Key Points:
- Hip dysplasia has a complex pathophysiology, with the predominant feature of laxity
- Clinical signs will vary depending on the stage of disease; however, an acute non-weight bearing lameness in a mature dog is rare and thus cruciate ligament damage should be considered
- A thorough physical examination and GOOD QUALITY WELL POSITIONED radiographs are needed for a diagnosis
- Early detection is key for appropriate treatment but also for owner education in terms of lifestyle changes and focusing on a lean body weight

Hip dysplasia (HD) was originally described in 1935 by Gerry Schnelle and has become one of the most common orthopedic conditions that leads to joint inflammation and secondary osteoarthritis (OA). Unfortunately, even after all of this time the exact etiology is unknown, but is considered to be multi-factorial. One such factor involved in the expression of HD is genetics. It is not a simple Mendelian pattern but rather a complex inheritance. This means there are multiple genes that are combined with environmental influences that lead to the clinical expression of HD. Joint laxity is considered the initiating cause of HD which in turn leads to hip subluxation and poor congruence between the femoral head and acetabulum. Multiple causes of hip laxity have been described such as abnormal hip development, biomechanics, genetic influences, increased joint fluid, pelvic muscle mass, nutrition, weight/growth, and hormonal and environmental factors. It's probably safe to assume that HD and the subsequent arthritis are the clinical manifestation of all of these.

At the very basic level dogs with HD have normal hips at birth. The hips will remain normal if complete congruity is maintained between the femoral head and acetabulum. However, if one or more of the previously discussed factors that leads to hip laxity is present then the dog will manifest as having HD. Laxity is typically defined by the distraction index (DI), which has been shown as a primary risk factor for the development of OA. Passive laxity as measured with various radiographic and diagnostic techniques is an estimation of functional laxity which permits hip subluxation. From a hip development standpoint the earliest dysplastic joint changes can be noted as early as 30 days where there is increased volume of the ligament of the head of the femur, and increased synovial fluid volume. The ligament of the femoral head is the primary stabilizer for the hip for the first 30 days of life. For the first 2 weeks of life the ligament is short so if the hip is forced to luxate the femoral head will fracture at the fovea. After about 2 weeks the ligament will begin to lengthen; in dysplastic dogs this lengthening allows lateral subluxation of the hip. This subluxation allows articular cartilage to become worn and roughened on the dorsal surface of the femoral head at its point of contact with the acetabular rim. The first radiographic signs of HD can be noted at 7 weeks of age where subluxation of the femoral head with under development of the cranio-dorsal acetabular rim may be noted.

From a biomechanics standpoint in a healthy congruent hip the forces are distributed across the entire cartilaginous surface of the acetabulum. The co-contractions of the gluteals and adductors, along with the biceps femoris, semimembranosus and semitendinosus, create a force to reduce and stabilize the femoral head into the acetabulum during the stance phase of the gait. During the swing phase the primary muscles used to advance the limb are the transarticular muscles of the rectus.
femoris, sartorius, and iliopsoas, which have long muscle bellies with lines of action more parallel to the axis of the femur. In patients with hip laxity and thus subluxation the transarticular forces must increase to compensate for lateralization of the center of rotation of the joint. Additionally, the cartilage stress is increased because the forces acting on the articular cartilage are spread over a reduced surface area. This ultimately results in two destructive events: forces crossing the joint increase while the area over which the forces are transmitted decreases. **What this means is that in patients with HD the femoral head subluxates during the swing phase of the gate and upon foot strike the larger hip extensors cause catastrophic reduction of the femoral head.** Additionally, less muscle mass during development is associated with an increase in joint laxity. There has been a disparity noted between the strength of pelvic muscles and rapid weight gain, which leads to joint instability. Also, muscle mass of dysplastic breeds such as German Shepherds is less than that of non-dysplastic breeds such as Greyhounds.

Nutrition is thought to be a large contributor to joint laxity and thus HD; however, no dietary deficiencies cause HD. Dietary excesses, on the other hand, can contribute to the development of HD. For example, increased calcium and vitamin D lead to alterations in endochondral ossification, and delayed bone remodeling. Diets containing excessive vitamin C can lead to hypercalcemia and diets with a high anion gap lead to increased synovial fluid production, which in and of itself has been shown to be a risk factor for hip laxity. Feeding diets to promote rapid growth have been shown to lead to a higher incidence of HD and also cause early fusion of the acetabular growth plates.

Increased body weight is not a cause of HD, but it certainly has very important clinical consequences in susceptible dogs. Therefore, weight reduction is an effective preventative strategy. In the lifespan study of 49 Labradors it was reported that heavier dogs (dogs allowed to eat ad lib) developed radiographic OA on an average of 6 years earlier than the dogs in the restricted feed group. Furthermore, heavier dogs required long-term treatment for OA on average 3 years earlier than their restricted diet littermates.  

The diagnosis of HD is made from the signalment, clinical signs, physical exam findings, and radiographs. Affected dogs are typically large breed, fast growing dogs such as German Shepherds, Rottweilers, Labradors, or Golden Retrievers. The age of presentation is typically biphasic and contributes to the type of treatment that may be recommended. Juvenile dogs will tend to present between 5-12 months of age with an acute onset of unilateral or bilateral hind limb lameness. These clinical signs are thought to be due to joint laxity. As dogs become older the long-standing joint laxity causes periarticular fibrosis, which may decrease or lessen the clinical signs. This is why some dogs will tend to have improvement in clinical signs until later in maturity when they present for clinical signs that are consistent with OA.

The severity of clinical signs depends on the stage/severity of the disease. Lameness can be intermittent, progressive, and range from mild to severe. In young patients with severe laxity a “popping” noise may be heard during ambulation. Both young and older patients may exhibit exercise intolerance and difficulty rising due to pain and discomfort. Disuse muscle atrophy is a common finding and the gait may be characterized as either “swaying” or hopping. **It is very important to remember that a non-weight bearing lameness in a mature dog is rare and thus other problems should be considered, such as a cranial cruciate ligament rupture.** Orthopedically pain in the hips along with crepitus may be noted. Many of these patients have decreased range of motion in extension and weight shifting to the forelimb. Evidence of joint laxity is determined through the Ortolani and Barlow’s tests. The Ortolani is performed with the patient in either lateral or dorsal recumbency. The first part of the Ortolani is the Barlow test where a force is directed through the femur through the dorsum to subluxate the hip. The Barlow test is considered a provocative test in that it creates subluxation in a lax hip. The second part of the Ortolani test is the true Ortolani
maneuver where the limb is abducted and a click or clunk can be heard as reduction of the hip occurs. The clunk is considered a positive Ortolani and is indicative of coxofemoral laxity. Some surgeons will use the angles measured during an Ortolani test as indications for a triple or double pelvic osteotomy. Unfortunately, Ortolani and Barlow’s only suggest laxity and do not predict later development of clinical signs of OA.

Radiographs are a mainstay for the diagnosis of HD, along with the characterization of the disease and any presence of OA. There are several ways to evaluate canine hips, which vary from using the hip extended view as is done with OFA, or developing a distraction index as is done with PennHip. OFA style radiographs are generally used in daily practice; this requires that the pelvic limbs are fully extended and parallel, the pelvis is symmetrical and the pelvic limbs are internally rotated. Sedation and/or general anesthesia is required. Malpositioned radiographs can lead to false assumptions. The two most notable and early signs with hip OA are the circumferential femoral head osteophyte (CFHO) and the caudo-lateral curvilinear osteophyte (CCO). The CFHO is a white line at the articular margin of the femoral head that may or may not extend completely around the femoral head. It is graded from I to III. The CCO is a well-defined linear density on the femoral neck between the greater trochanter and the capital physis. It is different from a puppy line in that a puppy line is an indistinct radiodense line on the femoral neck in dogs and should be gone by 18 months of age. It is in a similar location to the CCO but it is more subtle, more diffuse and shorter than the CCO. A puppy line is considered self-limiting and is not clinically significant.

Treatment for HD can be broken into prevention and/or laxity improvement utilizing the juvenile pubic symphysiodesis (JPS) or triple/double pelvic osteotomy (DPO or TPO). More definitive treatment can be accomplished with a femoral head and neck ostectomy (FHNO or FHO) or a total hip replacement (THA). Medical management can be utilized for HD, but it is important to realize this is considered palliative rather than definitive. In immature dogs that are still growing with no evidence of OA then medical therapy can be attempted. This includes promoting weight loss, daily activity, and formal rehabilitation therapy to improve muscle mass, range of motion, and comfort. Many of these patients benefit from NSAIDS, chondroprotectants, and omega-3 fatty acids. For those that are severely clinically affected or have failed medical therapy then either a JPS or DPO/TPO, FHNO, or THA can be considered. In mature dogs medical management is geared towards OA management. Older dogs that become refractory to medical management would then become candidates for either a FHNO, or THA. Regardless early detection is key. In susceptible breeds hip palpation should begin by 12 weeks of age. If they have a positive Ortolani or have a high DI after 16 weeks of age then JPS should be considered in at risk breeds. A JPS is a minimally invasive way to pre-maturely cause fusion of the pubic symphysis. This causes ventrolateral rotation of the acetabulum with growth of the animal (resulting in ventroversion and improved femoral head coverage). The procedure is completed with a small incision to the pubic symphysis; electrocautery is then used every 2-3 mm along the symphysis at 40 watts for 12-30 seconds. Best results are achieved in patients before 16 weeks of age, resulting in about 10-15 degrees of ventroversion if done at 16 weeks. No real benefit is gained if completed in animals greater than 20 weeks of age. The resultant hip changes are similar to what is seen with a DPO/TPO; however, it is easier and faster with fewer complications and no implants are needed.

A DPO/TPO involves osteotomies of the ischium (only with TPO, not with DPO), pubis, and ilium. It causes reorientation of the acetabulum to increase dorsal coverage of the femoral head (thus resulting in ventroversion). In theory there should be improved joint stability and congruence and hopefully reduction in the formation of OA. A DPO/TPO is reserved for patients that have no evidence of OA. The age restriction has been discussed as being less than 10 months of age (typically 6-9 months is ideal); however, I have performed TPOs in dogs 12-14 months of age with success if there is no OA present. It is said to result in about 92% improvement in lameness and slower progression of
OA. However, the complication rate approaches near 50% and includes excessive narrowing of the pelvic canal, temporary constipation, sciatic nerve injury, implant failure, screw loosening, and continued OA development.

A FHNO has typically been reserved for smaller dogs and cats; however, larger dogs can also be candidates. It involves removal of the entire femoral head and neck and relies on the formation of a pseudoarthrosis. Even though owner satisfaction is high it is a salvage procedure with 62-65% return to normal function from a gait analysis standpoint. Probably the biggest complication with a FHNO is leaving femoral neck behind; other complications include shortening of the limb, patellar luxation, muscle atrophy, limited hip extension, recurrent lameness and chronic pain. In my hospital patients are required to undergo formal rehabilitation therapy beginning 3-5 days after surgery and continuing for 6-12 weeks.

A THA or “hip replacement” is considered by most to be the gold standard treatment for severe HD that is refractory to medical management. In the past it has be reserved for larger dogs; however, it can now be completed in smaller dogs and even cats. A THA results in about 95% return to function from a gait analysis standpoint. Often unilateral THA is enough to provide adequate function in bilateral disease. The complication is less than 10% but this is very dependent on surgeon ability. Contraindications for a THA are local or systemic infection, neoplasia, concurrent cruciate disease, or neurologic dysfunction. Potential complications include aseptic loosening, implant failure, infection, femur fracture, coxofemoral luxation, and sciatic nerve damage.

In summary, HD has a complex pathophysiology with the predominant feature being joint laxity. There are many factors that contribute to joint laxity. Clinical signs will vary depending on the stage of disease, but remember an older dog that is acutely non-weight bearing will oftentimes have a cruciate rupture with underlying HD. A thorough physical examination with good quality radiographs is needed. Early detection is key, so that way a JPS can be performed.