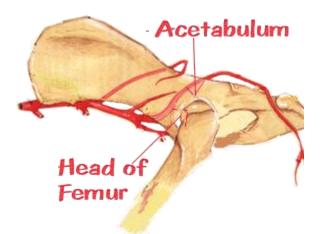
<u>Hínd leg Bone-Joint-Ligament Lameness</u>



Hip dysplasia:

Hip Joint Basics

- This malady is, unfortunately, a too-common occurrence. It happens primarily to large breed dogs and is caused by complex genetics (polygenic) and environmental factors.
- It is usually a disease of young dogs (<2yrs) but can develop in older individuals, as well.
- The <u>figure</u> above, left, illustrates a normal hip joint (the "coxofemoral joint"). The uppermost aspect of the thigh bone (the femur) consists of an angled appendage (the femoral neck & head) at whose end there is a hemispheric component (the head) that normally fits into a hollowed/concave hemispheric space known as the acetabulum on the pelvis (hip bone).
- This arrangement (the "articulation" of the femoral head within the acetabulum) comprises the coxofemoral joint. It is stabilized by fibrous ligaments within and around the joint.
- When the leg moves, the femoral head is supported by and rotates within the acetabulum. Both the head and the acetabulum are coated with a slippery cartilage and these are further surrounded with a thick, lubricating fluid which, together, provides an

environment that prevents friction from leading to damage of structures and maximizes functionality. Ultimately, this prevents joint inflammation (arthritis) and pain.

 In order for this joint to function optimally, the femoral head must articulate at a precise angle appropriate for maximal weight bearing; misalignment of any of the participating structures resulting from inappropriate force, or angle of articulation as well as malformation of the head or the acetabulum will lead to degenerative joint disease, and, potentially life-long pain.

• Definition and Causes

- Hip dysplasia is a laxity of the hip joint that develops between birth and skeletal maturity that can result in the abnormal alignment of articular surfaces. Hip dysplasia often leads to painful arthritis. Causes are multifactorial.
 - <u>Heredity</u>: Many genetic determinants may, collectively, be responsible; the mechanism(s) which lead to clinical the manifestation of signs is complex and incompletely understood.
 - <u>Diet</u>: The influence of diet on skeletal and joint anomalies has been discussed in considerable detail on this web site (see <u>Large Breed Puppy</u> <u>Diet</u>). Basically, rapid growth leads to large body mass. This results in greater stress forces on immature bone and growth plate cartilage. Consequently, microfractures within growth plates and exacerbation of (genetically predisposed) joint structure malalignments ensue.
 - <u>Excessive High-Impact Exercise</u>: This may similarly affect joint and growth plate integrity.
- Diagnosis
 - Clínical History:
 - Young-Dog Syndrome: A young (4-12 months) large-breed canine with sudden decrease in activity or onset of hind limb lameness may be associated with some loss of hip muscle mass.
 - Older-Dog Syndrome: An older (>12 months) large-breed canine with insidious loss of rear limb muscle mass, shifting weight while

standing (listing between left and right rear limbs), especially after exercise.

- Palpation of Coxofemoral Joint
 - Barden Method
 - Ortolani Sign: This is a complex series of moving leg through various ranges of motion while concurrently palpating the hip joint for signs of subluxation/laxity.
- Radíologícal Sígns:
 - Precise positioning for radiological evaluation of the hip joint usually requires heavy sedation or anesthesia.
 - With standard positioning for the evaluation of the hip joint, there are accepted abnormal radiological signs that are consistent with the diagnosis of hip dysplasia. Evaluation of the shape and congruity of the femoral head with the acetabulum, evidence for abnormal wear of articular surfaces, as well as the presence of degenerative changes are some of the parameters examined.

Treatment Options

- Conservative (Medical/Physical Therapy)
 - Goals:
 - Promote increased muscle mass. (Note that atrophy of thigh and pelvic muscles due to disuse [pain?] of hind limb(s) actually exacerbates the stress on the hip joint and spreads an additional support burden on the remaining limbs)
 - Correct Obesity
 - Exercise:
 - Exercise is required to prevent muscle atrophy and may help in the management of obesity.
 - Diet Modification (lower caloric density diet or fewer "treats". for example)
 - Examples: On Mondays, Wednesdays and Fridays, go for leash walks, swimming (minimal stress to joint while building muscle mass) and mild

retrieving...promote the "trot" rather than speed running (which is high impact).

- Such a regimen will make the dog sore....initially, during the first few weeks.
- Non-steroidal anti-inflammatory medication (check with your veterinarian) could be given on Tuesdays and Thursdays (the "off" days) if needed.
- Obesity Management:
 - Exercise regimen, as described above
 - Medical and laboratory evaluation to rule out underlying causes (e.g. hypothyroidism) of obesity.
- Surgical Options
 - Triple Pelvic Osteotomy (TPO):
 - This is a major surgical procedure in which the angle of the pelvis is changed to better accommodate the head of the femur within the acetabulum.
 - The TPO is ideally performed early (6-12 months of age) before the development of arthritis and degenerative joint disease.
 - RULES OF THUMB: This procedure should NOT be done on any animal with degenerative joint disease or who is greater than 18 months of age.
 - Total Hip Arthroplasty (THA):
 - This procedure involves replacing the entire hip joint with an artificial hip joint (a similar procedure is performed in people.
 - Reserved for clinically significant (PAINFUL!) arthritis and degenerative joint disease.
 - NOT appropriate if there is significant muscle mass atrophy or if there is infection *anywhere* (e.g. dental disease/gingivitis, urinary tract, skin).
 - Femoral Head and Neck Osteotomy (FHO):
 - Involves excision of the articular components of the femur (the head and

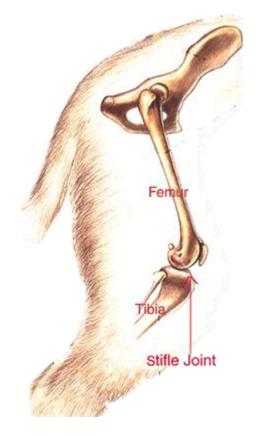
the "neck"[....the portion of bone that connects to the head of the femur]) and thus bypassing the normal hip joint by promoting the formation of a "false joint".

 This surgery promotes the generation of a fibrous capsule which, along with normal thigh and pelvic muscles, stabilizes and supports the limb and the pelvis. There is no articular surface, *per se*; hence, this is a "false joint".

Cranial Cruciate Ligament Avulsion

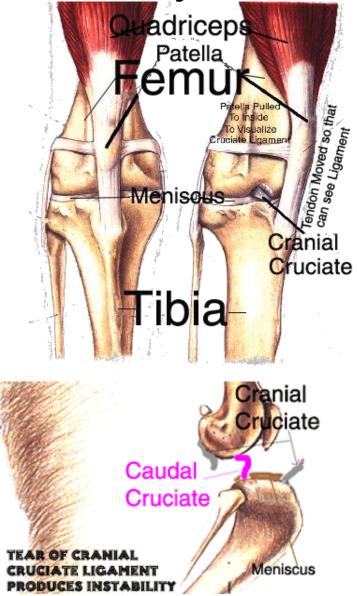
Stifle (Knee) Joint Basics

• The Stifle Joint's *Range of Motion* is illustrated in this <u>figure</u>



• The Stifle joint is stabilized by numerous ligaments, as well as the *gastrocnemius* and *quadriceps*

tendons. Two small but extremely important ligmentous structures are found *within* the joint and each is specifically connected to strategic portions of bone thus providing strength, stability and integrity. They are the Cranial (Anterior) and Caudal (Posterior) Cruciate ligaments. Together, they maintain the strict normal front-to-back alignment of the two major bones (the femur, or thigh bone, and the tibia, or shin bone). The following <u>illustrations</u> reveal the nature of this relationship under normal conditions and following rupture of the cranial cruciate ligament.



- During flexion of the joint, the cranial and caudal cruciate ligaments actually twist on each other; this prevents abnormal "inward" rotation of the tibia (relative to the femur).
- During extension, the cranial and caudal cruciate ligaments untwist; now stabilization and protection of the joint from abnormal rotation relies mainly upon the external (collateral) fibrous ligaments, each one connecting the femur to the tibia. There are two collateral ligaments: one on the inside (medial-facing the inner thigh) and one on the outside (lateral-the side easily seen in a standing dog) aspects of the stifle joint.
- A fibrocartilagenous pad...the *meniscus*...sits in the joint, tightly associated with the tibia and guides the normal articular motion; the meniscus is composed of lateral (toward the outside) and medial (towards the inside) components. The lateral meniscus is less rigidly attached than the medial meniscus.
- In addition to the smooth articular cartilage and viscous joint fluid which provide lubrication and minimize friction, the joint also contains a small amount of fat.

Causes of Cranial Cruciate Avulsion

- Excessive Forces: Most often, the result of running, frequent turning and shifting of direction, jumping; these type of activities result in severe forwardcompressive or rotational forces on the tibia, respectively.
 - When rupture follows excessive internal rotation of the tibia, a portion of the femur rubs over the meniscus, causing tears or actually rolling/folding the meniscus onto itself
- Degenerative changes: anatomical/ conformational anomalies result in inappropriate alignment of joint structures, inflammation then degenerative changes, including the weakening of internal ligamentous supports.
- Any inflammatory joint condition that results in degenerative joint disease weakens the ligamentous infrastructure and predisposes to rupture.

- Partial Ruptures: Sometimes only a portion of the cranial cruciate ligament is ruptured --i.e. some fibers are torn. In most, but not all instances, there is pain, swelling and inflammation and...Ultimately, complete rupture ensues.
- Often in animals undiagnosed partial or complete rupture is followed by the deposition of fibrous tissue around the joint. This is Nature's way of trying to provide some stability. These animals are never normal and intermittently are non-weight bearing on the affected limb.

Clinical Signs

- *In acute rupture*, the animal is usually non-weight bearing, the stifle is swollen and painful.
- When there is chronic or old, partial tears, the joint capsule is thickened (increased fibrous tissue), the animal is painful or intermittently painful and lame or intermittently lame.

Diagnosis

- Palpation:
 - Cranial Drawer Sign: With complete rupture, one is able to move the tibia forward (with respect to the femur)
 - Tibia Compression Test: The application of force upwards, from the hock (ankle), parallel to the tibia, results in the forward displacement of the tibia (as in the Cranial Drawer Sign).
 - Increased Joint Laxity: Abnormal internal rotation of the tibia.
 - Other: Note...some animals with partial tears or longstanding cruciate ligament avulsion may not demonstrate any of the above-mentioned abnormal responses. Sedation will likely be necessary to feel these changes.
- Imaging..
 - Radiographs (X-rays): May visualize malalignment of tibia with respect to femur. May see signs of joint swelling, including displacement of joint fat. With time, may see evidence of degenerative joint disease.

- Magnetic Imaging (MRI): May actually be able to assess ligament integrity.
- *Joint Fluid Analysis*: Is useful in eliminating other causes of stifle joint disease.
- Arthroscopy: Provides direct visualization of all internal joint structures.
- Exploratory Arthrotomy: Provides the most direct method for assessing and concurrently repairing damage to the joint.

Treatment..

- Aggressive medical management: In very small dogs with acute injury, it is occasionally possible to effect resolution via:
 - Strict Exercise Restriction and Joint Immobilization: At least 4-6 weeks of kennel confinement.
 - **Physical Therapy: Required for several months.**
- Surgery:
 - Most Animals: require surgical intervention. There are over 100 different surgical procedures and the appropriate procedure is tailored to the unique needs of each patient.
 - **Post-Operative Exercise Restriction:** At least 4-6 weeks of restricted activity, such as short leash walks to eliminate, etc.
 - **Physical Therapy:** As described for medical management scenario.
 - **Prognosis:** Is usually good with surgery (80%-90% return to normal function) but the success rate is less than 80% if there is concurrent damage to the meniscus.

Canine Eosinophilic Panosteitis

Definition

 Panosteitis is a disease in which there is inflammatory and degenerative changes of the medullary (the medulla= the middle compartment of long (e.g. leg) bones. • The medulla of bones is a soft tissue space surrounded by hard (cortical) bone; it contains marrow (fat and blood cell precursors).

Causes

• Viral: causes are presently unclear but there is considerable evidence that there is an underlying viral etiology. Some speculate that *Canine Distemper* infection or vaccination may each play an important role.

Clinical Signs

- Large/ Giant Breed Dogs: especially German Shepherds and Rottweillers.
- Young : Dogs are usually young--(5-12 months) but disease has been reported in animals as young as 2 months and as old as 7yrs
- Male Dogs: are more commonly affected than females.
- History includes:
 - variable inappetance or anorexia
 - Sudden Onset of Lameness without history of trauma
 - Wax-Wane/Shifting: lameness is variable and may shift to different limbs
 - Front Limbs more likely affected
 - Not Affected By Rest or Exercise
 - Physical Exam
 - Intermittent Fever
 - Pain on palpation of the bone (50% of patients)
 - Tonsillitis: some animals have this concurrently

Diagnosis

- Blood Evaluation may show elevated white blood cell counts--especially with elevated eosinophils (a type of white cell)---or results may be completely normal.
- X-Rays: radiographs of affected long bone(s) show variable changes in the density of the medullary

cavity; the appearance varies with the phase (early, middle or late) of the disease.

Treatment/Prognosis

- Supportive: management of pain is crucial--aspirin or other non-steroidal anti-inflammatory drug as needed during the painful (4-6 weeks) period.
- **Resolution:** the problem can persist for several months...but eventually will resolve.