Differential Diagnosis and Treatment of Pediatric Hip Pain

Differential diagnosis of any source of pediatric pain can prove challenging. If a child is old enough to answer subjective interview questions, the reliability of the child as a historian is often questionable. This may leave much of the history to the parent or caregiver, who is not directly experiencing the symptoms of interest. Pediatric hip pain is no exception to these challenges; additionally, the number of potential sources of hip pain in pediatric populations only contributes to this challenge. Direct hip trauma is typically a potential diagnosis. However, less apparent diagnoses such as transient synovitis, septic arthritis, osteomyelitis, autoimmune disease, leukemia, and sarcoma can also cause hip pain in pediatric populations.[1] Children may certainly present with hip pain that is orthopedic in nature as well; however, the diagnosis and even treatment is still not often simple. This tends to be the case with developmental dysplasia of the hip, Legg-Calvé-Perthes disease, and slipped capital femoral epiphysis.

Developmental dysplasia of the hip (DDH) is a broad diagnosis describing atypical development of the hip joint.[2,3] Depending on severity and involvement of dysplasia, a child may experience hip instability, subluxation, or dislocation. The most common cause of DDH relates to positioning of an infant in utero, or after birth, such that poor contact exists between the femoral head and acetabulum.[2]. The femoral head and acetabulum depend on one another structurally for optimal development.[4] If there is poor congruency within the hip joint, or malformation of either component, the hip joint is at risk for abnormal development.

DDH is estimated to occur between 1/1000 and 20/1000 live births.[2,4,5] The discrepancy in statistics is most like attributable to the broad nature of the diagnosis. DDH is most prevalent in females; additional risk factors include breech position, first gestation, improper lower extremity swaddling, multiple gestations, large birth weight, and positive family history.[2,3,4,5]

Breech positioning toward the end of pregnancy (as opposed to breech delivery) is the greatest risk factor, which is associated with 17 to 23% of diagnoses.[2] In frank breech, the infant's hips are positioned in flexion with bilateral knee extension, potentially resulting in suboptimal congruency at the hip joint.[2,4] Figure 1 in appendix A provides an image of frank breech positioning, as well as two additional types of breech that may result in poor hip congruency.

There is substantial emphasis on screening in infancy and early diagnosis due to the long-term effects of DDH if left untreated. The current recommendation is for all infants to be screened for DDH after birth and at any following pediatrician visits.[4] During the first few months of life, the Ortolani and Barlow maneuvers are commonly used as clinical screening tools. In the Ortolani maneuver, anterior pressure is applied to the proximal femur with the hip in 90 degrees of flexion and slight abduction, attempting to reduce a posteriorly displaced hip joint.[2,3] The Barlow maneuver consists of placing the infant's hip in adduction and 90 degrees of flexion, and applying slight posterior force to the proximal femur, attempting to posteriorly dislocate an unstable hip joint.[2,3] Both maneuvers are represented by Figures 2 and 3 in appendix A. A "clunk" felt with either maneuver indicates a positive result. Additional clinical signs include asymmetric soft tissue folds and limb length discrepancy.[4] By three months of age, the unstable hip may become fixed and hyperlaxity associated with newborn ligaments resolves, making the Ortolani and Barlow maneuvers less useful. Instead, the main clinical sign becomes limited hip abduction.[2,3] If hip abduction is found to be less than 60 degrees, or in a younger infant, DDH is suspected due to signs and maneuvers described above, additional diagnostic testing such as imaging may be warranted.[3] Ultrasound is typically used to diagnose DDH in children under three months of age because the femoral head is not yet ossified.[2,3] The ultrasound may capture the hip joint at rest, or during the Ortolani and Barlow maneuvers.[4] In older children

with ossified femoral heads, radiographs may be taken to identify any asymmetry, subluxation, or dislocation of the hip joint. Following imaging, the child's hip dysplasia may be graded I through IV according to the Graf system; grade I indicates no joint abnormality, and grade IV indicates frank dislocation.[3]

DDH diagnosed in infancy may resolve spontaneously; however, when intervention is warranted, bracing and casting are often trialed prior to surgical intervention. The goal of either conservative or surgical intervention is to achieve sustained congruency of the hip joint in order to promote typical growth of the femoral head and acetabulum.[2] During the first few months of age, a flexion-abduction brace is most likely to be used (see appendix A, Figure 4); later in infancy up to 18 months of age, or if reduction is not achieve with bracing, closed reduction with subsequent spica casting may be performed.[2,3,4] If the child is older than 18 months of age at diagnosis, or if closed reduction is still unsuccessful, open reduction is most likely to be required.[2,3,4]

In some instances, especially for children born in countries or areas without access to quality healthcare, DDH may remain undiagnosed until later in childhood. An older child presenting with DDH may report hip or low back pain and may demonstrate limb length discrepancy, a waddling gait pattern, and decreased range of motion of the affected hip.[2,5] If DDH is suspected in these children, radiographs may confirm the diagnosis. It is important to note that the older the child's age at diagnosis, the poorer the prognosis overall and increased likelihood of requiring open surgical reduction.[2,3,4]

Physical therapy is most often recommended for children following casting or open reduction. However, Gather et al recently demonstrated positive outcomes using early mobilization following open reduction rather than spica casting, which may expand the future

role of physical therapy in the course of DDH treatment.[6] Early physical therapy post-surgical intervention or immobilization may incorporate both aquatic and land-based intervention until full weightbearing status is achieved.[7] Therapy should include gradual progression of range of motion, strengthening, and balance activities; functional and recreational activities such as stair climbing, kicking, running, jumping, and navigating uneven terrain may eventually be introduced as the child progresses.[8] Children diagnosed with DDH at an older age may trial physical therapy as conservative treatment prior to surgical intervention.[9] A small body of recent literature has also reported positive outcomes using hippotherapy in conjunction with physical therapy as conservative management for DDH.[8] However, if symptoms do not improve with physical therapy or worsen, open reduction is likely indicated.

Legg-Calvé-Perthes disease (LCPD), named after three of the physicians to first describe this condition, is characterized by idiopathic necrosis of the epiphyseal portion of the femoral head.[10,11] The necrosis is instigated by disruption of the vascular supply to this area; however, the cause of disruption is typically unknown. Theories describing potential causes of necrosis are quite varied and include genetic mutations of type II collagen, repetitive loading of the hip joint in positions of extreme range of motion, exposure to second hand smoke, and hyperactive behavior.[10,11] Pinheiro et al proposed that delayed ossification of the epiphysis of the femoral head as well as decreased stiffness of articular cartilage may be additional causes of necrosis leading to LCPD.[12] Each of these individual mechanisms may render the bone tissue of the femoral head more susceptible to microtrauma, which may ultimately result in obstruction of the area's blood supply and necrosis. Once the disease process is initiated, growth of the area ceases; bone resorption begins to occur at a higher rate than bone deposition, leading to further decreased strength and undesirable reshaping of the femoral head.[12,13] During this time, the bone

is at high risk for fracture and/or fragmentation.[14,15] Reossification of the femoral head eventually occurs in its new shape, and typical growth and even revascularization resumes. This entire process may occur over the span of 2 to 5 years.[13] LCPD is most often seen in males between 4 and 8 years of age with no significant past medical history.[10,12] However, it may ultimately occur at any age between 18 months and skeletal maturity. Younger age at diagnosis is associated with more favorable prognosis; specifically, diagnosis at 8 years of age or older is typically associated with poorer outcomes.[13,14]

Children presenting with LCPD may report mild, activity-based hip pain of insidious onset that may or may not radiate into the groin, thigh, or knee.[10,13] The pain may also be exclusive to one of the areas aforementioned with no reported symptoms in the hip. Any pain associated with LCPD often does not manifest until fragmentation or fracture of the femoral head has occurred. Unfortunately, these factors often lead to delayed diagnosis and treatment of LCPD.[10,15] The child may also demonstrate a painless limp during ambulation, or a positive Trendelenburg sign, which often occurs up to months prior to seeking treatment.[10,13] Hip range of motion is generally first limited in abduction and internal rotation, followed by flexion and adduction in later stages of the disease. In the very latest stages, muscle atrophy of the lower extremities and limb length discrepancies may also be observed.[10,13]

If LCPD is suspected due to clinical signs and symptoms, radiographs may be obtained. The preferred view for observing LCPD is an anterior-posterior image of the pelvis captured with simultaneous flexion and abduction of the child's bilateral hip joints.[10,13] In radiographs of children positive for LCPD, the joint space may appear larger than expected, subchondral bone may appear more radiolucent, the femoral head may appear flattened, and the acetabulum may

have remodeled such that it no longer encompasses the femoral head.[10,13] In more severe cases, bone fragmentation may be observed or even collapse of the femoral epiphysis.[13]

There is currently no cure for LCPD; additionally, because only theories surrounding its etiology currently exist, there is no method for prevention of the disease.[14] However, it is crucial for diagnosis to occur prior to reossification of the femoral head.[10] The primary goal of treatment for LCPD is similar to DDH in that congruency of the hip joint must be preserved throughout the reossification process. This promotes desirable reshaping of both the femoral head and acetabulum in order to minimize functional restrictions in the future. The secondary goal of treatment is to preserve the child's range of motion.[14,15] Conservative methods of treatment begin with non-steroidal anti-inflammatory medications, protected weightbearing, activity modification, and physical therapy.[10,13,14] If these methods are unsuccessful, containment of the femoral head in the acetabulum may be attempted through bracing or casting, which maintains the hip in 30 to 45 degrees of abduction (see appendix B, Figure 5).[14] If conservative treatments, including bracing and casting, prove ineffective, surgical intervention may be required in order to restore the optimal shape of the hip joint.[11] Common surgical techniques for less advanced LCPD include femoral varus osteotomy and pelvic redirection osteotomy, such as Salter's innominate osteotomy. For more advanced cases of LCPD, triple pelvic osteotomy may be performed or a combination of the two techniques previously mentioned.[11] Generally children under 6 years of age tend to have positive outcomes with conservative management, while older children often require surgical intervention.[10]

As mentioned previously, physical therapy may be trialed as a conservative intervention for LCPD. Additionally, physical therapy may be performed pre- elected surgery, bracing, or casting in order to maximize range of motion prior to the period of required immobilization.[14]

Physical therapy intervention pre- and post-surgery often incorporates aquatic intervention until the child is able to achieve full weightbearing status. The first phase of therapy should focus on range of motion activities of low intensity and impact.[14] The second phase typically incorporates strength, balance, and coordination activities. The third and final phase includes return to sport and functional activities.[14] Wise and Binkley produced a table of aquatic exercises appropriate for this population, which is organized according to each phase (see appendix B, Table 1).[14] The authors suggest that progression through each phase should be guided by the patient's pain status and individual progress toward the goal of each phase. For example, the therapist may choose to initiate the second phase when the patient has achieved at least 80% of full, painless hip range of motion.[14] Throughout the course of rehabilitation, the patient should be encouraged to avoid high impact, repetitive activities in order to avoid damage to the healing hip joint. If the child's growth plates are open, this type of activity risks damage to the healing epiphysis and therefore the epiphyseal plate.[14] Gradual return to full weightbearing and eventually full participation in daily activities and sport should be guided by progress in therapy as well as healing of the femoral head as determined by the child's physician.[14]

Slipped capital femoral epiphysis (SCFE) refers to posterior displacement of the femoral head on the femoral neck.[10,16,17] Because of its attachment via the femoral head ligament, the femoral head remains in the acetabulum, resulting in anterior displacement of the metaphysis of the femur.[16] While the exact etiology of this displacement remains poorly understood, SCFE typically occurs during times of significant skeletal growth in childhood. During these periods of growth, growth hormone stimulates rapid formation of chondrocytes in growth plate cartilage in order to impose longitudinal bone growth.[16] This changing status at the growth plate may render it particularly weak, especially against shearing forces frequently imposed on the proximal

femur.[10,16] SCFE may be categorized as stable or unstable; in stable cases, some level of continuity remains between the epiphysis and metaphysis.[18] In this scenario, the patient is typically able to bear weight through the affected extremity with or without an assistive device. If the hip is unstable, and there is no continuity between the epiphysis and metaphysis, the patient is typically unable to bear weight even with an assistive device.[17]

SCFE is estimated to affect between 0.33 and 24.58 per 100,000 children, with males being 1.5 times more likely to be affected.[10,17] Higher incidences also occur in black and Hispanic children.[17] The average age for diagnosis of SCFE is 12.7 years.[10] However, this number has slowly decreased due to the rise in prevalence of pediatric obesity. Obesity results in greater forces crossing the growth plate, placing the child at even greater risk of SCFE during this period.[10] Additionally, many children with obesity are also diagnosed with type 2 diabetes. This results in increased presence of insulin throughout the bloodstream due to systemic insulin resistance.[16] Insulin is thought to stimulate chondrocyte production at the growth plates similar to process of growth hormone described previously, further adding to instability of the growth plate.[16] 20 to 80% of children diagnosed with SCFE have bilateral involvement; for those with unilateral involvement, the unaffected limb is likely to become involved within one year of diagnosis.[17]

Children presenting with SCFE may report activity-associated, dull pain of insidious onset located in the groin, thigh, or knee.[10,17,18] Similar to LCPD, the chief complaint may actually be knee pain, often resulting in delayed diagnosis and treatment.[18] In fact, the average length of time spent experiencing symptoms prior to diagnosis of SCFE is 4 to 5 months.[10] The child may first demonstrate decreased internal rotation, followed by abduction and flexion as severity of slippage increases. The child may even be observed sitting at rest with the involved

hip externally rotated. Antalgic gait pattern, or Trendelenburg may be observed as well as general lower extremity muscular atrophy and limb length discrepancy.[10,17] If a child presents with any of these signs or symptoms in combination, and is obese, a diagnosis of SCFE should be strongly considered.

If SCFE is suspected, radiographs must be obtained to confirm or rule out the diagnosis. An anteroposterior image is captured of the pelvis with the patient in bilateral hip flexion and abduction, similar to the imaging performed for LCPD.[10] However, if the child is in severe pain or the hip is deemed unstable, a lateral view may be obtained instead.[18] Three common radiographic observations are used to diagnose SCFE. The first is the Klein line, which is drawn along the superior portion of the femoral neck and should intersect the femoral epiphysis (see appendix C, Figure 6).[10,17,18] If the Klein line does not intersect the epiphysis, SCFE should be suspected.[10,17] The second observation is the metaphyseal blanch sign of Steel, which describes overlapping of the posteriorly displaced femoral head on the femoral metaphysis.[10] The third and final sign is widening and/or lucency of the growth plate.[10,17] This is most often seen during early stages of the disease process.[17] While magnetic resonance imaging (MRI) is not typically necessary in order to diagnose SCFE, it may be used to determine vascularity or quality of the bone if the slippage is deemed unstable.[10] MRI may also be used in the rare event that radiographic observations are negative, but SCFE is strongly suspected clinically.[17]

The primary goal of treatment for SCFE is to stabilize the displacement in order to prevent it from worsening.[10] If slippage is deemed mild and stable, in-situ fixation may be performed in order to further stabilize the joint while allowing continued growth of the femoral head and neck.[18] Another procedure often performed for stable SCFE is osteotomy, which can be used to maximize joint alignment.[18] Trochanteric osteotomy is typically performed for moderate

slippage; however, for more severe cases, osteotomy may be required more distally at the base of the femoral neck.[18] Because the shape of the femoral head and/or neck may be compromised during the disease process, the child is also at risk of femoroacetabular impingement (FAI). This can also be addressed during in-situ fixation or osteotomy in order to prevent the need for future surgical intervention.[18] Treatment for unstable SCFE is much more complex due to potential for increased involvement of the surrounding vascular supply and subsequent avascular necrosis. In these cases, arising secondary complications must be addressed as well as the slippage itself.[16] Surgical techniques for unstable SCFE are varied, but include modified Kramer's operation, Dunn procedures, capsular decompression, and epiphyseal reduction or pinning.[10,16,18] Regardless of surgical technique used, children with unstable SCFE generally have poorer outcomes; for example, limb length discrepancy, osteoarthritis, and even osteonecrosis of the femoral head may persist.[10]

Physical therapy is typically initiated shortly after surgical intervention for SCFE. The weightbearing status of the child will depend on whether the SCFE was deemed stable or unstable.[17] For stable cases, the child will likely utilize crutches for 2 to 3 weeks with rather rapid progression of weightbearing status. For unstable cases, crutches may be used for a minimum of 6 to 8 weeks, after which full weightbearing may still not be granted.[17] The first phase of rehabilitation generally focuses on pain control, protected mobility, and range of motion.[19] The second phase involves strengthening as well as weaning off of crutches and normalizing gait pattern. The third and fourth phases should initiate functional movements and endurance activities. Finally, the fifth phase focuses on return to activities and sport.[19] Progression through each phase is determined by patient response and communication between

the therapist and physician. If the child is obese, weight management should also be addressed throughout the course of rehabilitation in order to avoid future re-injury.[17]

Each of these similar yet distinct childhood pathologies share implications into adulthood. As mentioned, children diagnosed with DDH, LCPD, and SCFE are at increased risk for early onset osteoarthritis of the involved hip joints, especially if left untreated.[2,10,13] Karkenny et al reports that more than 50% of children diagnosed with LCPD will "develop disabling arthritis by the sixth decade of life."[10] It is important to note that the earlier treatment is provided, the lower the risk of the child developing early onset osteoarthritis.[2] Development of early-onset osteoarthritis also increases the potential need for early hip arthroplasty for these patients. For example, it is estimated that 45% of children diagnosed with SCFE will require a total hip arthroplasty within 50 years of diagnosis.[10] Even more concerning, these patients tend to have poorer outcomes following total hip arthroplasty compared to adults without childhood hip pathologies.[2] Because each of these diagnoses may involve undesirable reshaping of the hip joint, FAI may occur if left untreated. Limb length discrepancy may persist, leading to secondary issues such as low back pain. Decreased range of motion may limit functional activities, especially those requiring internal rotation.[10,13] For untreated DDH, hip instability may persist, leading to chronic pain, subluxation, dislocation, or avascular necrosis.[4] Unfortunately, even for children receiving treatment for DDH, LCPD, and SCFE, including surgical interventions, symptoms may return and continue even into adulthood.

Differential diagnosis of pediatric hip pain is complex. These three conditions alone may manifest in children of similar ages and have very similar clinical signs. However, the underlying pathology of each condition is very different. With the current trend toward direct access, children may present to physical therapy clinics with hip pain due to any one of these diagnoses.

Because early identification and treatment is critical to DDH, LCPD, and SCFE, physical therapists must be able to accurately determine the source of the child's pain and determine whether an outside referral is necessary. The therapist must also be skilled in educating the patient and family on protective measures to take until a physician is seen, such as protected weightbearing and avoidance of high impact activities. Because of the lifelong implications of these pediatric orthopedic hip conditions, affected patients may require physical therapy throughout their lifespans. As a result, physical therapists in all settings must have an understanding of these diagnoses, associated conservative and surgical interventions, and the possibility of lingering secondary issues. With this information in consideration, a physical therapist can help maximize both the short- and long-term health of the hip joint across the lifespan.

Appendix A - Developmental Dysplasia of the Hip

Figure 1: Breech positioning, especially frank breech, results in poor congruency of the femoral head and acetabulum.[2,4] Because the femoral head and acetabulum rely on each other structurally for development, an infant in any one of these positions is at risk of DDH. Image from Best Start Resource Centre, 2018.[20]

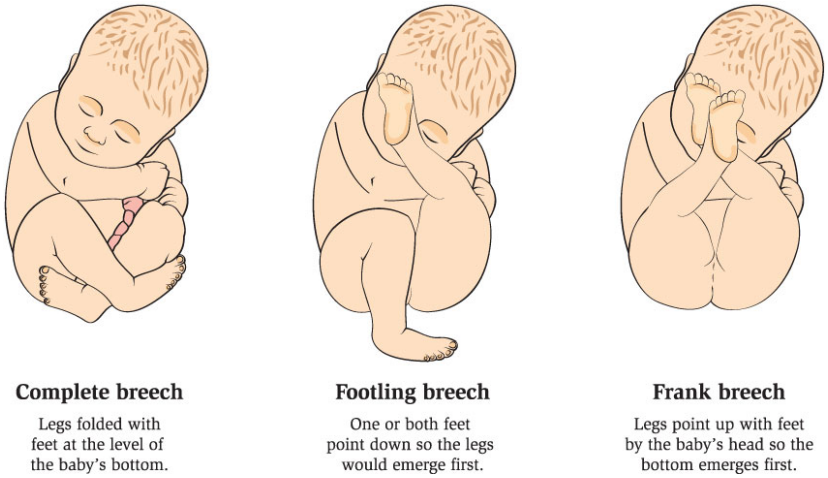


Figure 2: The Ortolani maneuver; a "clunk" felt when attempting to reduce the posteriorly displaced hip joint would indicate a positive result and high suspicion of DDH.[2,3] Image from Stanford Medicine, 2018.[21]

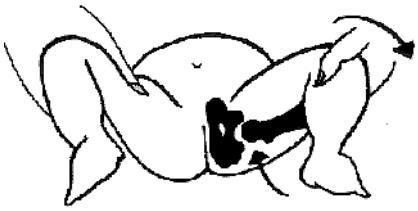


Figure 3: The Barlow maneuver; a "clunk" felt when attempting to posteriorly dislocate the unstable hip joint would indicate a positive result and high suspicion of DDH.[2,3] Image from Stanford Medicine, 2018.[21]

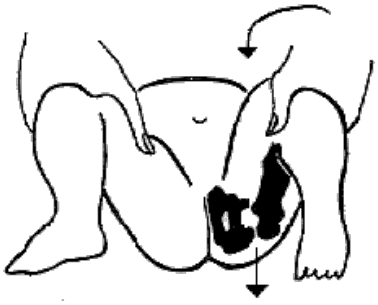
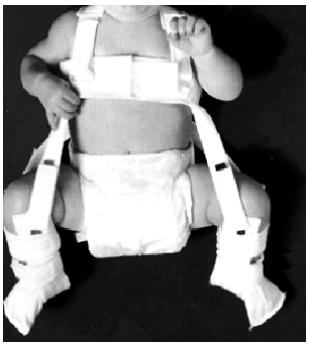


Figure 4: A flexion-abduction brace, such as this Pavlik harness, may be used to promote hip joint congruency in young infants with DDH. Image from OrthoInfo, 2018.[22]



Appendix B - Legg-Calvé-Perthes Disease

Figure 5: Abduction bracing may be used for patients with LCPD in attempts to contain the femoral head within the acetabulum.[14] Image from Orthotics & Prosthetics, 2018.[23]

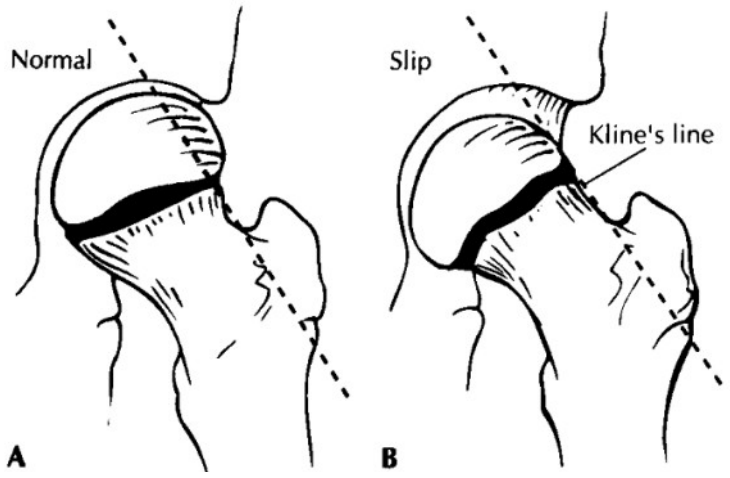
Table 1: Examples of aquatic exercises according to each phase of LCPD rehabilitation. Reproduced from Wise and Binkley.[14]

	Phase I (Range of Motion)	**Phase II (Strength, balance, proprioception)**	**Phase III (Sport-Specific)**
Deep Water Aquatic Exercises	Aqua-jogging Flutter kicks	Flutter kicks Shuttle run	Incorporate exercises from

	ABCs Bicycle Knee highs Cross-country ski	Backward running Scissors	previous phases as well as exercises more specific to the sport or daily activities
Shallow Water Aquatic Exercises	Kickboard Treasure diving Lateral shuffle ball toss Buoyancy-assisted abduction/internal/external rotation	Rocking horse Pad jumping Washing machine Dips Standing jumps Standing abduction (with resistance)	Skips Agility exercises Noodle swing-throughs Plyometrics

Appendix C - Slipped Capital Femoral Epiphysis

Figure 6: The Klein line is often used to diagnose SCFE on radiographic imaging.[10,17,18] In a healthy hip joint, the line should intersect the epiphysis (A). If the Kline line does not intersect the epiphysis (B), SCFE is a likely diagnosis.[10,17,18] Image from Houghton.[24]



References

1. Remler K. Pediatric hip pain: a case study. *J Pediatr Health Care.* 2017;31(6):713-716. doi:10.1016/j.pedhc.2017.08.004.
2. Auriemma J, Potisek NM. Developmental dysplasia of the hip. *Pediatr Rev.* 2018 Nov;39(11):570-572. doi:10.1542/pir.2017-0239.
3. Jackson JC, Runge MM, Nye NS. Common questions about developmental dysplasia of the hip. *Am Fam Physician.* 2014 Dec;90(12):843-850.
4. Shaw B, Segal LS. Evaluation and referral for developmental dysplasia of the hip in infants. *Pediatrics.* 2016 Dec;138(6):10.1542/peds.2016-3107.
5. Pollet V, Percy V, Prior HJ. Relative risk and incidence for developmental dysplasia of the hip. *J Pediatr.* 2017 Feb;181:202-207. doi:10.1016/j.jpeds.2016.10.017.
6. Gather KS, von Stillfried E, Hagmann S, Muller S, Dreher T. Outcome after early mobilization following hip reconstruction in children with developmental hip dysplasia and luxation. *World J Pediatr.* 2018 Apr;14(2):176-183. doi:10.1007/s12519-017-0105-7.
7. Beckloff K, Wong S. Aquatic therapy for early mobility in a child with late onset developmental dysplasia of the hip: a case study. *J Aquatic Phys Ther.* 2017;25(2):67.
8. Aldridge R, Schweighart F, Easley M, Wagoner B. The effects of hippotherapy on motor performance and function in an individual with bilateral developmental dysplasia of the hip (DDH). *J Phys Ther.* 2011 Jan;2(2):54-63.
9. Kraeutler MJ, Garabekyan T, Pascual-Garrido C, Mei-Dan O. Hip instability: a review of hip dysplasia and other contributing factors. *Muscles Ligaments Tendons J.* 2016 Dec;6(3):343-353. doi:10.11138/mltj/2016.6.3.343.

10. Karkenny AJ, Tauberg BM, Otsuka NY. Pediatric hip disorders: slipped capital femoral epiphysis and Legg-Calvé-Perthes disease. *Pediatr Rev.* 2018 Sep;39(9):454-463. doi:10.1542/pir.2017-0197.
11. Leroux J, Abu AS, Lechevallier J. Legg-Calvé-Perthes disease. *Orthop Traumatol Surg Res.* 2018 Feb;104(1):S107-S112. doi:10.1016/j.otsr.2017.04.012.
12. Pinheiro M, Dobson CA, Perry D, Fagan MJ. New insights into the biomechanics of Legg-Calvé-Perthes disease: the role of epiphyseal skeletal immaturity in vascular obstruction. *Bone Joint Res.* 2018 Feb;7(2):148-156. doi:10.1302/2046-3758.BJR-2017-0191.R1.
13. Dobbe AM, Gibbons PJ. Common paediatric conditions of the lower limb. *J Paediatr Child Health.* 2017 Nov;53(11):1077-1085. doi:10.1111/jpc.13756.
14. Wise SL, Binkley HM. Current management and rehabilitation in Legg-Calvé-Perthes disease. *Athletic Ther Today.* 2010 Jul;15(4):30-35.
15. Smith C. Increasing awareness of Legg-Calvé-Perthes disease. *Br J Sch Nurs.* 2014 Feb;9(1):21-23.
16. Jingushi S, Suenaga E. Slipped capital femoral epiphysis: etiology and treatment. *J Orthop Sci.* 2004;9(2):214-219. doi:10.1007/s00776-003-0755-2.
17. Castillo C, Mendez M. Slipped capital femoral epiphysis: a review for pediatricians. *Pediatr Ann.* 2018 Sep;47(9):e377-e380. doi:10.3928/19382359-20180730-01.
18. Otani T, Kawaguchi Y, Marumo K. Diagnosis and treatment of slipped capital femoral epiphysis: recent trends to note. *J Orthop Sci.* 2018 Mar;23(2):220-228. doi:10.1016/j.jos.2017.12.009.

19. Peck DM, Voss LM, Voss TT. Slipped capital femoral epiphysis: diagnosis and management. *Am Fam Physician.* 2017;95(12):779-784.

20. Best Start Resource Centre. About breech position and birth. Ontario Prenatal Education. https://www.ontarioprenataleducation.ca/breech-delivery/. Published 2018. Accessed December 3, 2018.

21. Stanford Medicine. Barlow and Ortalani maneuvers. Newborn Nursery at Lucile Packard Children's Hospital. https://med.stanford.edu/newborns/clinical-rotations/residents/residents-newborn-exam/barlow-and-ortalani-manuevers.html. Published 2018. Accessed December 3, 2018.

22. OrthoInfo. Developmental dislocation (dysplasia) of the hip (DDH). Diseases & Conditions. https://orthoinfo.aaos.org/en/diseases--conditions/developmental-dislocation-dysplasia-of-the-hip-ddh/. Published 2018. Accessed December 3, 2018.

23. Orthotics & Prosthetics. Scottish Rite orthosis. Orthotics & Prosthetics. http://www.360oandp.com/Orthotics-101-Scottish-Rite-Orthosis.aspx. Published 2016. Accessed December 3, 2018.

24. Houghton K. Review for the generalist: evaluation of pediatric hip pain. *Pediatr Rheumatol Online J.* 2009 May;7:10. doi:10.1186/1546-0096-7-10.