Proceedings of the European Veterinary Conference Voorjaarsdagen

Amsterdam, the Netherlands
Apr. 5 - 7, 2012

Next Meeting:
Apr. 18 – 20, 2013 - Amsterdam, the Netherlands

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INTRODUCTION TO LCP
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The advent of internal fixation with bone plates and screws represents a tremendous advancement in the methods of fracture fixation. During this era, the emphasis was on anatomic reconstruction and rigid internal fixation. Although these principles are applicable to a great number of fractures, not all can be treated in this manner. In fact, the disruption of the soft tissue envelope required to perform an anatomic reconstruction of a highly comminuted fracture and ensuing disruption of healing potential can contribute to nonunion. Gradually, a shift in philosophy has occurred, and now the emphasis is on preservation of fracture biology, with spatial realignment of the major bone segments; this technique is known as biological osteosynthesis.

LCP Implant Design
The LCP features a uniquely designed combination hole that accepts standard bone screws as well as locking screws, and allows the plate to be used as a conventional plate (compression), a locking plate (internal fixator principle) or as a combination of both principles. The ends of the plate have a “slipper toe” design that facilitates tunneling the bone plate under soft tissues in a minimally invasive manner. The underside of the plate has scalloped undercutts similar to the LCDCP, which creates a uniform area moment of inertia to minimize stress concentration at the plate hole, as well as mitigate disruption of extraosseous blood supply. The locking screws are designed to tolerate the shear loads resulting from angle stable fixation. The core (diameter of the screw between threads) is larger than standard bone screws, with a smaller thread profile. In addition, the pitch of the screw is smaller, and matches the pitch of the thread on the head of the screw. The smaller pitch minimizes the distance the screw travels prior to the threads of the screw head engaging the bone plate; this diminishes plate compression onto the bone surface. Finally, the locked screw has a conical, double-lead thread design that facilitates alignment with the threaded plate hole, and ensures that the screw thread engages the plate thread with no more than one-half turn.

Figure 1: Locking plate combi-hole with locked screw (center) and standard screw (right).

Figure 2: Locking screw (left) and standard cortex screw (right).

Biomechanics
The design goals of the LCP are to increase the stiffness of the construct while maintaining the biological fracture environment. There are distinct differences between the design of conventional bone plates and locking plates. Conventional plates depend on friction at the plate-bone and screw-bone interfaces to maintain fracture fixation. These implants fail due to cortical screw toggling which leads to screw loosening and loss of plate-bone fixation. Therefore, the system depends on each screw’s resistance to loosening and pull-out strength.

The LCP is a fixed angle construct that does not rely on friction at the plate-bone and screw-bone interfaces. Rather, the system relies on friction at the threaded screw-plate interface. Once the screw is locked into the plate, the fixed, angle stable design converts shear stress into compressive stress at the screw-bone interface. The load applied to the limb is redirected such that it is perpendicular to the screw axis. The redirection of stress explains why locking screws are designed with smaller threads, since they do not generate compression between the plate and the bone. However, they have a larger core diameter which ensures greater bending and shear strength and dissipates the load over a larger area of bone. In addition, the screws feature the new StarDrive head that withstands 65 percent greater insertion torque than conventional hexagonal drivers. The StarDrive is...
also self-retaining, thus the screw stays on the screw driver without a holding device.

In conventional plating, it is critical to contour the plate accurately to optimize plate-bone frictional forces, and mitigate malreduction during plate application. In contrast, the locked plate frictional forces, and mitigate malreduction during plate application. In contrast, the locked plate does not need to be precisely contoured. During application of a locked plate, the fracture must first be reduced and then conventional screws are placed if used at all. Next, the locked screws are added for the fixed construct stability. Unicortical locked screws have two points of fixation, one in the bone and the other in the bone plate, and therefore they resist axial load to failure better than standard unicortical cortex screws in bone, which may be prone to loosening.

Several technical details remain to be elucidated in the application of the LCP to veterinary patients including, the ideal number of locked screws on either side of the fracture, the number of unicortical versus bicortical screws necessary to achieve adequate stability, indications for and magnitude of plate contouring required, the effects of combining conventional screws and locked screws in the same construct, indications for double plating or adding additional implants, such as intramedullary pins, and the potential advantageous biological effects on fracture healing when LCPs are placed in a minimally invasive fashion.

References:
- Gautier E, Sommer C: Guidelines for the clinical application of the LCP. Injury 34:S-B31-S-B42, 2003
- Wagner M: General principles for the clinical use of the LCP. Injury 34:S-B31-S-B42, 2003

Clinical Performance
To date, there are no randomized clinical trials in human or animals comparing the LCP plate to conventional plates (DCP and LC-DCP) in patients with similar fractures. The plates have been studied and compared in vitro (human and animal) and in case series’ and these studies form the basis for LCP principles and indications. The reported indications for LCP application include patients with poor quality bone (ostoporosis, osteomyelitis), complex periarticular fracture, particularly when contouring may be difficult in the metaphyseal area, fracture configurations in which it is difficult to achieve the minimal number of conventional screw cortices, periprosthetic fractures, cases in which only bridge plating is possible or indicated, nonunions from failed fixations in which cortex or cancellous screw are stripped or have backed-out, and polytrauma cases in which the fractures cannot be anatomically reconstructed. In vitro studies in bone models do show that locked screw constructs fail at higher loads than cortex screws and their advantage is magnified in osteoporotic bone.

**Figure 3:** “Toggle” mode of screw loosening in conventional plating.

**Figure 4:** Locked stable design of the LCP resists axial load.
The need for implants that could be used with minimally invasive surgical technique following the philosophy of ‘biological osteosynthesis’ was a driving force behind the development of locking plates. In addition, plates that had improved mechanics and less impact on bone blood supply were considered to be desirable. It should be appreciated that the Zespol external plate fixator developed in Poland in the 1980’s was a concept that was ahead of its time.

In principle, the Zespol was the same as modern locking plates, because it had screw heads that could be locked into plate. These screws were applied perpendicular to the plate, and were “fixed angle”. The Zespol did not rely on friction between the plate and the bone for stability. Indeed, the plate was situated just outside the skin, similar to an external fixator.

There are currently several different types of locking plates available that they may be useful for fracture fixation in small animals, including the No Contact Plate and the Locking Compression Plate (LCP). The LCP (Synthes) have combination locking and compression holes, or so called “Combi hole”. This allows the plate to be applied with either fixed angle locking screws in the threaded part of the Combi hole, or standard cortical screws that are placed in the dynamic compression unit (DCU) part of the Combi hole.

Application of the LCP with entirely locking screws results in fixed angle construct.

Used it this way, there is not any compression of the plate to the bone, or between the fracture fragments. The most important use of this device comes in non-reducible shaft fractures when the plate is acting as a bridge plate. Once the locking head screws (LHS) engage the plate, no further tightening of the screw is possible. Therefore the implant locks the bone fragment in their relative position regardless of the degree of reduction. Accurate contouring of the plate to the bone is not essential. Furthermore, by locking the screws to the plate, the risk of loss of reduction due to screw toggling and fracture collapse is reduced.

Since the plate can sit off the bone, and locking of screws prevents compression of the periosteum by the underside of the plate, then blood supply to the bone may be improved. In case of reducible fractures, once the metaphyseal fragment has been fixed with locking screws, the fracture can be compressed using standard screws in the DCU portion of the Combi hole.

**Pitfalls with LCP**

1. Need to be able to reduce the fracture, or obtain correct alignment of the bone, prior to application of the LCP.
2. The plate needs to positioned so that screws are centered over the intramedullary space. If there is axial malalignment of the plate and bone, then some of the screws near the end of the plate will not have adequate bone purchase, and thus fail.
3. Cross threading of screws can cause permanent locking of screws especially with titanium implants. While cross threading of locking screws does not necessarily compromise the stability of the fixation, implant removal may involve cutting of the plate.

The threaded portion of the combi hole in the straight LCP only allows for placement of screws that are perpendicular to the plate. This can be problematic in the metaphyseal region. Development of ‘anatomically’ contoured plates has overcome this problem, for example the distal tibial plate for humans.

**Clinical application**

First clinical application of the LCP plate in Veterinary Medicine has been reported in 2005 by Schwandt in a young dog affected by radio-ulnar and tibia fracture.

Recently Haaland reported a retrospective study of a case series of 47 dogs affected by fracture treated by LCP plate. Most of the fractures (60%) were comminuted. The LCP plate had been mostly used as an internal fixator or in a “plate and rod” fashion. Fracture healing had been achieved within 5-8 weeks. Complication rate was around 11%: five implants failures and one osteomyelitis. All implants failures were related to technical mistakes during surgical procedures.

In our experience we treated with the LCP system a case series of 25 patients affected by fracture (n 22) and non-union (n 3). The LCP plate had been used as neutral plate (n 12) as compression plate (n 7) and buttress (n 6).
healing was achieved in all patients within 6-11 weeks. Functional recovery was scored excellent in 17 patients, fair in 6, sufficient in 2. Complication reported were: implant failure (n 1), LHS pull-out (n 1) and carpal joint arthrosisinovitis.

Figure 1: Tibia comminuted fracture pre-operative X-Ray (A, B), temporary plate fixation with push and pull device: radiographic (C) and intra-operative view (D). The fracture has been treated with a 14 holes LCP plate in a MIPO fashion

Figure 2: Same patient of figure 1 Immediate post-operative radiographs (A, B), 1 month follow-up (C), 2 months follow-up (D) showing bone healing

References:
- Schwandt CS Montavon PM: Locking compression plate fixation of radial and tibial fracture in a young dog. VCOT 18:194-198, 2005
In addition the length of the plate itself, the number and relative position of screws which need to be inserted, as well as the type of the screws (standard cortical screw, locking head screws, mono or bi-cortical screw) remain under debate. Thus a lot of additional decisions have to be taken by the surgeon when planning and performing plate osteosynthesis with an LCP.

The three following main factors influence the stability of the fixation and the loading condition of the bone-plate construct:

- the length of the plate
- the number of the screws
- the position of the screws

**Lenght of the plate**

Utilizing the newer minimally invasive techniques or indirect reduction, subcutaneous or submuscular plate insertion and splinting as a stabilization concept, the plate length can be chosen to be very long without the need of additional soft tissue section and devascularization.

Theoretically the plate can be equal the whole length of the broken bone. But at least the length of the plate can be determined by means of the two factors:

- the plate span width
- the screw density

Plate span width is defined as the quotient of the plate length and overall fracture length. Empirically we find that the plate length should be two or three times higher than the overall fracture length in comminuted fractures and eight to ten times higher in simple fractures. The second factor is the plate screw density which is the quotient formed by the number of screws inserted and the total numbers of plate holes. Empirically are recommended values below 0.5, indicating that less than half of the plate holes are occupied by screws.

**Plate span ratio:** in comminuted fracture the plate length should be 2 to 3 times higher than the overall fracture length.
In simple fracture this ratio should be raised to a value of 8 to 10.
From E Gautier “Bridging Plate”, AO Dialogue 24:27, 2009

**Number of Screws**

From the mechanical point of view, two screws (bicortical or monocortical) on each main fragment, is the minimum number of screws needed to keep the bone plate construct stable. Unfortunately, such a construction will fail if one screw breaks due to overload or when the interface between bone cortex and screws threatened due to bone resorption with subsequent screw loosening. Thus, for safety reasons a minimum of three screws are recommended in each the proximal and the distal main fragment.

Adjusting the plate screw density to a maximum value of 0.5 the plate length will not be chosen below 12 hole plate for treatment of a diaphyseal fracture. I would like to underline that the “plate screw density” is a concept coming from Human medicine and could be adapted to Veterinary medicine accordingly with small animal bone anatomy.

**Plate screw density:** the plate screw density should be kept below a value of 0.5, indicating that less than half of the plate holes are occupied by screws.

**Position of the screws**

When using the concept of interfragmentary compression in a simple fracture pattern, load sharing condition between plate and bone are present and in this way the
screws in the middle of the plate can be inserted as close as possible to the fracture.

When using the concept of splinting in a simple fracture pattern, the middle plate segment is bent over a short distance enhancing the local strain within the implant. To avoid high implant strain, the innermost screws should be spread apart, which increases the length of the plate segment bending, thus decreasing the implant strain. This protect the plate against fatigue failure.

In the case of comminuted diaphyseal fractures, the plate spans over the fracture like a non gliding splint. A longer distance between the two screws adjacent to the fracture is dictated on the one hand, by the fracture pattern itself, and on the other hand, by mechanical reasons, by the spreading out of the innermost screws thus decreasing implant loading - but only when there is a distance limitation on the opposite cortex. Without distance limitation the deformation of each plate segment in the middle depends on the acting bending moment. Each plate segment is deformed according to the external loading condition - thus, the overall deformation is much higher and the implant strain can become high and critical, as in the situation where is bridging a small gap with a short plate segment between the two innermost screws.

**Take home messages for plating**

- Splinting (bridging) is a sound stabilization principle for fixation of comminuted fractures
- Splinting can be used for stabilization of simple fractures - when, on the one hand, a long plate is used to improve the lever arm of each screw, decreasing the screw pullout and, on the other hand, the two innermost screws are spread apart leaving at least two or three plate holes unoccupied at the fracture site to decrease plate loading.
- Interfragmentary compression remains a sound stabilization tool for fixation of simple fractures under the prerequisite of careful soft tissue section and handling
- The use of locking head screws advantageous from the biological point of view. Such an internal fixator does not compress the periosteaum and thus reduces the amount of avascularity of the bone cortex adjacent to the plate. In addition callus formation is possible in the gap between the plate and the bone cortex.

References:
- Gautier E, Sommer C: Guidelines for the clinical application of the LCP. Injury 34:S-B63-S-B76, 2003
- Wagner M: General principles for the clinical use of the LCP. Injury 34: S-B31- S-B42, 2003

**EVALUATING AND MONITORING DOGS WITH OSTEOARTHRITIS IN PRACTICE**

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**History**
The patient’s history should be documented in chronological order. Important general questions relate to the use of the animal (working/pet), diet, vaccinations, etc. The animal’s level of exercise, episodes of previous illness, or previous musculoskeletal disease may be relevant to the current situation.

There is increasing interest in the use of clinical metrology instruments (owner questionnaires) for musculoskeletal disease. Such instruments do serve to formalise collection of some basic data and also to “stage” the condition as seen by the owner. The scores generated by such instruments can be useful to track clinical progression and response to treatment. We have recently published an owner-administered clinical metrology instrument and this has been partially validated in canine elbow osteoarthritis (Hercock and others 2009). You are welcome to download the instrument and the accompanying user guide for use in individual patients in your clinic at:

**LOAD:** http://www.liv.ac.uk/sath/services/LOAD.pdf (copyright ‘University of Liverpool’)

**User guide:** http://www.liv.ac.uk/sath/services/NOTES.pdf
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Other clinical outcomes measures have also been partly validated (Brown and others 2008, Hielm-Bjorkman and others 2003). Criterion validity for LOAD (comparison to force platform) was recently demonstrated to be superior to these two other instruments (Walton and others 2011).

Examination of the patient

Examination of the patient with osteoarthritis should start with a general physical examination. Then one should pay particular attention to areas of inflammation, contusion, deformity, malfunction/disuse, atrophy/hypertrophy, increased/decreased range of motion, disarticulation or fracture. There are definite sections to a logical lameness examination:

- Observation
- Palpation: muscle mass, pain, swelling, heat
- Manipulation: joints, muscles, tendons, spine
- Neurological function

Locomotion can be divided into phases. These are “swing” and “stance” and are detailed below: Observation with the dog at rest allows assessment of conformation, deformity, distribution of bodyweight - uneven distribution points to discomfort. Ask yourself the following questions:

- Is weight distributed forward?
- Is weight distributed backwards?
- Is weight distribution asymmetric?
- Is weight shifting?

Videos can be useful tools to assess lameness and function. Smart phones provide an opportunity for owners to readily capture their animals function in the home environment or field situation. High-speed videos can be useful in the clinic and are now very affordable. These allow for detailed analysis of function. Increasingly, specialist clinics are investing in objective measures of gait such as force platforms, pressure mats and motion capture systems.

References and further reading:


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**Imaging Joints: Making the Most of Radiographs and Other Modalities**

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**Introduction**

The clinician is currently faced with many choices when imaging synovial joints. Whilst this an enviable position to be in, there are issues around making the correct choice(s) of imaging modality to use on specific cases. Whilst previously the X-ray room would be the automatic choice, we are now sometimes faced with choices including X-ray, ultrasound, scintigraphy, computed tomography (CT) and magnetic resonance imaging (MRI).

The choice of imaging modality may be driven by various factors. The signalment and history of the patient along with the clinical examination findings may dictate the clinician’s choice. The anatomical area to be examined will also have a bearing the selection. For instance, ultrasound is proving to be very useful around the shoulder joint but may be less useful for the elbow joint where it is the bony structures in which one is often interested. Availability of equipment and expertise is also a major factor in many clinics. Even where equipment is available, for some modalities such as ultrasound and...
MRI, the operator and radiologist need to have the required level of expertise.

The shoulder
The shoulder can be a complex joint to image. In the immature dog when one is suspicious of OCD, it is relatively straightforward; plain and contrast radiographs will usually suffice (Figure 1). In the adult animal, one is often interested in the soft tissue support structures: the collateral ligaments, capsule and tendons. Plain and contrast radiographs are often unhelpful in this instance. Ultrasound can provide useful information on the biceps tendon of origin, as well as the infraspinatus, supraspinatus tendons. In the right hands, it can also provide information on the subscapularis tendon and the medial glenohumeral ligament. MRI is commonly used to evaluate the human shoulder and it is likely to be useful for the canine shoulder too (Figure 2).

The elbow
The elbow joint is a common seat of lameness in immature and mature dogs. Because of their size and location, “primary” lesions such as fragmentation of the medial coronoid process (FCP) are often not visible. The clinician may often rely on osteophytosis at typical locations to signal intra-articular pathology. 97% of immature dogs with elbow lameness have FCP and, in the absence of other pathology, the appearance of osteophyte in an immature elbow joint can give rise to a high suspicion of FCP. Osteophytes may be very subtle, for weeks or months, and the clinician needs to be aware of the typical primary sites for osteophyte development (Figure 3). Beyond radiography, CT seems to be the most useful imaging modality. It can be used to detect FCP and also incomplete ossification of the humeral condyle (IOHC).

Figure 3: Mild osteophytosis often appears on the proximal border of the anconeal process - a flexed mediolateral projection is required to see this.

The carpus and tarsus
Radiography is still the primary modality for imaging the carpus. For gross injuries this is usually sufficient but for more subtle injuries, radiography can be insensitive. CT provides excellent detail and both carpi or tarsi can be scanned together. There are also some reports of MRI of the canine carpus but the resolution of MR can be an issue for evaluating some of the very small ligaments of the canine carpus.

The hip
Radiography is still the workhorse for imaging canine hips although dynamic studies can provide more useful information regarding laxity of the hip. Hip dysplasia and osteoarthritis are the main pathologies of the canine hip joint and as such it is often the conformation of acetabulum and femoral head that is of concern, along with their relationship to each other. The standard hip-extended ventrodorsal view of the pelvis and hips has been used for decades to assess hip conformation but it has significant limitations. One such limitation is the underestimation of hip laxity because of capsular tightening during extension of the hip joint. Dynamic studies such as the PennHIP® have contributed much to the understanding of the pathogenesis of hip dysplasia.

The stifle
Radiography provides a sensitive method with which to detect arthropathy of the stifle joint. Effusion and osteophytosis appear rapidly in the stifle joint. Compression of the infrapatellar fat-pad and distension of the caudal joint capsule are indicators of effusion. However, plain radiography is not very specific for what pathology might be affecting the stifle. Of course cranial cruciate ligament (CCL) disease is very common but it should not be assumed that this is the case. Further investigation of the stifle can include ultrasonography; a skilled operator can see the CCL and the menisci. However, the clinician is likely to use other diagnostic tests such as arthrocentesis and arthroscopy. MRI can be useful in selected cases, e.g. when meniscal injury is suspected (Figure 4).
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Figure 4: MMRI of the canine stifle joint showing the medial meniscus

References:

MEDICAL MANAGEMENT OF OSTEOARTHRITIS: WHAT’S THE EVIDENCE?
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Introduction
Treatment of osteoarthritis (OA) falls into two categories: conservative and surgical management. This discussion will concentrate on the former. Management of OA is most often conservative and multimodal. Figure 1 summarises the relationship between various treatment modalities and disease severity. The vast majority of cases are managed with NSAIDs combined with nutritional, weight and exercise management.

Weight management
A recent development is the use of weight loss pharmaceuticals and there are now two such licensed products for use in dogs. Mitratapide is a microsomal triglyceride transfer protein inhibitor. Dirlotapide is also a microsomal triglyceride transfer protein inhibitor but, in addition, it appears to have appetite suppression activities in dogs. Both drugs are designed to be used in the context of a weight management programme and although there are no published trials of their use in dogs with osteoarthritis in particular, it is likely that these agents could be useful in the management of bodyweight in arthritic patients.

Medical management of osteoarthritic pain
Drugs for the treatment of OA can be classified as symptom- or structure-modifying agents 152-154. The former category includes agents that are designed to treat the pain associated with OA and the latter category includes agents that are designed to retard, stop or reverse the pathological changes in articular tissues. There are many symptom-modifying agents for use in canine OA (e.g. NSAIDs) and a restricted number available for feline OA. The criteria for a drug to be validated as a structure-modifying agent are stringent and include demonstrating efficacy of retarding or stopping cartilage degeneration.
Further large, multi-centre studies on these newer agents are required to formulate evidence-based recommendations for long-term treatment of canine OA based on a balance between efficacy and safety. NSAIDs are always likely to be problematic in cats and their use in this species should be cautious.

Other analgesics for osteoarthritis

Amantadine
Amantadine is a N-methyl d-aspartate (NMDA) receptor antagonist. The analgesic effects of amantadine in dogs with chronic OA pain was investigated in a study comparing dogs treated with meloxicam alone compared to dogs treated with meloxicam and amantadine (Lascelles, Gaynor et al. 2008). The results indicated some benefits of amantadine treatment over and above treatment with meloxicam alone, as measured by veterinarian lameness score and owner questionnaires, after 42 days of treatment. To date, this represents the only study on the use of amantadine for treatment of canine OA.

Acetaminophen (paracetamol)
Acetaminophen is a well-established centrally-acting analgesic for the treatment of OA in human patients but is much less widely used in animals. It should not be prescribed to cats under any circumstances because it is extremely toxic in this species but it can be used safely in dogs given at the correct dose (10-15mg/kg every 8 or 12 hours). The mechanism of action of acetaminophen is still unclear.

Mesenchymal stem cell therapies for OA
Mesenchymal stem cells (MSCs) are multipotent stem cells that can differentiate into a variety of cell types including chondrocytes and osteoblasts. In the past decade or so, there has been a surge of interest in the use of MSCs to repair damaged connective tissues. At the current time, techniques offered commercially for OA in dogs involve harvest of autologous adipose tissue and extraction of the stromal cells. The stromal cells in suspension are then injected intra-articularly after extraction. A proportion of these stromal cells will be mesenchymal stem cells. The mode of action of such procedures is unclear at the current time and may involve soluble factors released by the stromal cells or actions of the cells themselves.
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Sequential samples from the same joint may be required in some instances, such as monitoring treatment of infective or immune-mediated arthritis.

Technique for arthrocentesis
Arthrocentesis in the dog is a simple procedure and can be performed under general anaesthesia or heavy sedation. The needle insertion sites are illustrated in Figure 2. The hair over the site should be clipped and the site aseptically prepared and if the operator wishes to palpate the insertion site, sterile gloves should be worn. If the operator is familiar with the insertion site landmarks, a “no touch” technique can be used as an alternative. The needle used should be of sufficient diameter to facilitate the flow of viscous synovial fluid (i.e. 0.9mm-0.67mm [20-23 gauge]) and be long enough to reach the joint cavity through the soft tissues. The length of the needle varies with the joint being sampled with proximal joints requiring needles 25 - 63 mm (1 – 2.5”) long and distal joints requiring 16mm - 25mm (5/8 – 1”) needles. The fluid is placed into appropriate sample tubes, usually EDTA and a blood culture bottle and squash smears should be made. Rapid air drying is useful to reduce cell shrinkage artefact and can be achieved by directing a hair dryer on warm setting at the back of the slide held at a distance of about 15cm.

Evaluation of synovial fluid

Cytology
Qualitative and quantitative cytological examination of synovial fluid is the most useful test to assist the clinician in classifying the disease process within the joint. Examination of a smear is useful but the sensitivity, specificity and reliability of qualitative assessment is poor. Whenever possible, a total and differential cell count should be performed. Appropriate dilution of the sample should facilitate this and the clinician should use a laboratory that is prepared to perform these analyses. There is significant overlap in the cytological changes between different articular disease processes, and cytological results need to be interpreted in the light of patient history, clinical signs and results of other diagnostic modalities.

It is generally possible to distinguish between “inflammatory” and “non-inflammatory” joint disease on sufficient fluid from these: in this situation, sample other larger joints (shoulder, elbow, hip, stifle) as these will often also have inflammatory changes.

References:
the basis of total cell count and the percentage of neutrophils, but there is considerable overlap between the inflammatory conditions (infective arthritis and immune mediated disease) and the results must be interpreted in the light of other clinical information. As a general principle, if multiple joints show evidence of inflammatory change in a symmetric pattern with a predominance of neutrophils, the likely diagnosis is immune mediated polyarthritis. Infective arthritis of multiple joints in dogs and cats is rare, particularly in adult animals, and is less likely to be symmetrical in distribution. The discussion below will briefly describe the cytological features of the various arthropathies. Further summaries of the features of these diseases are detailed at the end of this chapter.

Non-Inflammatory arthropathies

Osteoarthritis

Cytologically, there is significant overlap with normal joints, as cell numbers are usually normal or very slightly raised (up to 5 x 10⁹/L) with less than 2-4% neutrophils. Thus the majority of cells observed on cytology are large mononuclear cells (synovial lining cells and macrophages 60-90% of cells) and lymphocytes (3-30% of cells) with <5% neutrophils. Synovial lining cells are round cells with a single round nucleus and a moderate amount of basophilic cytoplasm.

Recent joint injury/haemorrhage

Joint injury or haemorrhage will cause a haemarthrosis, reflected as large numbers of red cells. In acute haemarthrosis the differential nucleated cell count may be similar to that of peripheral blood and platelets may be present. With more long-standing haemorrhage there are increased numbers of neutrophils and macrophages and platelets are absent (these disintegrate rapidly within the fluid). Erythrophagocytosis and/or haemosiderin or haematoxidin crystals in macrophages provide evidence of pre-existing haemorrhage into the joint; these features are not seen with iatrogenic blood contamination.

Infective arthropathies

These can be divided into two main groups: immune-mediated and infective arthropathies. Both result in a moderate to marked increase in numbers of neutrophils with variable numbers of large mononuclear cells.

Immune mediated polyarthritis (IMPA)

The synovial fluid cytology for all types of IMPA varies between joints and between patients. Typically, the white cell count is 20-90 x 10⁹/L with 20-90% neutrophils (non-degenerate) and varying proportions of lymphocytes and large mononuclear cells.

Infective arthritis

Infective arthritis generally causes a large increase in neutrophils and these become the predominant cell type. In acute infection, neutrophil counts are usually within the range of 95-98%. With more chronic disease (weeks), neutrophil counts may drop to the region of 70-95%. Neutrophils may appear degenerate and bacteria may be noted on cytological inspection.
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What is osteoarthritis?
Osteoarthritis is a disease of the whole joint – the articular cartilage, bone and synovium (Innes 2011). The relationship between the pathology in each of these tissues is poorly understood but is the articular cartilage that has received most attention in terms of research. Although joint biomechanics undoubtedly have an important role in disease initiation and progression, biochemical changes occur in all joint tissues and contribute to joint failure. OA is a heterogeneous disease and assessment of the disorder is difficult. The poor correlation between radiographic and clinical data highlights this difficulty. The typical example of this, in dogs, is the dysplastic hip with secondary OA - severe radiographic changes may be present in a clinically silent joint. Expression of different facets of the disease seems to vary between individuals and even between different joints in the same individual. In small animal medicine this is exemplified by differences in osteophyte expression which clearly do not tally with the clinical picture. One current model of OA (fig 1) attempts to incorporate the heterogeneous nature of OA and how various contributing factors may interact. It is thus helpful to think of OA as a disease process rather than a disease entity.

Basic structure of articular cartilage
The architect of cartilage is the chondrocyte which produces the extracellular matrix. The matrix is composed of glycosaminoglycans (hyaluronan and proteoglycan) and collagens (mainly type II). The collagen forms a dense network that retains the proteoglycan. The proteoglycan is highly charged and attracts water into the tissue. Thus cartilage is 75% water. In normal cartilage there is a very slow turnover of collagens but the proteoglycan is constantly being renewed. The proteoglycans are aggregated into large molecules (“aggrecan”) with a protein core and many side chains of keratan sulphate and chondroitin sulphate. This core is in turn bound to hyaluronan chains with each chain containing many proteoglycan molecules. Aggrecan and water provide the compressive stiffness to the tissue whereas collagen provides the tensile strength.

Disease mechanisms of OA
One major target in OA therapy is effective disease-modifying agents which might slow down the degradation of articular cartilage. With this in mind, the following section of text aims to highlight some of the advances that have been made in the understanding of the pathogenesis of OA. In gaining a handle on the cellular and intercellular processes at work, we can understand how drugs might be designed to interrupt or slow down the destructive processes and/or stimulate repair mechanisms.

The main tissue affected in OA is the cartilage but the subchondral bone and synovium are also affected and indeed may be important in terms of disease progression. In the wake of improved understanding of the disease will undoubtedly come therapeutic agents.

Figure 1: Current model of OA (Dieppe PA and Lohmander LS [2005] Lancet 365:965-73)

Figure 2: Components of articular cartilage. Aggrecan molecules attract water in to the tissue and create a swelling pressure. The type II collagen network resists this pressure.

Proceedings of the European Veterinary Conference - Voorjaarsdagen, 2012 - Amsterdam, Netherlands
Orthopaedics

Chapter 2

Companion Animal Programme

Pathological changes of OA
The morphological changes seen in OA include:

- cartilage loss, especially in areas of increased load,
- subchondral bone remodelling (loss of bone initially followed by sclerosis),
- marginal osteophytosis,
- variable synovial inflammation.

The biochemical changes in the cartilage include:

- loss of proteoglycan,
- upregulation in the degradative and synthetic activities of chondrocytes,
- disruption of the collagen network increase in water content

These changes reduce the elasticity of the cartilage leading to fibrillation and fissuring of the cartilage with eventual loss of tissue. If this continues eburnation of subchondral bone may result.

Intercellular events in OA
The chondrocytes themselves are upregulated and the rates of proteoglycan synthesis and degradation are increased with the overall balance towards matrix depletion. It seems likely that the activity of the chondrocytes is increased following the binding of cytokines to the cell surface. Cytokines are cellular messengers produced locally in the tissues in response to various biological stimuli such as inflammation. It is proposed that the cytokines responsible for stimulating cartilage degradation in OA are interleukins 1 and 6 (IL-1 and IL-6), tumour necrosis factor-α (TNF-α) and oncostatin M.

Binding of these cytokines to the chondrocyte stimulates the production of enzymes that have been shown to be capable of degrading all the components of the cartilage matrix. Synovial cells also release natural inhibitors of these cytokines such as IL-1 receptor antagonist (IL-1ra). There are also cytokines which stimulate synthesis of matrix and likely candidates for this include the insulin-like growth factors I and II (IGF-I and IGF-II) and transforming growth factor-β (TGFβ). Recently published studies have demonstrated that the normal availability of IGF-I and IGF-II may be limited in OA (Fernihough and others 2003)

Cartilage degradation
Catabolic cytokines can stimulate the chondrocyte to produce and release degradative enzymes. The enzymes studied in most detail in this respect are the matrix metalloproteinases (MMPs) and the family of endopeptidases, the ADAMTS-4 and -5 (A disintegrin and metalloproteinase with a thrombospondin motif). ADAMTS-4 and -5 are also known as aggrecanases. MMPs and aggrecanases can cleave the protein core of aggrecan so as to release the majority of the molecule from the matrix (Innes and others 2005). Under normal circumstances the chondrocyte also produces a natural inhibitor of these enzymes known as tissue inhibitor of metalloproteinase (TIMP). TIMP production appears to be decreased in OA.

Summary
Like many body tissues, cartilage exists in equilibrium between anabolism and catabolism. In OA, the balance is shifted towards breakdown. Clearly there are possibilities for intervention in these mechanisms.

References:

Figure 3: Degradation of the interglobular domain of aggrecan by MMPs or aggrecanases; evidence is accumulating the aggrecanase is the major activity in arthritic joints.

Proceedings of the European Veterinary Conference - Voorjaarsdagen, 2012 - Amsterdam, Netherlands
Introduction
Total hip replacement (THR) has been performed in dogs since the 1970s. Early systems were two-component cemented systems with little accommodation for the variance in patient size and conformation. THR is now a routine procedure in orthopaedic referral practice. Modular canine cemented and cementless systems are available from several manufacturers. The success rate of the procedure is reported as good (85-95%). Recent innovations include “nano” and “micro” systems that can be used in small (~7kg and upwards) dogs and larger domestic cats. In recent years, systems for elbow replacement have appeared and, in some cases, disappeared. A canine knee replacement is also commercially available.

Total hip replacement
Indications
Total hip replacement is indicated for the intractably painful hip dogs. Hip dysplasia and osteoarthritis are the most common indications although chronic or recurrent luxation, malunion of fractures of the femoral head or neck, and Legge-Perthes disease are also indications. Dogs should have exhausted conservative measures in terms of weight control, exercise management and medical treatments (NSAIDs and analgesia) to the point where quality of life is insufficient. The risk-benefit ratio of THR must be contemplated. If there is a complication with the procedure, necessitating implant removal, the function of the hip is severely compromised; this risk is in the region of 5-10% and thus owners must be made fully aware of the possible complications as well as the potential benefits.

Contraindications for THR include:
- Persistent infection of another body system (e.g. severe skin, ear or dental disease)
- Debilitating musculoskeletal disease of the ipsilateral limb
- Dogs less than 10-12 months of age (in the author’s opinion)
- Dogs too small for the implant components
- (Debilitating musculoskeletal disease of other limbs)

Technique
A THR can be placed through a craniolateral approach to the hip. The femoral head is luxated and the head and neck cut using a template. The acetabulum is reamed and the acetabular component placed (cemented or non-cemented). The femur is then drilled and cancellous bone removed. The femoral component is placed and the appropriate femoral head applied to the femoral neck. The hip is reduced and the capsule sutured for stability.

Patient monitoring
In human joint arthroplasty, many countries operate joint arthroplasty registries such that patients and outcome are monitored prospectively. Such a registry has now been established for canine hip replacement and is supported by the British Veterinary Orthopaedic Association and hosted at University of Liverpool (www.caninehipreplacement.org). Surgeons from any country can submit cases for this ethically-approved programme. Owners of operated animals are asked for annual feedback via an online outcomes assessment tool (in English).

Total elbow replacement
Dogs with severe elbow lameness which is not controlled adequately with medical management, may be candidates for total elbow replacement (TER). This is a relatively new procedure and the first report of successful TER in clinical canine patients was in 2003 (Conzemius, Aper et al. 2003). This “Iowa State” total elbow replacement (IS-TER) involved placement of a high density polyethylene combined radioulnar component and a cobalt-chrome humeral component and the initial success rate was reported as 16/20 dogs. The author performed 16 IS-TERs with some success but there were short and longer term complications. The instrumentation for IS-TER evolved over time and this solved and created issues. Post-operative complications included luxation, infection, ulnar fracture. Longer term complications included polyethylene wear and heterotopic ossification. The IS-TER has essentially been discontinued, or at least is no longer supported by training programmes. Biomedtrix now produce the TATE TER system although peer-reviewed publications on the system are lacking (Dejardin and Guillou 2011). Over the last five years, the author has...
worked with a UK bioengineer and a surgical colleague at Liverpool (Rob Pettitt) to develop the Sirius TER system (Figure 1). Phase I trials are underway at the current time with encouraging results. In addition, Arthrex Vet Systems are launching a unicompartmental elbow system (CUE). The CUE Arthroplasty System is designed to provide a surgical treatment option for medial compartment disease (MCD) of the canine elbow. Again, peer-reviewed publications have not appeared at the current time. TER is a rapidly developing field and, hopefully, we are edging closer to clinically-acceptable results from such prostheses. However, short, medium and long-term evaluation will be required as well as ongoing refinement.

References and further reading:

Figure 1: The ‘Sirius’ total elbow joint replacement

Total knee replacement
A canine total knee replacement has emerged as a spin-out from orthopaedic research using dogs as a model for human knee replacement. The system is now marketed by Biomedtrix and there are some published reports on small numbers of patients (Liska and Doyle 2009). The indications are similar to other joints: intractable pain, unresponsive to medical management. One of the common issues with knees is that many candidates have had previous knee surgery (e.g. cruciate ligament or meniscal surgery) and this is likely to increase the incidence of post-operative infection.