Hip Dysplasia: Conservative and Rehabilitation Management

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Key Points:
- Hip Dysplasia has a complex pathophysiology, with the predominant feature of laxity
- The conservative management of hip dysplasia is considered to be palliative
- The goals of conservative management are to reduce clinical signs of pain, improve hip range of motion, improve strength, maintain muscle mass, and slow the progression of OA

Hip dysplasia (HD) has become one of the most common orthopedic conditions that leads to joint inflammation and secondary osteoarthritis. Medical/conservative management is an option in patients with HD. It is important to understand that conservative management is considered a palliative approach rather than a definitive approach in both the immature and mature dog. Since the conservative approach to HD is considered palliative, the approach should be multimodal. The goals are to **reduce the clinical signs of pain**, **improve hip range of motion (ROM)**, **improve hip strength**, **maintain muscle mass**, and **slow down or minimize progression of OA** in the immature dog. In the mature dog the goals are **similar to management of osteoarthritis (OA) in any joint**.[1]

To appropriately achieve the goals there needs to be a focus on **weight reduction, strength, and fitness**. To be able to even focus on these areas the patients have to be comfortable, so **minimizing joint pain is paramount**. This can be accomplished with the use of pharmaceuticals or disease modifying OA agents (DMOAs). In addition, to assist not only with pain management but also strength and fitness, **formal rehabilitation** can be considered through the use of manual therapies, therapeutic exercises, and physical modalities.[1]

Weight Reduction, Strength, and Fitness:

From a weight reduction standpoint, a higher plane of nutrition or anything that leads to rapid growth has been shown to lead to a higher incidence of HD. Earlier fusion of acetabular growth plates has been thought to lead to dysplastic hips. Increased body weight is an influential environmental factor. Increased body weight alone does not cause HD, but it plays an instrumental role in the manifestation of the disease phenotype. Furthermore, weight reduction has been accepted as a highly effective preventive strategy for delaying or preventing the onset of OA in susceptible dogs. In Gail Smith's lifespan study of Labs[2], it was reported that heavier dogs developed radiographic OA on an average of 6 years earlier than the restricted fed group. Heavier dogs required long-term treatment for OA on average 3 years earlier, which was reported by Kealy et al.[3,4]

To achieve weight reduction the goal is for 10% reduction. This is derived from humans where losing 10% of body weight improved comfort in patients with OA. For this to be done safely I shoot for 1-2% weekly. From a diet standpoint this is established by feeding 60% of the calories needed to maintain the current body weight. Unfortunately, diet alone is rarely successful for long-term weight management. The other important consideration is a controlled low impact exercise program. Daily exercise can only occur in the presence of a pain free joint; therefore, minimizing joint pain is paramount. Daily exercise can consist of leash walking, waking in water, and swimming. When performed on a daily basis exercise can help to maintain strength and range of motion.

In humans with OA it is reported that muscle strength decreases due to disuse, and reflex

inhibitions due to the contractions of muscles adjacent to arthritic joints. The loss of strength can be measured by both quantitative (due to a loss of muscle mass) and qualitative (due to a loss of muscle performance) methods. At this time there is nothing in the literature reported on the loss of strength in dogs. This is likely because it would be hard to actually quantify. However, loss of muscle mass is reported commonly in dogs with hip dysplasia and we assume this is due to disuse. From this we can suspect that loss of muscle mass may be correlated with loss of muscle strength. Daily exercise helps maintain fitness and endurance. To achieve this it requires repeated motion over a given period of time. In dogs this will not be achieved through self-driven activities in the back yard, but rather with the compliant owner that is willing to perform daily activities with their dog. Again, daily exercise at the very least needs to consist of long leash walks. Achieving daily exercise requires that the patient is comfortable and pain is minimized.

Minimizing Joint Pain:

Minimizing Joint pain is absolutely the most important factor when it comes to the conservative management of hip dysplasia. This is achieved through multiple routes such as optimizing the living conditions or in other words making lifestyle changes, tailoring activities, encouraging weight loss, pharmaceuticals, and disease DMOAs. From an optimization of living conditions standpoint in the dog little is actually known. From an OA standpoint clinical signs include periods of calmness (the so called “status quo”) and periods of exacerbation of signs known as flare-ups. Over time these flare-ups will become more frequent, severe, and last longer. Commonly, flare-ups result from excessive activity in an unfit dog or the so-called “weekend warriors”.

Flare-ups may last several hours to several weeks. Determining if a flare-up is present is critical. In most mature dogs that present to you for a “hip consult” they are in the middle of a flare-up; otherwise, why would the owner have elected to come see you? If there is a history of a sudden loss of performance, or inability to climb stairs or exercise intolerance then this could indicate a flare-up. Making irreversible decisions such as surgery or euthanasia during a flare-up should be avoided. During a flare-up the focus should be on rest and pain management to get the patient back to status quo. The goal is to avoid strenuous activities such as jumping up, chasing, retrieving, or playing with other dogs. Unfortunately, owners like these activities because dogs love them, they are easy to do, and require minimal to no owner participation. Once the flare-up is under control then the patient can go back to their status quo with a focus on daily exercise to maintain muscle strength and range of motion as well as work to maximize the duration between flare-ups.

By far and large the biggest aspect of conservative management is controlling joint pain with pharmaceuticals because without proper pain relief nothing else matters. We will not be able to successfully achieve strengthening, maintenance of muscle mass or ROM. To adequately control pain we must be able to interfere with both the peripheral (inflammation and damage) and central (neuropathic) aspects of pain. The goal of addressing the peripheral component is decreasing the transduction and transmission of noxious stimuli. Non-steroidal anti-inflammatories (NSAIDs), and piprants (Galliprant®) seem to be at the center of this in both human and veterinary medicine. In the dog NSAIDs have been shown to improve both peak vertical force and vertical impulse.[5] In addition they have a positive impact on comfort and mobility. NSAIDs can have infrequent but potentially serious side effects and should be used cautiously in dogs, especially in patients with compromised liver or renal function or gastrointestinal disease. This becomes relevant since many older dogs with OA secondary to hip dysplasia are older when disease conditions are more likely to be present. Periodic enzymatic and liver function screening is recommended in dogs receiving NSAIDs. While there is no consensus on the frequency of these screenings, it seems reasonable to perform them before the administration of NSAIDs, after a few weeks of administration, and when clinical signs arise. The central component of minimizing joint pain is to manage the peripheral sensitization and spinal cord “wind-up”. For this to occur there has to be a multimodal approach with the use of other

analgesics adjunctive or as an alternative to NSAIDs.

From a central component there are various medications that can be used. One big question that is becoming discussed more and more is the use of tramadol in dogs. From a pharmacokinetic standpoint we know there are big differences between the dog and humans. Interestingly, Steven Budsberg[6] presented data from a 3-year project that was a double-blinded, placebo controlled crossover study that used both positive and negative controls. They found that tramadol was no different than using a placebo when it came to objectively measuring pain and that the positive control revealed that the dogs did indeed objectively improve proving that their model worked. Tramadol may act as a good selective serotonin reuptake inhibitor (SSIR). In some dogs it can act as a good sedative and dogs tend to become "spacy" on it. I suspect this is what people perceive as it being effective. In addition the metabolism is rapid and variable in dogs. When combined with other SSIRs it can increase the likelihood of serotonin sickness. Other analgesic options include the use of codeine (1-1.5 mg/kg q8h), which is my preferred choice of opioid. Amantadine (3-5 mg/kg q24h) is actually the only drug that has been studied to manage canine OA. It can act as an NMDA inhibitor and when combined with other analgesics can provide good pain relief. Gabapentin (5-10 mg/kg q8-12h) has become a commonly used medication in the management of OA for central wind-up pain. It acts as a calcium channel blocker; however, some of the mechanism of action is unknown. It should be noted that we have no data supporting its use in OA management. Amitriptyline (0.5-1.0 mg/kg q12-24h) is another medication that can be used. It is a selective serotonin reuptake inhibitor so caution must be used when combining it with tramadol.

One of my biggest soapboxes is this current thought that using tramadol and an NSAID is appropriate for long-term management of painful conditions. Opioids are not designed to be used for everyday management of pain associated with OA. Why do you think we have a current opioid crisis in the US?? They are designed to be used short-term and only as needed. They are beneficial during periods of flare-ups. I would love to see everyone move away from the NSAID/tramadol approach and stating, "that's all we can do" theory.

In addition to the other aspects of minimizing joint pain, DMOAs can be considered. In humans over 30 compounds have been shown to provide some degree of pain relief when analyzed prospectively. There is a lack of data in veterinary medicine, but it does exist. In dogs we tend to focus on glucosamine/chondroitin sulfate, polyunsaturated (omega-3) fatty acids, and polysulfated glycosaminoglycans. In general, the world of joint supplements can be deep and dark. If you only recommend a joint supplement to an owner, they are going to buy the cheapest they can find; however, they need to be told that not all are created equal. The FDA has no regulations on nutraceuticals; therefore anyone can start a nutraceutical company. No company has to meet label claims, and in fact 93% of companies fail to meet label claims. The source, purity, ethical issues, safety, and good manufacturing protocols (GMP) all have to be considered when choosing a company. What has also been shown is we need a product that has both glucosamine/chondroitin sulfate, given that the chondroitin sulfate has such poor bioavailability alone. Also, a synergistic effect has been shown with both a glucosamine/chondroitin sulfate product. Remember that source plays a huge role in this, so just because a product has both does not mean it will be synergistic. Furthermore, Sherman Canapp demonstrated not only efficacy but also a protective effect justifying the use before injury or evidence of radiographic OA.[7] Omega 3 fatty acids are important for managing inflammation. Essentially, omega-3 fatty acids reduce levels of arachidonic acid in the cell membrane, which lowers the production of certain prostaglandins; this in turn reduces inflammation. Interestingly, canine chondrocytes store EPA, which is a type of omega-3 fatty acid. This helps decrease aggrecan degradation and turns off the signaling responsible for making degradative enzymes. Inflammation begins with chondrocyte damage leading to the release of arachidonic acid from the cell membrane

phospholipid. As the inflammatory cascade continues lipoxygenase is released, causing the release of leukotrienes. Furthermore cyclooxygenase is released from arachidonic acid leading the production of prostaglandins. Both leukotrienes and prostaglandins cause continued inflammation and tissue damage. Omega-3 fatty acids replace the omega-6 fatty acids and help inhibit the production of lipoxygenase and cyclooxygenase. The dose of omega-3 FA for OA management is much higher than for other conditions; the current recommendation is for 150-175 mg/kg of DHA/EPA.

Polysulfated glycosaminoglycans are the only other class of drugs approved by the FDA to treat OA in the dog. These are considered disease modifying osteoarthritis drugs and are also approved for use in horses. Polysulfated glycosaminoglycans are water-based, for intramuscular injection at a dosage of 2 mg/lb. body weight twice weekly for up to 4 weeks, with a maximum of 8 injections). The specific MOA is not known, however in vitro studies show inhibition of serine proteinases, PGE_2 synthesis metalloproteases, hyaluronidases, and others as well as increased synthesis of protein, collagen, proteoglycans, and hyaluronic acid. Anecdotally "maintenance" injections monthly are considered helpful. The label is designed for IM usage; SQ usage is considered acceptable, but it should be known that it is off-label.

Formal Rehabilitation:

Rehabilitation can play a huge role in achieving our goals of conservative management of HD. The definition of rehabilitation is the treatment or management of physical disability, malfunction, or pain using physical agents. There are various methods to achieve this such as physical modalities like therapeutic ultrasound, neuromuscular electrical stimulation (NEMS), trans-cutaneous electrical stimulation (TENS), therapeutic laser, magnetic field therapy, shock wave therapy, manual therapies and therapeutic exercise. The goals of rehabilitation are to maintain muscle mass, build muscle support around joints, reduce pain, and achieve a healthy weight.

The basics of manual therapy include cold/warm therapy in addition to massage, acupuncture, and electro-acupuncture. It should be noted that cold therapy and massage should always be performed after exercise, not before. Cold therapy should be applied for 10-20 minutes 2-3 times daily. It will decrease nerve conduction and edema so it can be very useful for the management of central pain management. It is also helpful during flare-ups. Low-level warm packs in addition to acupuncture or electro-acupuncture can help with decreasing myofascial pain and tension. They help by stimulating the Aβ sensory fibers while sparring the Aδ and C fibers. Low-level heat allows for vasodilation and normalization of blood flow; it should be applied for 10 minutes 2-3 times daily.

Little is known about the impact of OA on joint motion; however, in patients with hip dysplasia we know that they lose hip extension, not flexion. Normal hip extension is approximately 160 degrees; interestingly a loss of less than 10 degrees is probably not clinically relevant. Because it is easier to maintain motion than to regain it when it is lost, assessing joint motion through goniometry is vital. ROM and stretching are typically incorporated early in the course of rehabilitation to increase flexibility, prevent adhesions, remodel periarticular fibrosis, and improve extensibility. This is accomplished with 10-30 reps 2-4 times daily for range of motion and 20-40 second holds for stretching. Technically, range of motion is completed without muscle contraction while moving the joint through its full range of motion. Any force applied at the end of ROM is considered stretching. Both range of motion and stretching are more effective when tissues are heated. In addition, ROM/stretching can be incorporated into part of the daily exercise program to maintain mobility, enhance blood/lymphatic flow, and improve synovial fluid production.

Therapeutic exercises are really the occupational aspect of rehabilitation. While little is known about proprioception in dogs with OA or in immature dogs with hip laxity it is logical to assume that some sensory deficits are present. The focus of therapeutic exercises should be on

balance and proprioception. This can be accomplished with lots of different things such as weight shifting (15-25 repetitions 2-4 times daily) and 3-legged standing (10-20 repetitions 2-4 times daily) to more advanced exercises using balance boards, wobble boards, or exercise balls. Patients can also walked on uneven surfaces such as air mattresses, foam rubber, sand, etc. The overall goal is to do things that promote extension of the hip. Probably the most effective and simplest form of therapeutic exercise out there is controlled leash walking. However, when we discuss this approach in patients with hip dysplasia it is important to consider what we are trying to achieve and how walking will affect this. For example walking on level ground will generate 35° of motion in the hip. Simply walking on an inclined treadmill will increase hip extension by 3°. Interestingly, trotting does not seem to increase ROM any more than walking. Walking up stairs can increase hip extension by 10° while walking up an incline does not help in increasing hip extension. My plan is to start and build up to level ground walking for 20 minutes 2-3 times daily and slowly continue to add time to get to 45 minutes. Once the dog is at 20 minutes comfortably, then inclined walking with stair ascent and descent should be added. Lastly, add declined slopes and uneven terrain. Some simple therapeutic exercises that can be taught to owners to do at home are Cavalettis, sit- to-stand, and dancing exercises. Cavalettis will improve total hip ROM. Increasing the height of the bars will result in further increasing total hip ROM. Sit-to-stands are excellent to improve total hip ROM. It won't directly lead to increased hip extension; however, it is excellent to use in the early phases when hip extension is painful. Dancing exercises are some of my favorite. Some consideration has to be made regarding the direction that dogs are being asked to dance. Forward dancing results in less hip extension and therefore is probably safer and more comfortable in the early phases. Once the dog's hip extension is improved or more comfortable then walking backwards can be incorporated as walking backwards puts more stress on hip extension.

If I were asked to provide my basic at home exercise plan for both the immature dog for hip dysplasia management or the older dog with OA I would start with a 5-10 minute warm up followed by walking. The goal is to get to 20 minutes on flat ground before adding in inclines, stair ascent/descent, and finally declined slope and uneven terrain. Once returned from walking I then have the owners perform 10-20 repetitions of sit-to-stand exercises along with 10 repetitions of dancing exercises. The dancing is initially performed forward, and once comfortable performed backwards. Following the exercises I have the owners perform 10-20 repetitions of ROM along with 20-40 second holds for stretching. Lastly, the dogs are allowed 5-10 minutes of a cool down. All of this is performed 2-3 times daily.

The goal of physical modalities in formal rehabilitation is to augment therapeutic exercises as well as help with tissue healing. Various modalities such as therapeutic ultrasound, electrical stimulation, shock wave, therapeutic laser, and hydrotherapy can all be included. Therapeutic ultrasound (US) can be beneficial in patients with hip dysplasia to aid improving ROM and decreasing pain and muscle spasm. It allows heating of the tissues deeper than what can be achieved with hot packing alone. The thermal effect of therapeutic US may increase collagen extensibility, blood flow, and pain threshold. To achieve the maximum benefit an intensity of 1.0-2.0 W/cm2 is used for a treatment time of about 8-10 minutes. Since the thermal effects of therapeutic US are short-lived, ROM and stretching exercises are commonly performed during the US session. Electrical stimulation is useful for increasing muscle strength, ROM, and muscle tone. In addition it can improve pain control, decrease edema and muscle spasm. There are 2 types of electrical stimulation; neuromuscular electrical stimulation (NMES) is helpful for muscle re-education, prevention of muscle atrophy, and enhanced joint movement. Transcutaneous electrical stimulation (TENS) is helpful for pain control. In patients with hip dysplasia with long standing muscle atrophy NMES can be used twice weekly for 10-20 minutes; alternatively in patients with hip dysplasia and pain TENS can be used 2-3 times weekly for 30 minutes. Also, laser therapy is gaining in popularity for various disease conditions. It can be useful to relieve pain, reduce inflammation, and increase

microcirculation through the concept of photobiomodulation. My biggest thing is that laser therapy is used as part of rehabilitation, not as standalone rehabilitation. For patients with hip dysplasia and secondary OA I typically recommend 8-10 J/cm2. A generalized protocol is to start with 6 treatments over a 3-week period followed by maintenance treatments every 3-4 weeks. Regardless of the treatment protocol, the treatment should be designed to achieve the appropriate dose, number of treatments, and interval between treatments for the best outcome. Also, as a side note please ensure to wear goggles (both the 2 and 4 legged individuals). Extracorporeal shockwave therapy (ESWT) uses shock waves to target tissue to cause a release of energy. This release creates a biologic response to promote analgesia and decreases inflammatory mediators. The full analgesic effect is unknown, but thought to be due to the release of cytokines and growth factors centered around decreasing inflammation and swelling. One study showed that 4 weeks of treatment can improve ground reaction forces (GRF) in dogs for 3 months. Commonly sedation or anesthesia is needed to deliver 500-1,000 shocks at mJ/mm2 and 180 pulses/min. Ideally, there are 2 treatments spaced 3-4 weeks apart. Hydrotherapy is commonly recommended as part of the conservative management of hip dysplasia. We typically consider underwater treadmill therapy (UWTM) more beneficial than swimming. The changes in joint flexion are improved with both UWTM and swimming; however, almost near normal joint extension is noted in UWTM compared to swimming where hip joint extension is limited. In addition, the UWTM encourages a more normal gait pattern than what is achieved with swimming. The benefits of UWTM are the benefit of buoyancy, which allows active muscle contraction with decreased weight bearing, the hydrostatic pressure that reduces edema and decreases pain, and the resistance that helps strengthen muscles and improves endurance. In general UWTM will improve ROM and comfort while increasing muscular fatigue and building endurance. Initially, most patients should be started at 2- 5 minutes 1-2 times weekly with the goal to work up to 10-20 minutes with as few of breaks as possible.

When choosing a treatment program it comes down to what stage of hip dysplasia the patient in. In young immature dogs with hip laxity there should be a focus on daily exercise and weight management. However, it is important not to push aggressive exercise until after maturity. I will also add in DMOAs around 6 months of age. Also, to help promote hip extension I will have the owners incorporate ROM, sit-to-stands and dancing exercises. In addition daily play is very important. In mature dogs with OA and minor locomotion problems there should be a focus on decreasing pain, maintaining limb and core strength, maintain flexibility and stimulating proprioception. Simple pharmacologic steps are usually adequate such as using an NSAID at the lowest dose possible as infrequently as possible in addition to DMOAs. These patient should begin and remain on a long-term daily exercise plan with lots and lots of walking in addition to the ROM/stretching, sit-to-stand, dancing, and walking over/around obstacles. In mature dogs with moderate to severe OA the first step is pain control. This is accomplished through a multimodal pharmaceutical approach, as well as incorporating ice/heat, massage, acupuncture, TENs, and rest. Once pain is under control then the goal is to initiate a conservative and lightly progressive exercise program. This typically includes formal rehabilitation for 4-6 weeks along with daily walking and a home exercise plan. The absolute overall goal is to improve the patient's mobility, strength, and proprioception so we can give them the best quality of life as possible.

In summary our overall goals are to reduce the clinical signs of pain, improve hip ROM, improve hip strength, maintain muscle mass, and slow down or minimize progression of OA. To appropriately achieve the goals there needs to be a focus on weight reduction, strength, and fitness. To be able to even focus on these areas the patients have to be comfortable, so minimizing joint pain is paramount. This can be accomplished with the use of pharmaceuticals or disease modifying OA agents (DMOAs). The "meats and potatoes" of the long-term home exercise plan is daily walking and therapeutic exercises such as sit-to-stands and dancing exercises. Also, the addition of ROM/massage/stretching can be beneficial in maintaining hip ROM. To assist not only with pain

management but also on strength and fitness, formal rehabilitation can then be considered through the use of manual therapies, therapeutic exercises, and physical modalities. A patient-centered approach should be undertaken to improve the quality of life. If the conservative approach does not lead to an improvement in quality of life then surgical intervention should be considered.

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