight, otoscope without speculum, transilluminator, or an ophthalmoscope is used. PLRs are tested early in the examination so you can start to chemically dilate the pupils for evaluation of the posterior structures of the eye.

The PLRs are affected by the psychic state of the animal, room illumination, age, many topical and systemic drugs and the intensity of the light stimulus. If an animal is highly nervous or frightened, the pupils may be dilated and respond poorly to low intensity light. However, with acclimation or with a strong light source, this effect is minimized. Older animals may exhibit slow and incomplete PLRs resulting from atrophy of the iris sphincter muscle. This is common in small dogs, especially poodles. The pupillary margin may have an irregular or scalloped appearance. Incomplete iris atrophy may give an irregular pupil shape.

The rapidity of pupillary light response, extent of miosis and ability to maintain miosis to constant light stimulation are evaluated. The consensual pupillary reflex is normally equal to the direct. The pupillary light reflexes require integrity of retinal neural cells, optic nerves, optic chiasm, optic tracts, midbrain (Edinger-Westphal nuclei), and parasympathetic fibers via the oculomotor nerve, ciliary ganglia and the iridal sphincter musculature. The reflex is subcortical and should be considered an evaluation of the retina and optic tracts, **not of vision**.

Drug induced mydriasis is not used indiscriminately. The instillation of mydriatics is avoided in animals with predisposition to glaucoma, those who are suspicious of glaucoma and lens luxation. Young puppies dilate slowly, often incompletely, and may require multiple drops. Mydriasis produced by darkening the room may permit a cursory but not a complete examination of the ocular fundus. 1% Tropicamide (Mydriacyl-Alcon Laboratories) provides mydriasis within 15 to 20 minutes in a normal eye. During this time the anterior ocular structures are examined. Additional mydriatics, 2.5% phenylephrine (Mydfrin), may be required in eyes which do not dilate well.

Corneal/conjunctival cultures and cytology
Corneo-conjunctival cultures and cytology are helpful in the diagnosis and classification of corneal and conjunctival diseases. The procedures are especially valuable in chronic, severe and non-responsive external ocular conditions. The cultures should be done before any administration of drops, since many of the drugs contain bacteriostatic agents. Topical anesthetics are used prior to the collection of cytologic material. Sterile swabs are used to collect material for culture. The swab should be moistened. The moistened swab is rubbed over the area to be cultured taking care to avoid skin, hair and other nearby structures.

Bacterial identification and disc sensitivity tests aid in the choice of antimicrobial therapy. To obtain a specimen for cytologic examination topical anesthetic is instilled 2-3 times over a few minutes and the animal's head and muzzle are held firmly by the assistant. To obtain a conjunctival scraping, the lower eyelid is everted and the ventral conjunctival surfaces are vigorously rubbed with a stainless steel or platinum spatula. The collected material is distributed onto glass slides. Ideally, conjunctiva should be scraped vigorously enough to obtain basilar cells without inducing hemorrhage. To obtain a smear of exfoliated cells, a moistened Dacron tipped applicator is rubbed along the conjunctival cul-de-sac and then rolled on glass slides. The specimens are stained with new methylene blue, Gram's, Wright's, Giemsa's, or modified Sani's methods.

Nasolacrimal system and tear production
The nasolacrimal system and preocular tear film are evaluated by considering both the secretory and excretory components.

Schirmer tear test
The precorneal tear film is essential in maintaining normal corneal health. Measurement of tear production is an important diagnostic test when deficiency of the lacrimal system is suspected. The tear-producing system is evaluated qualitatively by examination of the corneal surface for moistness and luster and quantitatively by the **Schirmer tear test**. The diagnosis of "dry eye" or keratoconjunctivitis sicca (KCS) may be missed if the Schirmer tear test is not routinely used. The Schirmer tear test measures only the aqueous aspects of tears. Currently, aqueous tear production is most commonly measured using the Schirmer tear test.
Schirmer values:
Dog: 21.9 +/ 4.0 mm wetting/minute
Cat: 20.2 +/ 4.5 mm wetting/minute
Horse: 24 +/- 5 mm wetting/minute

Excessive manipulation of the eyelids, topical anesthesia and exposure to other topical and systemic drugs (such as tranquilizers and atropine) are avoided before the test. Increased tear production because of corneal irritation during the test appears to be of little significance in the dog and the cat. The round end of the test paper is bent while still in the envelope and positioned without contamination in the lacrimal lake at the junction of the lateral and middle thirds of the lower eyelid. The animal usually closes its eyelids during the test. After one minute the paper is removed and measured on a millimeter scale on the paper envelope. The STT strip should be left in position for one minute. It is not a linear test, so if you obtain a value of 7 mm/30 seconds this does not mean it will be 14mm/min!!!! If you get an abnormal value <15mm in less than one minute the test should be repeated leaving the strip in for a full minute.

Phenol red thread (PRT) test
The Phenol Red Thread Test is a new, fast and equally accurate method to assess tear production. In the PRT tear test, the thread is 75 mm long and is impregnated with phenol red, a pH-sensitive indicator. A 3 mm indentation at the end of the thread is inserted into the inferior conjunctival sac for 15 seconds. The alkaline tears turn the pale yellow thread red. A test time of 15 seconds is required compared to the 5 minutes needed for the STT in humans or the 1 minute in dogs.

Anesthesia is not necessary for the PRT tear test because the subject has little or no sensation from the thread. It is theorized that the minimal sensation and short test time give a more accurate indicator of the volume of residual tears in the inferior conjunctival sac of the eyes. Mean length of absorption for the PRT tear test in cats is 23.0 mm ± 2.2 mm/15 seconds. The

normal range in cats for the PRT tear test is 18.4 to 27.7 mm/15 seconds. In dogs the mean length of absorption using the PRT tear test is 29.7 to 38.6 mm/15 seconds. The mean PRT absorbance value in cats (23.0 mm/15 seconds) is approximately two-thirds the mean PRT absorbance value in dogs (34.2 ± 4.4 mm/15 seconds). The mean normal Schirmer tear test value in cats (20 mm/minute) is also less than the mean normal Schirmer tear test value in dogs (22 mm/minute).

Tear drainage

The excretory component of the nasolacrimal system is evaluated by the presence or absence of medial canthal tearing; passage of fluorescein instilled onto the eye; nasolacrimal flush; catheterization of the entire system, and by dacryocystorhinography. The nasolacrimal drainage apparatus consists of two puncta and canaliculi, a poorly developed nasolacrimal sac and the nasolacrimal duct. The oval puncta are situated in the upper and lower medial eyelid margins about 1 to 2 mm in the palpebral conjunctiva. A partial to complete ring of pigment may surround the puncta and facilitates their detection.

Passage of fluorescein from the eye to the external nares is a reasonable test for patency of the nasolacrimal system. A strip of fluorescein is moistened with a few drops of sterile eyewash and touched to the upper bulbar conjunctiva. The dye usually appears at the external nares in 3 to 5 minutes. Both sides should be performed at the same time to compare passage times. Ultraviolet light enhances detection of the dye. Fluorescein passage in brachycephalic dogs and is not reliable as the dye may exit more readily into the nasopharynx. The animal's tongue and saliva should be examined with a UV light in these cases.

The nasolacrimal flush determines patency of the system and the treatment of many of its disorders. The upper punctum is cannulated with a 22-23 g blunt lacrimal needle under topical anesthesia. Tranquilization or general anesthesia is seldom necessary for the dog but often necessary for the cat. A 2 to 3 ml plastic syringe with sterile saline is used to inject the solution through the upper punctum, canaliculus, nasolacrimal sac, lower canaliculus and out the lower punctum. Once this "arc" is established, the lower punctum is compressed digitally and the solution is forced through the nasolacrimal duct and out the external nares. If the dog's head is positioned upward, the dog will swallow or gag on the solution. Excessive pressure should be avoided to minimize the danger of rupturing the N-L system above an obstruction.

The nasolacrimal system may be catheterized with a fine stiff nylon suture or very fine polyethylene tubing under general anesthesia. Catheterization of the nasolacrimal system is valuable in the diagnosis and treatment of obstruction of the nasolacrimal sac and duct. Retrograde flushing or cannulation in the dog is difficult but can be done. In the horse, retrograde nasolacrimal lavage is the method of choice. The complete nasolacrimal system can be outlined by radiopaque contrast material (dacryocystorhinography).

External Ophthalmic Stains

A. FLUORESCEIN

Examination of the cornea is incomplete without utilization of topical ophthalmic stains. **Fluorescein is used to demonstrate the presence or absence of corneal ulcers.** For topical use, fluorescein impregnated paper strips are preferred to fluorescein solution to insure sterility.

B. ROSE BENGAL: Rose Bengal is retained by the cornea and conjunctiva in keratoconjunctivitis sicca, early fungal keratitis, pigmentary keratitis, exposure keratitis, viral keratitis, and certain other corneal ulcers.

Intraocular Pressure Measurement (Tonometry)
The routine use of Schiotz tonometry is more accurate than digital tonometry, is inexpensive and is a valuable diagnostic aid. The tonometer consists of a corneal footplate, plunger, holding bracket, recording scale, and 5.5, 7.5, 10.0 and 15.0 gm weights. The low friction plunger within the corneal footplate indents the cornea proportional to intraocular pressure. The accuracy of Schiotz tonometry depends on the clinician, patient and instrument.

Measurement of IOP with the Schiotz tonometer in small animals is relatively easy. Topical anesthetic is applied to both eyes. The animal is placed in a sitting position, or lateral or dorsal recumbency. During restraint of the animal, the area around the jugular veins is avoided to prevent increased venous pressure. The eyelids are held open some distance from the lid margins, usually at the bony orbital rim. The animal's head is elevated dorsally and a few seconds are permitted for the globes to move upward. The Schiotz tonometer is held vertically and placed on the center of the cornea just long enough for the scale to be read. The conversion table provided with the instrument (for humans) is adequate for IOP estimation in the dog and cat. The tonometer must be kept clean to insure accurate results and reduce the chance of ocular contamination.

Applanation tonometers (especially the **Tonopen** type) are very accurate and easy to use. Applanation tonometers are becoming more common in practices. The Tonopen applanation tonometer has made it much easier to diagnose and treat the animal glaucomas.

IOP is 16.8 ± 4.0 mm Hg in dogs
20.2 ± 5.5 in cats
23.2 ± 6.9 in horses

Direct ophthalmoscopy
Direct ophthalmoscopy is used more frequently by practitioners than indirect ophthalmoscopy. However, both techniques have advantages that complement each other when used together. The method is termed "direct" because a condensing lens is not positioned between the ophthalmoscope and the patient's eye. The examiner has a direct optical image of the patient's eye. The fundus image is real, upright and approximately about 17 to 19 times magnified in dogs and cats. The fundus area visualized is about 10 degrees or approximately 2 disc diameters. The direct ophthalmoscope head also offers a range of lenses to enable focusing at various depths within the eye. These lenses are calibrated in diopters. A lens with a power of 1 diopter will focus light from an infinite source (parallel rays) at 1 meter. The higher the diopters, the more converging power the lens possesses. Negative diopters denote diverging lenses. When an emmetropic eye (observer) looks into an emmetropic eye (patient) with an ophthalmoscope the retina of the patient should be in focus at the 0 diopter setting. Minor lens corrections are usually needed to focus on the patient's fundus. Within the eye, a distance of 3 diopters equals 1 mm.

In performing ophthalmoscopy, the patient's body and head are minimally restrained by an assistant. The examiner holds the muzzle and/or lids with one hand and with the other hand holds the ophthalmoscope to make the necessary diopter changes. It is preferred to view the tapetal fundus several inches from the patient and then move to 1 to 2 inches from the patient's eye when the optimum focus is achieved and the animal has adapted to the restraint. The diopter setting is usually started at **"0"** and adjusted to between +3 to -3 diopters to provide the sharpest image possible. By using more positive lenses the lens can be seen at +8 to +12 diopters and the cornea at +20 diopters.

Direct ophthalmoscopy has certain limitations. Penetration of cloudy or partially crystallized media is limited. Because of magnification, there is a small field of view. Examination of the peripheral fundus is difficult. There may be difficulty in compensating for refractive errors and eye movements. Stereopsis is absent, and depth of focus is limited. The small working distance between examiner and patient may be hazardous to certain species of animals. The **PanOptic ophthalmoscope** is available and provides an intermediate level of magnification to the direct and indirect techniques.

Indirect ophthalmoscopy

Indirect ophthalmoscopy complements direct ophthalmoscopy. Because the limitations of one are the advantages of the other, use of both techniques is desirable. To perform indirect ophthalmoscopy a fairly bright light source is directed into the eye. A condensing lens is interposed between the light source and the eye. Incident light is condensed to illuminate the fundus. The reflected light then is condensed by the same lens to form a virtual, inverted, and reversed image between the lens and the light source.

The advantages of binocular indirect ophthalmoscopy are penetration of cloudy media, large field of view (hence an excellent survey instrument), examination of the peripheral fundus, ease of compensation of refractive errors and eye movements, Stereopsis, greater distance between examiner and patient, two to three simultaneous observers and the ability to readily examine the more intractable patients with less hazard to the examiner. The disadvantages include less magnification for studying particular areas, and the need for drug-induced mydriasis. Indirect ophthalmoscopy can be employed with only a light source and a lens. Several commercial indirect ophthalmoscopes are available. Regardless of the light source used, the power and type of lens used determines the ease and accuracy with which the fundus exam will be conducted.

The indirect ophthalmoscope is adjusted so the light is slightly off center of the examiner's visual field (to reduce glare). The patient's muzzle is held gently and the lens is positioned three to five cm from the cornea and the upper eyelid retracted. The lens is usually held close to the cornea initially to permit observation of the ocular fundus and then moved away from the eye until the image is maximum size. When the hand lens is interposed between the light source and the eye, the fundus is visualized. Image magnification (2X to 4X) is dependent on the dioptric power of the hand lens. The +20 lens is the most
versatile. Occasionally, an annoying light reflection occurs and is remedied by slightly tilting the hand lens. Image magnification is dependent on the dioptric power of the hand lens. The +20 D lens is the most versatile.

The canine lateral and axial magnification for the direct ophthalmoscope, and indirect lenses of 14, 20 and 30 D is 17.2 and 405; 2.6 and 9; 1.74 and 4; and 1.2 and 1.68X respectively. The feline lateral and axial magnification for the direct ophthalmoscope, and indirect lenses of 14, 20 and 30 D is 19.5 and 508; 2.9 and 11.3; 1.95 and 5.08; and 1.26 and 2.11 X respectively. The horse fundus is minified with 20 and 30D lenses, so only the 14D lens, or better yet, a 5D lens should be used.

Ocular ultrasonograpy

Ultrasonography (as in a ship's sonar system) has become increasingly useful in the diagnosis of intraocular disease in the past few years. High frequency sound waves are directed through the eye. A portion of these sound waves bounce off the tissue interfaces. These echoes are amplified and projected onto an oscilloscope. Echoes from the corneal surfaces, the anterior and posterior lens surfaces, the retina, and any abnormal intraocular material will

project an image which aids intraocular diagnosis. This is especially useful when dense corneal opacity or mature cataract obscures the view of the fundus.

NASOLACRIMAL SYSTEM

Keratoconjunctivitis SICCA (dry eye): This is an aqueous **deficiency** of the precorneal tear film (PTF) causing progressive inflammatory changes of the cornea and conjunctiva. Clinical signs are mucopurulent discharge, conjunctival hyperemia, corneal pigmentation and a dull lusterless cornea that varies with the amount of tear production . Breeds at Risk include English Bulldog, West Highland White Terrier, hasa Apso, Pug, Cocker Spaniel, Pekingese, Yorkshire Terrier, Shih Tzu, Miniature Schnauzer, Boston terrier. Burmese cats and many more. Atropine and edotolac topically, and systemic sulfadiazine, salicylazosulfapyridine (Azulfidine®), Tribrissin®(small dogs at increased risk) can cause KCS. Other causes of KCS include canine distemper virus, conjunctivitis scarring the lacrimal ducts, neurogenic damage to the lacrimal gland, immune related disorder of the lacrimal gland (suspected in up to 80% of dog cases), hypothyroidism, Hyperadrenocorticism, Diabetes mellitus, Demodectic mange, SLE, RA, and removal of the superficial gland of the nictitating membrane.

S**chirmer tear test:**
Normal 15-25 mm/minute
Suspicious = 8-10 mm/minute
Low = <8 mm/minute
Rose Bengal stain: epithelium of conjunctiva and cornea will remain red if devitalized or necrotic.
The mean PRT absorbance value in cats (23.0 mm/15 seconds) is approximately two-thirds the mean PRT absorbance value in dogs (34.2 ± 4.4 mm/15 seconds).

KCS Treatment:

1. Medical: Always attempt 1-2 months of medical treatment because the problem may be transient. Owner compliance may be difficult. Goals are to remove pain and mainctain vision. Replace tears with topical Hypotears (CIBA Vision) Tears Naturale (Alcon), Lacrilube (Allergen), Duratears (Alcon) or Lacriserts (Merck). Stimulate production of tears with t**opical 0.2% Cyclosporine (CSA)**, (**OPTIMMUNE**): **DRUG OF CHOICE** F**OR KCS.** It is reported to increase tear production in 80% of cases which had not responded to other modes of therapy. CSA inhibits T lymphocyte induced apoptosis of lacrimal gland acinar cells. It also interferes with prolactin, and has anti-inflammatory activity. It greatly reduces pigmentary/ inflammatory keratitis. The dose is 1/8-1/4 inch strip twice daily but may take up to 12 weeks before increasing tear production. 1%-2% Cyclosporine compounded at a licensed pharmacy may be used in cases that are not responsive to *Optimmune*. No systemic toxic signs have been noted from topical application. Local irritation has been reported. Topical 0.02% Tacrolimus and 1% Pimecrolimus may have the same effect and may work in place of CSA. Topical 2% Pilocarpine at 2 drops per 20 pounds well mixed in the food twice a day per OS can help ICS in some dogs. . Increase dose slowly until tears produced or signs of toxicity occur. Signs of oral pilocarpine intoxication: salivation, emesis, diarrhea, tachycardia, heart block, pulmonary edema. Give in small amount of food (snack) due to bitter taste. Control Bacterial Flora with topical broad spectrum antibiotic BID (triple antibiotic ointment) and control inflammation with topical corticosteroids, may combine with topical antibiotic (TriOptic-S, triple antibiotic with hydrocortisone); USE ONLY IF NO CORNEAL ULCERATION!!

HYPERTROPHY AND PROLAPSE NICTITANS GLAND (CHERRY EYE)

This is primarily seen in young dogs, less than 2 years, in Beagles, American Cocker Spaniels, Bulldogs, and Pekingese. The gland protrudes above free border of the TE, becomes inflamed

and enlarged and may have epiphora, a mucoid discharge and conjunctival inflammation. Treatment is topical corticosteroids and surgical replacement of the gland. Excision of the gland may predispose to keratoconjunctivitis sicca (KCS)

ULCERATIVE KERATITIS: The most important disease of the cornea.
Ulcerative keratitis (corneal ulceration) means that the corneal epithelium and possibly varying amounts of underlying corneal stroma are missing. In simple traumatic corneal injuries in which a small amount of epithelium is absent, healing is rapid. Normal corneal epithelium is a very effective barrier against invading bacteria. If the ulcer becomes infected or the epithelium is unable to attach to the underlying stroma, healing is delayed.

In chronic or infected ulcers, proteases and collagenases digest protein and collagen of the stroma and may greatly speed the progression of an ulcer to a descemetocele, rupture of the cornea, and then to iris prolapse (within 12-48 hours in some cases).

Corneal dissolution and liquefaction under the influence of proteases is often referred to as "melting". Ulcers in which proteases are active have a grayish-gelatinous liquefied appearance around the ulcer margin which must be distinguished from corneal edema.

Ulcerative keratitis is the **most serious** ocular disease for veterinarians. **Regardless of the initial cause, all ulcers have the potential to progress to endophthalmitis if not treated.**

CLASSIFICATION/CHARACTERIZATION OF CORNEAL ULCERS
Superficial ulcerations or abrasions should heal rapidly if they do not get infected. They can be traumatic in origin. It has been shown It has been shown that normal horse corneal epithelium migrates at 0.6 mm per day. We guess that the dog and cat are similar or faster. The following types of ulcers will be covered in detail: recurrent superficial corneal erosions; deep stromal ulcers; fungal keratitis; descemetoceles; perforating ulcers (iris prolapse); and corneal lacerations (superficial and full-thickness).

There are multiple causes of corneal ulcers. Corneal ulcers can result from mechanical causes such as traumatic abrasion; corneal or eyelid foreign bodies; and eyelid anomalies (entropion, distichia/districhiasis, ectopic cilia, and trichiasis). Infectious etiologies also cause corneal ulcers. Infectious organisms can be bacterial, fungal, or viral. Culture and sensitivity are important diagnostic tools to use with infectious ulcers.

Keratoconjunctivitis sicca (KCS or "dry eye") can result in corneal ulceration. This is especially true with cases of acute onset KCS, in which corneal ulceration can occur rapidly and progress quickly. Ulcers are less common with chronic KCS.

The majority of animals with corneal ulcers present with pain as evidenced by blepharospasm. Corneal sensation is one of the major protective factors that the eye exhibits. Corneal sensory nerves are located mostly in the superficial cornea, and the nerves lose their myelination as they cross from the periphery into the center of the cornea. An "axon reflex" is thought to exist in the cornea such that when corneal touch and pain receptors are stimulated, miosis of the pupil, hyperemia, and increased protein levels in the aqueous humor occur. The axon reflex is responsible for the clinical signs of anterior uveitis observed with painful corneal conditions. These results appear to be mediated by prostaglandins, histamine, acetylcholine, and possibly substance P. Other clinical signs seen commonly with corneal ulceration include epiphora, photophobia, and corneal edema, causing a change in transparency.

Culture and sensitivity should be performed routinely when an ulcer is infected or "complicated." Not all small animal ulcers need to be cultured the first time you see the patient. However, if the corneal ulcer appears to be "melting" or if the ulcer has not responded to proper treatment, these ulcers should be cultured.

Schirmer tear test and/or phenol red thread test should be performed on all canine patients presenting with corneal ulceration. A large percentage of dogs with dry eye with initially present with corneal ulceration. An eye with an ulcerated cornea should have excessively high tear production resulting in epiphora. If the Schirmer tear test value is in the normal range or similar to the normal fellow eye, then KCS should be suspected.

Cytology can be performed using topical anesthetic. Remember to collect culture samples and perform Schirmer tear tests prior to applying topical anesthetic, as the anesthetics can interfere with interpretation of results.

ALL ulcers should be stained with fluorescein and sometimes with Rose Bengal. Fluorescein stain (which is hydrophilic) will adhere to exposed stroma, but will not stain epithelium or Descemet's membrane. Rose Bengal is used to evaluate mucin tear layer defects, and devitalized epithelium that is still attached but not healthy.

THERAPY OF CORNEAL ULCERS:

There are multiple steps in the treatment of a corneal ulcer.

The deeper the ulcer, the more aggressive is the medical and likelihood of surgical therapy.

The etiology is determine if possible, and it is then removed or **eliminated**. This means evaluating
the eyelids and eyelashes, tear production, corneal culture, and corneal cytology.

Broad-spectrum antibiotics are usually administered; culture and sensitivity tests can a guide to selection in recurring, non-healing, or infected ulcers.

Prevention of collagen breakdown and ulcer progression is also important in ulcer therapy. Collagenases and proteases are derived from leukocytes in the tears and wound. This can be powerful in the destruction of corneal stroma. There are several drugs that can be used to help inhibit protease activity. Serum contains an alpha-2 macroglobulin with anticollagenase activity. Blood is drawn from the patient or an animal of the same species, spun down, and serum drawn off and stored in the refrigerator in a dropper bottle or serum tube for up to 14days. It should not be stored at room temperature, but at the time of therapy, the dose can be warmed to room temp immediately before administration. Serum is non-toxic, and should be used as many times a day as possible. EDTA (0.17%) can be given several times a day as well. Acetylcysteine (5-10%) **is** used topically for its collagenase and protease inhibiting properties. Acetylcysteine is unstable at room temperature, so the solution must be kept refrigerated. Frequency of treatment is decreased from every 1 to 2 hours for the first few days to 3 or 4 times daily for the next 7-10 days.

Secondary Anterior Uveitis is treated with topical mydriatic/cycloplegics such as atropine BID.

Treatment of deep corneal ulceration and **Descemetocele** most often require surgery to provide corneal support. Coverage with one of the various kinds of conjunctival flaps and other biological tissue should be maintained for 10-28 days.

REFRACTORY SUPERFICIAL CORNEAL EROSIONS

Synonyms include Boxer ulcer, indolent ulcer, persistent ulcer, rodent ulcer, refractory epithelial erosion, recurrent corneal erosion syndrome. Middle to old age groups are most commonly affected, and there may be an increased incidence in females. Breed predilection has been demonstrated in the Boxer, Corgi, Pekingese, and Lhasa Apso, but refractory ulcers have been documented in more than 24 breeds of dog. History, signalment, and ophthalmic findings are all important in the diagnosis of refractory corneal laceration.

Refractory corneal ulcers in the dog are usually primary. However, they can also be seen secondary to eyelash or eyelid abnormalities, corneal edema, infection, or tear film abnormalities. It is important to rule out conditions that can secondarily cause indolent ulceration in order to successfully treat the syndrome. The specific pathogenesis of refractory ulcers is still not known. Normally the corneal epithelium attaches to the underlying stroma via hemidesmosomes in the basal epithelial cell membrane. Some animals with refractory corneal ulcers have been shown to have fewer hemidesmosomes as well as abnormalities in the epithelial basement membrane. Histologically, there are focal areas of epithelial separation with splitting of the basement membrane and edema (in and between the basal cells) with accumulation of a basement membrane-like material.

Variable pain (manifested by tearing, blepharospasm, and photophobia) is present, and there is no history of traumatic injury. On ophthalmic examination, a superficial corneal ulceration with an overlying lip of unattached epithelium around the edge of the erosion is evident. The use of fluorescein staining will illustrate the ulcer bed as well as reveal the degree of unattached epithelium as the underlying stroma will take up stain.

Debridement of unattached and loosely attached epithelium is essential. Topical anesthetic and dry cotton-tipped applicator are used to remove abnormal epithelium.

Superficial keratectomy and grid keratotomy has revolutionized the treatment of indolent ulcers, especially in terms of decreasing recurrences. This requires more specialized skill, equipment and magnification. Most small animals require only topical anesthesia, or rarely light sedation. A 25- 22 gauge needle is used to make cross hatches ("tic tac toe") through the ulcer bed with the scratches approximately 1-2 mm apart into adjacent normal epithelium and anterior stroma. This technique has been shown to increase the healing rate of refractory ulcers. Chemical removal of the epithelium can also be accomplished with dilute topical povidone iodine or phenol. This is only recommended if all other therapy has failed.

Topical broad spectrum antibiotic solutions 4 times per day, topical cycloplegics (1% atropine) as needed to relieve pupillary spasm, topical hyperosmotic agents (5% NaCl) to decrease corneal edema; 1-4
times daily, serum: 2-8 hours, and a bandage soft contact lens can aid therapy.

ANTERIOR UVEITIS

Infectious uveitis is common to cats (FIP, FIV, FELV, herpes, toxoplasmosis, cryptococcus), can be secondary to corneal or scleral disease, and can be immune-mediated as in lens-induced uveitis (LIU). It may be caused by systemic diseases and trauma. Clinical signs include enophthalmia, prolapsed nictitans, hyperemia of conjunctiva, corneal edema, keratic precipitates, aqueous flare in anterior chamber, hyphema or hypopyon, iris is swollen. Miosis, iris color change and rubeosis iridis and synechiae with hypotony. Treatment of anterior uveitis includes topical atropine, steroids, NSAIDs and antibiotics. Complications of uveitis

include persistence, leukomas, iris bombe, endothelial degeneration, cataracts, phthisis and glaucoma.

CATARACTS

Strictly defined, a cataract is any opacity of the lens or its capsule. It may be congenital, inherited, or caused by disease, toxicity, trauma, or age. Many purebred dogs are predisposed to developing cataracts. Cataracts and nuclear sclerosis are both associated with advancing age. Nuclear sclerosis is a normal lenticular alteration in most dogs over 6 years. Nuclear sclerosis is not a true cataract, and there is no pathologic alteration of the lens fiber pattern.
Another common cause of cataracts in dogs is diabetes mellitus. Initially, diabetic cataracts begin as equatorial cortical vacuoles but rapidly (weeks to months) progress to form complete or resorbing cataracts. Advanced cataracts produce vision loss. In comparison, focal incomplete cataracts have varying degrees of blindness.

If the cataracts are mature, clues such as careful assessment of the history, the pupillary light reflex and dazzle response, in addition to ocular ultrasonography and ERG, may be required to rule out concurrent retinal disease.
Terms frequently used to describe cataract severity include: **immature or incomplete, mature or complete, and hypermature or resorbing cataracts.** By convention, the degree of completeness of a cataract is related to the amount or percentage of tapetal reflection that it blocks. Hypermature cataracts are often associated with a deep anterior chamber, wrinkled anterior lens capsule, and signs of uveitis. An incipient cataract is synonymous with an early cataract. An intumescent cataract describes a lens that has "swollen" and enlarged due to an imbibition of fluid. The lens can actually swell enough to alter aqueous outflow dynamics and increase intraocular pressure (IOP).

PHACOEMULSIFICATION

Phacoemulsification is the extracapsular removal of a cataract using ultrasonic vibrations generated by a special piezoelectric hand piece. The lens is simultaneously shattered, the anterior chamber maintained by irrigation, and the fragments of lens removed by aspiration. The major advantage of phacoemulsification is that it is performed using a small corneal incision. The major disadvantages of phacoemulsification are cost. CATARACT **S**URGERY IS BEING DONE EARLIER NOW.

LENS LUXATION AND SUBLUXATION

Luxation is when the lens is totally free of zonular attachments. Subluxation is when the lens is only partially freed from its zonular attachments and remains in the patellar fossa of the vitreous face. This is common in terriers. Management of lens luxation depends on whether the animal is painful in the affected eye or not, and whether the animal has the ability to see out of this eye or not.

CANINE AND FELINE GLAUCOMAS

Aqueous humor is produced in the ciliary body by active secretion and ultrafiltration of plasma. The enzyme carbonic anhydrase participates in the energy-dependent secretory phase of aqueous production. Most of the aqueous humor flows from the posterior chamber, through the pupil, to the anterior chamber, and exits at the iridocorneal angle into the intrascleral venous plexus. A small percentage of the outflow in dogs and cats (uveoscleral or nonconventional) also exits through the iris, ciliary body, choroid, and sclera. The balance between formation and drainage of aqueous humor maintains intraocular pressure (IOP) within a normal range of approximately 15 to 25 mm Hg.

By definition, glaucoma is increased IOP with associated visual deficits. In most cases in dogs and cats, glaucoma is caused by obstruction or stenosis of the aqueous humor outflow pathways. It remains a challenge to the veterinarian to detect the early subtle disturbances of glaucoma and to effectively treat this condition. Delayed or inadequate therapy can lead to irreversible blindness and a painful, cosmetically unacceptable eye.

All ocular tissues are eventually affected by the elevated IOP. The presence, individually or as a group, of a "red eye," corneal edema, mydriasis, blepharospasm, blindness, and buphthalmos can be explained by the increased IOP. If the IOP cannot be reduced, an overall increase in the size of the globe may result (buphthalmos). This change may occur more rapidly in young dogs and cats. Ruptures of the cornea's inner limiting (Descemet's) membrane may accompany the elevated corneal tension and buphthalmos to produce multiple, linear corneal striae. Persistent corneal endothelial damage can result in corneal edema. Buphthalmos causes increased tension on the lens zonules. Zonular disinsertion results in lens subluxation or luxation.

Pupillary light reflexes may be normal, slow, or absent in early glaucoma, depending on the functional status of the iris sphincter muscle, retina, and optic nerve. Acute elevation of IOP (greater than 45 mm Hg) causes paralysis of the iris sphincter and dilator muscles. Prolonged or recurrent elevations of IOP lead to degeneration of the retina and optic nerve, with excavation or cupping of the optic nerve head.

Glaucoma is divided into primary (including congenital) and secondary categories. The iridocorneal angle may be open, narrow, or closed in either type. Abnormal development of the iridocorneal angle (goniodysgenesis) has been noted in some breeds. Evaluation of the iridocorneal angle is performed with gonioscopy in the dog but may be performed with focal illumination in the cat.

Primary glaucoma in dogs is a breed-related, hereditary condition. Predisposition to primary open-angle glaucoma in the Persian and Siamese cat breeds has also been noted, but in the author's experience, domestic short-hairs are more often affected. In both dogs and cats, affected animals may present with only one eye involved, but the risk is very high for development of glaucoma in the other eye.

Secondary glaucoma is more commonly encountered than primary glaucoma in dogs and cats. The elevated IOP results from other disease processes within the eye. The glaucoma may be open or closed angle, and in some instances is associated with pupillary block. The condition tends to be unilateral without an inherited basis.

The presentation of a patient with a painful, red eye requires that glaucoma be ruled out among the possible diagnoses of conjunctivitis, uveitis, or keratitis. Pain manifested as depression, anorexia, rubbing at the eye, and squinting is common. Congestion of episcleral vessels, diffuse corneal edema, a fixed and dilated pupil, and blindness will occur as the IOP increases. The onset of clinical signs in cats is often insidious, as cats are less likely to demonstrate the acute intense corneal edema and episcleral congestion exhibited in dogs. Signs of chronic glaucoma are dramatic. They include combinations of the early signs with buphthalmos, lagophthalmos, exposure keratitis, luxated lens, corneal striae, optic nerve atrophy with cupping, and retinal atrophy.

IOP must be accurately measured to diagnose glaucoma. The normal canine and feline IOP is 15 to 25 mm Hg. An IOP greater than 30 mm Hg is considered pathologic and diagnostic for this condition. It is possible to crudely evaluate IOP digitally if the IOP is very high or low, but

this is not satisfactory to evaluate clinical response to therapy. The Schiotz's indentation tonometer allows the practitioner to diagnose and evaluate treatment in small animals with glaucoma. The human Schiotz table is accurate for the dog. The Tonopen applanation tonometer has made it much easier to diagnose and treat the animal glaucoma.

The objectives of therapy are to maintain vision and eliminate pain by (1) increasing aqueous outflow, (2) decreasing aqueous production, and (3) preventing or delaying glaucoma in the other eye. Primary glaucoma may be more difficult to control than secondary glaucoma because it is eventually bilateral, and blindness is a possible sequela despite therapy. I nevertheless recommend prophylactic therapy for the unaffected eye in animals afflicted with unilateral primary glaucoma. In secondary glaucoma, the inciting cause is identified and either removed or suppressed. Topical corticosteroids may be indicated to diminish inflammation when nonseptic anterior uveitis is also present.

Medical therapy is the treatment of choice in animals with a history of acute primary or secondary glaucoma. Treatment should be instituted to reduce the IOP as soon as possible to alleviate pain and preserve vision. Animals presented with a history and clinical signs of chronic glaucoma should be considered for medical and surgical therapy. The iridocorneal angle gradually closes in most types of glaucoma and the initially effective treatment becomes inadequate. Surgery is the only option available when vision continues to diminish in spite of maximum medical therapy.

Multiple drug therapy to decrease IOP by reducing production of aqueous humor and diminishing the resistance to aqueous humor outflow is the most effective approach. Treatment of the ocularly normotensive eye in a purebreed dog with apparently unilateral glaucoma can delay the onset of overt ocular hypertension in the second eye a median of 30 months. Betaxolol and demecarium were each effective at delaying onset of glaucoma in dogs when administered topically. Carbonic-anhydrase inhibitors reduce ciliary-body production of aqueous humor independent of diuresis. These drugs can cause metabolic acidosis, and the dosage should be carefully adjusted to minimize side effects, which include panting, nausea, and vomiting. Non-carbonic anhydrase-inhibiting diuretics do *not* significantly reduce IOP! Topical parasympathomimetic drugs act primarily to cause ciliary muscle contraction, increasing the outflow of aqueous humor. This action is independent of their effect on the iris sphincter muscle. Parasympathomimetics are contraindicated in glaucoma associated with anterior uveitis. They should be used with caution in glaucoma associated with anterior lens luxations. Sympathomimetic drugs reduce IOP by increasing production of aqueous humor and increasing outflow. These drugs are most effective in reducing IOP when combined with parasympathomimetics. ß-adrenergic antagonists decrease production of aqueous humor, but the specific mechanism of action is not known. The ocular hypotensive effects are additive to those of carbonic-anhydrase inhibitors and parasympathomimetics.

Oral and intravenous hyperosmotic agents lower IOP rapidly by osmotically reducing the volume of the vitreous. They are used in the emergency treatment of acute glaucoma but are ineffective or impractical for long-term or maintenance therapy. Intravitreal glutamate levels are elevated in canine glaucoma. Glutamate is extremely toxic to the retinal ganglion cells. It overstimulates them. Glutamate excitotoxicity is mediated by intraneuronal calcium influx. Intraneuronal homeostatic imbalance induces apoptosis and cell death. The use of glutamate receptor antagonists and calcium channel blocking drugs to protect the retina and optic nerve is being studied.

Surgical procedures are divided into those that increase aqueous humor outflow and those that decrease aqueous humor production. Surgery should be considered when the IOP cannot be controlled medically, especially when vision is still present. Anteriorly luxated lenses should be removed in functioning eyes to relieve pupillary block and prevent corneal damage due to the lens touching the corneal endothelium. Cyclocryotherapy has been found to be effective in decreasing production of aqueous humor by the transcleral freezing of the ciliary body with nitrous oxide. This may require repeated applications for optimal IOP control. The YAG laser is preferred over nitrous oxide by the author to cause ciliary body necrosis (cyclophotocoagulation). The eye is less irritated postoperatively and the IOP stays low for longer periods of time. Only 6% of dogs with cyclophotocoagulation will still be visual at one year after installation. Gonioimplant sare available to passively shunt aqueous humor to the subconjunctival space. They tend to fail by fibrosing shut in dogs. Only 18% of dogs with gonioimplants will still be visual at one year after installation. Enucleation or evisceration with prosthetic silicone implants is indicated when vision is lost in uncontrolled glaucoma. The source of pain is removed, and no further medication is necessary. The cosmetic appearance of the prosthetic implant is sometimes preferred to that of enucleation. Prosthetic implants should not be used when glaucoma is or may be associated with intraocular infection or neoplasia. The use of intraocular silicone prosthesis (ISP) implants have been used successfully in cats with buphthalmos and absolute glaucoma.

Pharmacologic Agents for Medical Treatment of Glaucoma

Carbonic-anhydrase inhibitors (oral)
a. Acetazolamide (Diamox, Lederle): 10 to 25 mg/kg divided 2 to 3 times daily
b. Dichlorphenamide: 10 to 15 mg/kg divided 2 to 3 times daily
c. Methazolamide (Neptazane, Lederle): 5 mg/kg divided 2 to 3 times daily

Parasympathomimetics (topical)
a. 1 to 2% pilocarpine every 6 hours
b. 0.125 to 0.25% demecarium bromide: 1 to 2 times per day

Sympathomimetics (topical)
a. 0.1 to 0.5 % dipivalyl epinephrine (Propine, Allergan): 2 to 3 times per day

Beta-adrenergic antagonists (topical)
a. 0.5% timolol maleate (Timoptic, Merck): 2 to 3 times per day
Timolol may precipitate or aggravate feline asthma due to systemic absorption and bronchoconstriction.
b. betaxolol (0.5%) (Betoptic, Alcon, Ft Worth, TX): 3 times per day.

Hyperosmotics (parenteral)
a. 20 % mannitol: 1 to 2 mg/kg IV; repeat in 6 hours if necessary
b. 50 % glycerol: 1 to 2 mg/kg PO; repeat in 8 hours if necessary

Topical Carbonic-anhydrase inhibitors
a. Dorzolamide (2% Trusopt, Merck): 1 drop TID
b. Brinzolamide (1%), Azopt, Alcon, Ft Worth, TX: 1 drop TID

Topical Prostaglandins
a. Latanaprost 0.005%, Pharmacia, 1 drop BID
b. Travaprost, Alcon, BID

Calcium Channel Blockers
a. Norvasc, amlodipine. 0.625 mg/10 lbs PO SID