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Growth and Development of the Child's Hip

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The normal child's hip is the result of an intricate balance between a growing acetabulum, a growing proximal femur, and the vasculature that accommodates to the bony changes. The program for hip development begins with a genetic template actuated by a cascade of cell signaling factors. Within the outline provided by the genetic code, embryonic, fetal, and childhood development of the hip continue while changing to a variety of environmental and biologic factors.

Understanding the sequential steps of the hip's development, along with the growth of its blood supply, is critical to elucidate the pathobiologic mechanisms of hip disease and deformity in the child. Even more critical is the ability to devise and apply rational treatments for pediatric orthopedic diseases that can take advantage of known growth mechanisms. This article discusses the current knowledge of the growth of the normal child's hip from the embryo to adolescence. Abnormal growth of the pediatric hip is then examined through an analysis of two common disease processes and their treatments.

Normal development of the child's hip

Prenatal cellular development

Prenatal human development is separated into an embryonic stage and a fetal stage. The embryonic stage begins when the oocyte is fertilized and ends at approximately 8 weeks postfertilization. During the first three weeks of the embryonic stage, the primitive ectoderm, mesoderm, and endoderm germ layers are formed in the embryonic disc. It is during the fourth to the eighth weeks of development that the majority of joint differentiation is completed [1]. The fetal stage encompasses the period from the eighth week of life to birth. During this period, the limbs and joints undergo growth and maturation in relative proportions and pre-established spatial orientations [1].

Limb formation begins at 4 weeks of development with protrusions of the ventro–lateral wall of the embryo, termed limb buds. The upper limb buds usually appear 2 to 3 days earlier than the lower. Each limb bud consists of an outer ectoderm shell, from which skin, nails, and hair develop, and an inner cellular mass of mesoderm, from which bone, cartilage, muscle, tendon, and the synovial joints arise.

By the sixth week of intrauterine life, the lower limb buds have elongated and now include paddle-like ends termed foot plates (Fig. 1). Vigorous cell multiplication and differentiation occur in the substance of the limb bud. Primitive chondroblasts condense at the proximal, central, and distal ends of the cellular femur template. The club-shaped cartilage model of the future femur follows from these centers through successive chondrification of the precursor cells and fusion of the chondrification centers [1].

The acetabulum begins at 6 weeks as a shallow depression proximal to the head of the femur and is formed by the differentiating precursor cells of the future ilium, ischium, and pubis (Fig. 2). The cartilage model of the acetabulum is formed concurrently with the cartilage model of the pelvic components. Condensations of cartilage cells appear first in the primitive ilium, and then the pubis, and finally the ischium. Chondrification proceeds from these

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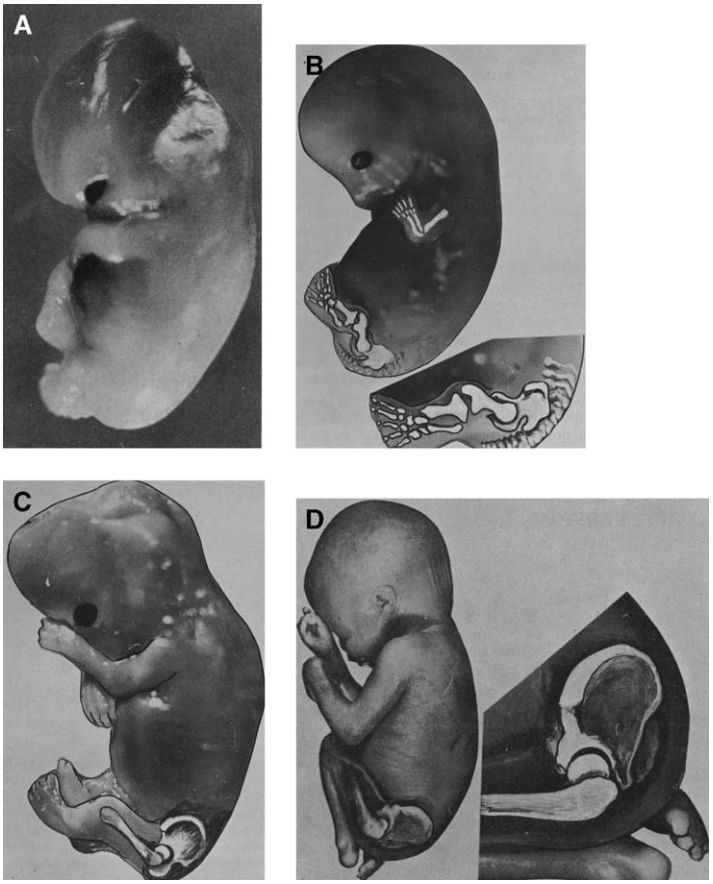

Fig. 1. (*A*) Viewed macroscopically, at the age of 6 weeks, the embryo is 1 cm in size and the limb buds have begun the process of differentiation. Hand and foot plates are visible. (*B*) At the age of 8 weeks, there has been further differentiation of limb buds such that the hip, knee, ankle, and feet are well-formed structures. (*C*) At the age of 11 weeks, there has been a rapid differentiation of the hip joint; and the infantile configuration of the femoral head and acetabulum is now present. (*D*) At the age of 16 weeks, the fetus now measures 100 cm. The lower extremities lie in a position of stability for the fetal hip joint, specifically flexion, adduction, and external rotation. (*Adapted from* Watanabe RS. Embryology of the human hip. Clin Orthop 1974;98:8,12,14,20; with permission.)

centers toward each other until fusion occurs. The chondrification centers fuse quickly, with the ilium and ischium joining first, followed by the ilium and the pubis. The pubis and ischium centers are the last to fuse, closing during the seventh week, and leave a small opening laterally, corresponding to the apex of the developing acetabular fossa [2]. Note that differentiation of the acetabulum, especially the ilium, lags behind that of the femoral head and shaft at all stages.

By the seventh week of gestation, the cartilaginous model for both the femur and acetabulum are complete. The mass of primitive cells between the femoral and acetabular cartilage models now undergo apoptosis to yield a fluid-filled cleft, the beginning of the future hip joint [2]. Theoretically, this stage is the earliest time in development during which a hip dislocation may occur [2].

By the eighth week of development, at the transition from the embryo stage to the fetal stage, the primary ossification center of the femur now appears in its shaft. Ossification proceeds proximally and distally from this center. The soft tissue components of the hip have begun to take shape also. A grouping of cells adjacent to the femoral head identifies the future site of the ligamentum teres and is continuous with the condensation of cells marking the future transverse acetabular ligament inferiorly. The ligamentum teres is defined as the joint space expands and is seen to attach to the medial border of the acetabular fossa, separating from the transverse

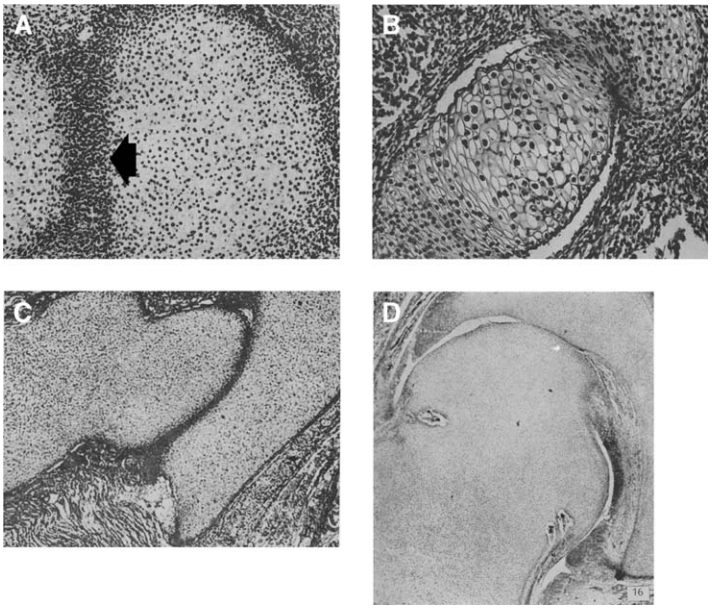

Fig. 2. (*A*) Viewed microscopically, at 4 weeks, condensation of the cells in the interval between the primitive femoral head and the acetabulum occurs to form the hip joint. (*B*) At 7 weeks, the femoral head has a spherical contour; and the acetabulum develops rapidly around it. Note the beginning of a joint cavity at this stage. (*C*) At 8 weeks, the early cartilage model of the acetabulum and femoral head has begun to form. The majority of hip structures, including the labrum, are identifiable at this stage. (*D*) At 11 to 14 weeks, blood vessels are observed to enter through the small lacunae hollowed out of the head and neck. The labrum along the periphery of the acetabulum and the ligamentum teres of the femur are visible. (*A–C adapted from* Watanabe RS. Embryology of the human hip. Clin Orthop 1974;98:10,11; with permission, and *D adapted from* Strayer Jr LM. Embryology of the human hip joint. Clin Orthop 1971;74:236; with permission.)

acetabular ligament to lie behind it. The acetabular labrum is observed at 6 weeks as a condensation of cells lying along the periphery of the developing acetabulum that rides the enlarging rim of the ilium, ischium, and pubis out over the femoral head. By the eighth week, it has begun to assume its triangular configuration in coronal section. The joint capsule and synovia may be distinguished at 8 weeks as a layer of cells lying under the muscular primordia and over the acetabular labrum, joining the perichondrium of the femur below.

Although hip differentiation continues until approximately 20 weeks of development, the major anatomic structures of the hip are identifiable microscopically by the eighth week. Passage into the fetal stage at this point is characterized by a shift from differentiation to growth and maturation of the hip. By 11 weeks, all portions of the hip are visible macroscopically, and the infantile configuration of the hip joint is achieved. The femoral head is formed fully with spherical contour, short femoral neck, and a primitive greater trochanter (see Fig. 2D). A well-defined capsule is present, as are the acetabular labrum and transverse ligament. The hip can be actively dislocated at this time.

At 16 weeks, the ossification of the femur is complete up to the level of the lesser trochanter. The primary centers of ossification have appeared in the ilium, ischium, and pubis, although the acetabular ossification centers do not appear until adolescence [2]. The hip joint space is now completely formed, and the articular surfaces are covered with mature hyaline cartilage. All of the muscle structures are mature, and active motion of the extremities can now be observed.

Limb position, femoral anteversion, and neck–shaft angle

At 4 weeks, the embryo has begun to show slight flexion at the knee [2]. During the eighth week of development, the lower limb begins rotating internally to direct the flexed knee anteriorly and completes the rotation by the end of the embryonic stage. At 11 weeks, the hip and knee are flexed and the leg is adducted. By 16 weeks, further flexion

occurs at the hip and knee; and the left leg is noted to slightly overlap the right leg. With continued growth and accommodation of the developing fetus to a closed space, further flexion of the fetal hip and knee occur until the fetal position is assumed [2].

Femoral anteversion is first able to be discerned at 11 weeks and measures 5° to 10°. Jouve and colleagues [3] studied 87 femurs from 44 formalin-preserved fetuses and demonstrated a wide variability in anteversion position at each fetal age, especially during the first half of fetal life, but noted an increase with increasing fetal age, measuring on average 45° at 36 weeks. Femoral anteversion then decreases in postnatal development. Fabry and colleagues [4] studied 432 healthy children (864 hips) and developed a normal baseline of femoral anteversion at each age from 1 to 16 years. The mean angle of femoral anteversion was 31.1° at age 1 year, decreasing to 15.4° by age 16 years [4].

Neck–shaft angle in fetal development appears to decrease with fetal age, ranging from approximately 145° at 15 weeks to 130° at 36 weeks [3]. Following birth, the neck–shaft angle progressively decreases with age. Zippel [5] studied 400 children (800 hips) and developed a normal baseline of neck–shaft angle at each age from 1 to 20 years. The mean neck–shaft angle at age 1 year was 136.2°, whereas at age 18 years, the value dropped to 127.3°.

The mechanism for femoral anteversion and neck–shaft angle formation in the fetal period remains elusive. Initial speculation that anteversion results from the normal internal rotation of the lower limb during development is countered by the observation that femoral anteversion changes long after the completion of limb rotation. Watanabe [2] noted that excessive internal rotation or external rotation of the fetal limb in a specimen was associated with excessive femoral anteversion or retroversion, respectively. He theorized that the proximal femur position is likely related to the muscular forces that act on the hip during prenatal development.

The argument that muscular forces across the hip joint influence femoral anteversion and neck–shaft angle is more cogent in the postnatal hip. In a cross-sectional study of 267 hips in 147 patients who had cerebral palsy between the ages of 2 and 18 years, Bobroff and colleagues [6] demonstrated radiographically that femoral anteversion remained relatively constant at each postpartum age in the cerebral palsy group, whereas it decreased in the historical control group. Further, patients who had cerebral palsy were noted to have a markedly increased neck–shaft angle at each age as compared with historical controls. The difference is presumably the result of muscle spasticity and soft tissue contracture about the hips of patients who have cerebral palsy.

Cell signaling in intrauterine joint development

The formation of the joint is a complicated sequence of cellular events that involves the creation of mesenchymal aggregates, the condensation of mesenchymal precursors on either side of the joint with the formation of a less dense interzone, and cavitation of the interzone to form the future joint space. The ossification of the cartilage anlage of the long bone occurs simultaneously. Advances in molecular techniques have allowed the bone morphogenetic proteins (BMPs), a family of secreted cell-signaling molecules with key roles in development, to be identified as the crucial components of joint formation. An understanding of the synergistic and antagonistic roles of the myriad known signals for ordered joint formation remains obscure, however.

Growth and differentiation factor 5 (GDF-5) is a much-studied BMP necessary for proper joint development. GDF-5 is localized as stripes at the future sites of joints in mouse embryos [7,8]. GDF-5 is expressed in almost all of the developing joints of the limb from early stages of cellular condensation to joint cavitation [9,10]. When creating a null mutation of GDF-5 in mice, a brachypodism (short-limbed mutant) phenotype results where the appendicular skeleton is shortened and the formation of approximately 30% of the joints of the limb is disrupted [9]. The relevance of GDF-5 to human joint development is clear when considering the skeletal dysplasias that result from mutations in the human homologue CDMP1, including Hunter-Thompson-type acromesomelic chondroplasia, autosomal dominant brachydactyly type C, and Grebe-type chondrodysplasia [11–13].

A solution to the message for joint formation lies not in defining the function of the various molecular signals, but rather in their complex interaction. Examination of the BMP inhibiting protein noggin exemplifies this complexity. In mice homozygous for the noggin loss of function allele, a single cartilaginous limb element results with no joint formation (Fig. 3) [8]. The implication is that unchecked BMP activity disturbs the patterning of the joint. The concept is reinforced by examining the activity of BMP-7, which has been shown to be highly expressed in the perichondrial cells of the avian embryo surrounding condensing chondrocytes and promotes cartilage formation [7]. BMP-7 is absent in the cell

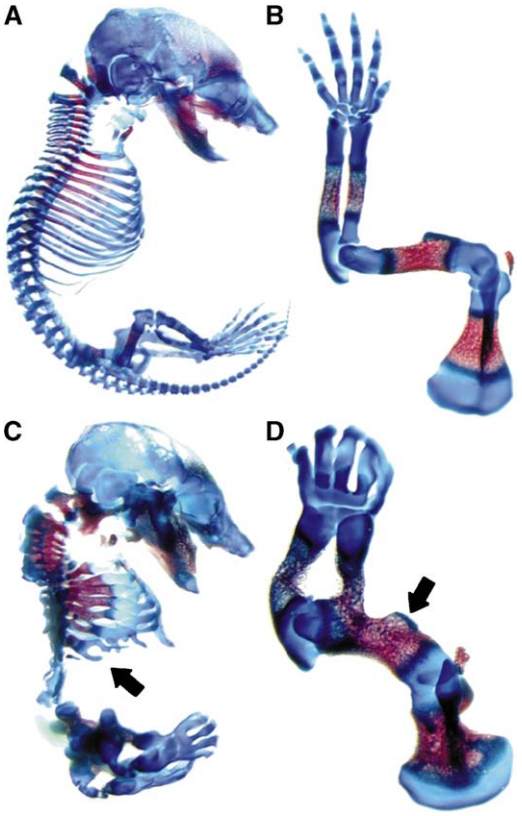

Fig. 3. Skeletal abnormalities in noggin homozygous mouse mutants. Skeletons, with forelimbs removed, from wild-type (*A*) and mutant (*C*) embryos 18.5 days postcoitum were stained blue for nonmineralized cartilage and red for mineralized cartilage and bone. The forelimbs are shown in (*B*) and (*D*), respectively. In (*C*), the solid arrow points to multiple rib deformities and fusions. Note the failure of formation of the spine, knee, ankle, hindfoot, and midfoot. In (*D*), the solid arrow points to continuous ossification from the radius to humerus. Note the failure of formation of elbow and carpus. (*Adapted from* Brunet LJ, McMahon JA, McMahon AP, et al. Noggin, cartilage morphogenesis, and joint formation in the mammalian skeleton. Science 1998; 280(5368):1456; with permission.)

condensations marking the future joints. When beads secreting BMP-7 are implanted into these condensations, joint formation is inhibited [7]. The mechanisms that clear BMP-7 expression at the sites of joint development are therefore crucial for normal joint development.

The temporal expression of various protein elements further complicates the understanding of cellular joint morphogenesis. Each stage of development may be thought to contain a specific cell-signaling milieu. The action of each signal does not occur in a vacuum, but is modified by the presence of other signals. To exemplify, BMPs can induce death or differentiation depending on the stage of development. Application of excessive BMP-2 or BMP-7 beads to the developing chick limb bud before mesenchyme condensation induces apoptosis and a subsequent loss of skeletal elements [7]. If the BMP-2 and BMP-7 beads are applied two days later, cartilage formation is induced and the limbs resemble those of noggin mutants. BMP-2 and BMP-7 activity is changed completely by other proteins either present or absent at different times of development. This "context dependency" of signal action is an important general principle in the development of the vertebrate limb and provides an underpinning for understanding the role of various molecules [14].

Postnatal development of the child's hip

Acetabular development

At birth, the acetabular cartilage complex consists of the saucer-shaped acetabular cartilage laterally and the Y-shaped triradiate cartilage medially (Fig. 4). These two components of the acetabular cartilage complex are continuous, and their coordinated growth results in the final acetabular shape [15]. Eventually the triradiate cartilage will form the nonarticular medial wall of the acetabulum; and the acetabular cartilage will form the cup-shaped rim of the acetabulum.

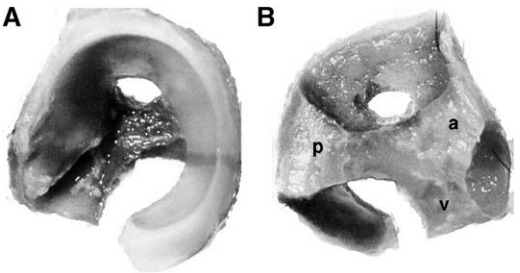

Fig. 4. Lateral view (*A*) and medial view (*B*) of the normal acetabular cartilage complex of a one-day-old infant. The ilium, ischium, and pubis have been removed with a curette. The lateral view shows the cup-shaped acetabulum, and the medial view shows the three flanges of the triradiate cartilage. The anterior flange (*a*) is located between the ilium and pubis and is slanted superiorly; the posterior flange (*p*) is horizontal and located between the ilium and ischium; the vertical flange (*v*) is located between the pubis and ischium. (*Adapted from* Ponseti IV. Growth and development of the acetabulum in the normal child. Anatomical, histological, and roentgenographic studies. J Bone Joint Surg Am 1978;60(5):576; with permission.)

The acetabular cartilage complex is composed of mostly hyaline cartilage. The hyaline cartilage is covered by growth plate cartilage at all areas where it lies adjacent to the bony pelvis and by articular cartilage at all the points of contact with the femoral head. The labrum forms the outer margin of the acetabulum, increasing its relative depth, and is made of fibrocartilage.

The acetabular cartilage complex is an epiphysis and develops in much the same way as the iliac crest and the epiphysis of long bones with the appearance of secondary ossification centers. Three main acetabular ossification centers develop in the acetabular cartilage in humans. The os acetabuli is the largest and forms in the cartilage contributed by the pubis. It is the functional epiphysis of the pubis, as it is separated from the pubis by a growth plate. The os acetabuli initially occupies the anterior part of the acetabular floor and eventually forms the anterior wall of the acetabulum. The iliac acetabular cartilage center forms the superior acetabular bone and joint surface. The ischial acetabular center, the smallest of the three, develops to form the posterior acetabulum. All ossification centers appear by 8 to 9 years of age and fuse by 17 to 18 years of age. Since most of the acetabular shape is determined by 8 years, this age is important for prognosis in many pediatric hip disorders [16,17].

The growth of acetabular height and width depends on the interstitial growth of the triradiate cartilage. Growth in depth and the construction of the final acetabular shape, however, heavily depends on the interaction with a spherical femoral head. When the femoral heads in growing rats were excised or dislocated, Harrison [18] found that the acetabular socket failed to develop in depth and there was atrophy and degeneration of the articular cartilage, while the triradiate cartilage remained histologically normal. The acetabulum requires the spherical femoral head as a template about which it forms. In fact, the condition of proximal focal femoral deficiency punctuates the interplay between the developing femoral head and the developing acetabulum. Presence in part of the proximal femur allows development of an acetabulum. Complete absence of the proximal femur yields an absent acetabulum [1].

Proximal femoral development

The ossification of the cartilaginous femoral shaft proceeds proximally during the fetal stage to reach the greater trochanter and femoral neck at birth. The cartilage template of the proximal femur that has not been replaced by bone defines three growth plates: the longitudinal growth plate of the neck (LGP), the greater trochanteric growth plate (TGP), and the femoral neck isthmus (FNI) [19] (Fig. 5). These three growth plates work concurrently to support the longitudinal growth of the femur and to develop the shape of the proximal femur. Of note, the growth plate of the lesser trochanter and its iliopsoas attachment are poorly studied but do not appear to influence the growth of the proximal or distal femur.

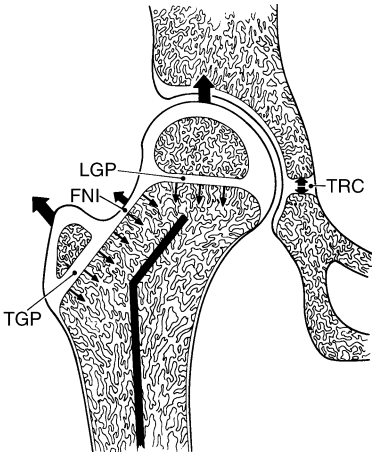

Fig. 5. Development of the hip during infancy and childhood occurs by proliferation of growth cartilage (unshaded) of the acetabulum and the proximal femur. The acetabulum grows appositionally through growth of the articular cartilage and interstitially through growth of the triradiate cartilage (TRC). The head of the femur and greater trochanter enlarge by appositional growth. The three growth zones of the proximal femur are: the longitudinal growth plate (LGP), the trochanteric growth plate (TGP) and the femoral neck isthmus (FNI). (*Adapted from* Siffert RS. Patterns of deformity of the developing hip. Clin Orthop Relat Res 1981; Oct(160):16; with permission.)

The LGP anatomically lies within the head of the femur during infancy and initially contributes to the maintenance of its sphericity. As the neck elongates, the geographic center of the head moves proximally until the LGP achieves its final position at the junction of the femoral head and neck. The LGP grows proximally and medially, contributing to the longitudinal growth of the femur and neck, as well as to the lateral width of the femoral neck.

The TGP lies at the base of the cartilage template of the greater trochanter. Like the LGP, it contributes mainly to the longitudinal growth of the proximal femur and to the lateral width of the femoral neck. Similarities emerge when examining the relationship

of the greater trochanter and the femoral head to their respective proximal femoral growth plates. Both the greater trochanter and femoral head enlarge through appositional growth of their cartilage precursors with subsequent ossification; however, their final positions in space relative to the femur and each other are determined by the proximal femoral growth plates upon which they rest. The greater trochanter is forced proximally and laterally by the TGP, and the femoral head is forced proximally and medially by the LGP.

The FNI is a small cartilage isthmus spared by ossification that connects the trochanteric and femoral neck plates along the lateral border of the femoral neck. The FNI dynamically contributes to the lateral width of the neck, keeping pace with the TGP and LGP. Since no growth in femoral neck width occurs along the medial border of the neck, the varus and valgus angulation of the neck is controlled by the contributions to the lateral growth of the neck by the three growth plates.

The dynamic relationship of the LGP, TGP, and FNI can be examined in terms of growth vectors. The TGP and FNI have growth vectors that are oriented divergently with respect to the LGP. All three growth plates are at an angle relative to the long axis of the femoral shaft. Concurrent function of the three growth plates yields not only growth along their respective axis, but also a common vector of growth directed along the axis of the femoral shaft. A disturbance in any one of these growth plates can lead to angular abnormalities of the proximal femur. Further, growth rates may be controlled by altering the position of the growth plate relative to the femoral shaft. For example, during infancy and childhood, the LGP is relatively horizontal and perpendicular to the long axis of the femur. As the TGP and FNI growth rates increase toward adolescence, the LGP begins to tip medially, allowing for a constant rate of longitudinal growth and allowing a more medially directed vector to balance proximal growth of the femoral neck. A disruption of this normal growth pattern is seen in Ogden type II avascular necrosis, where the FNI and lateral aspect of the LGP suffer a vascular insult. The femoral head tips into a valgus position as growth stops along the lateral aspect of the LGP and continues along the medial.

Just as a located femoral head is necessary for acetabular development, it is also necessary for correct femoral head development. The contact pressures exerted on the femoral head cartilage by the tight-fitting acetabulum result in its spherical appositional growth, as increasing pressure inhibits growth. Likewise, the pressure exerted by the femoral head on the acetabulum is critical to the achievement of the complementary acetabular shape. Proximal femur and acetabulum development are inextricably linked to achieve the end goal of a congruent joint.

Development of the arterial supply to the child's hip

The origin of the arterial organization of the child's hip may be divided into the development of the vessels along the femoral and acetabular sides. The arterial supply to the proximal femur begins its development with the appearance of the primary ossification center in the femoral shaft during the eighth week of development. Capillaries break through the periosteum at the middle third of the femoral shaft cartilage template, at the level of the nutrient artery in the adult femur, and carry fibroblastic and hematopoeitic cells into the marrow. For many weeks in development, it is the only intraosseous blood supply in the entire femur [1]. By 12 to14 weeks of development, a ring of vessels has begun to form around the neck of the femur, consisting of the future medial and lateral circumflex vessels, the obturator, and the superior and inferior gluteal vessels. At this point, blood vessels connected to this ring have invaded the cartilage model of the head and neck of the femur; and capillary tufts form along the neck of the femur at the future site of the retinacular vessels.

The entry of blood vessels into the acetabulum occurs just after the entry of blood vessels into the head and neck of the femur during weeks 12 to14 of development [1]. However, the ligamentum teres and the fibrofatty tissue filling the acetabular fossa, known as the Haversian gland or the pulvinar, have evidence of capillary invasion by 8 weeks. The significance of the vasculature at these two sites for further growth and development of the hip is questionable. Strayer [1] notes that only one of seven large fetuses examined showed vessels entering the femoral head from the ligamentum teres and that vessels from the ligamentum teres invade the head only after ossification is well under way. No anastomosis with the distal arterial terminals in the femoral head was observed until around age 15, when ossification of the head is nearly complete [20]. The findings suggest that the artery of the ligamentum teres does not offer a significant contribution to the blood supply of the developing femoral head. In fact, removal of the ligamentum teres during open reduction of a dysplastic hip does not result in adverse growth consequences.

The organization of the blood supply to the proximal end of the femur established during pre-natal development endures throughout the growth of the child. Chung's perfusion study [20] of 150 proximal

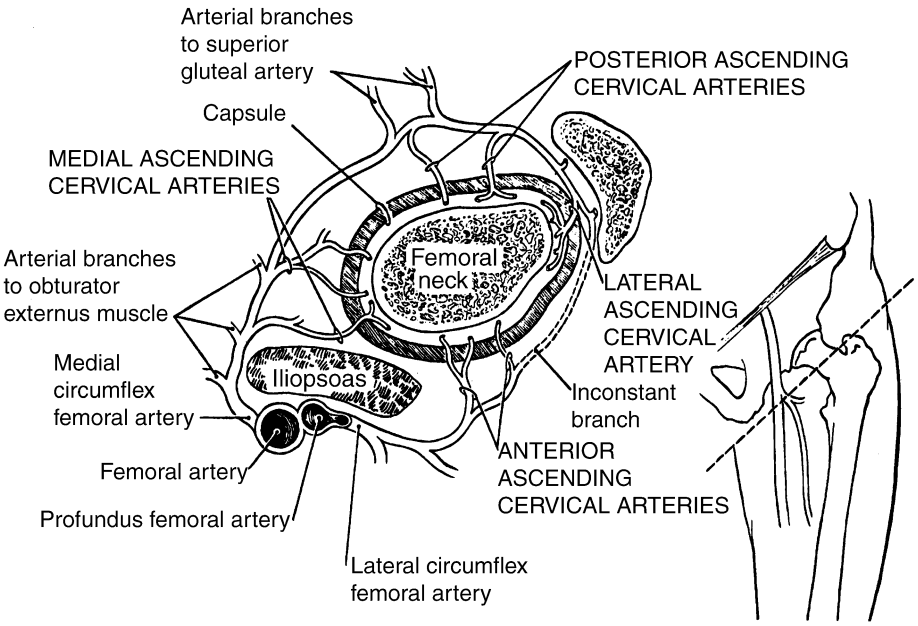

Fig. 6. Cross-section of proximal part of the left femur at the base of the neck, showing the extracapsular arterial ring. Broken lines indicate inconstant connections between anterior and lateral ascending cervical arteries. The lateral ascending cervical artery branches after traversing the capsule. In young children, it lies in the narrow space between the femoral neck and greater trochanter. (*Adapted from* Chung SM. The arterial supply of the developing proximal end of the human femur. J Bone Joint Surg Am 1976;58(7):964; with permission.)

femurs from autopsied fetuses and children aged 26 weeks to 14 years demonstrated that the vessel configuration largely maintains the final adult anatomy during development.

The proximal femur arterial supply in a growing child consists of (1) an extracapsular arterial ring (Fig. 6), (2) intracapsular ascending cervical arteries (Figs. 7–9), and (3) an intracapsular subsynovial ring (Fig. 10). The extracapsular arterial ring rests at the base of the femoral neck and is formed by the union of branches from the medial and lateral circumflex arteries. From the extracapsular ring, thin ascending cervical or retinacular vessels pierce the hip capsule and travel in a subsynovial, intra-articular location along the femoral neck toward the head. Four groups of such vessels are identified and named based on anatomic location relative to the femoral neck: lateral, posterior, medial, and anterior. Branches from the ascending cervical arteries pierce the neck of the femur and travel distally to the metaphysis to follow one of three fates. First, these branches may turn laterally and supply the greater trochanter. Second, they may anastomose with the ascending nutrient vessels from the femoral shaft. Finally, they may turn medially and supply the femoral neck.

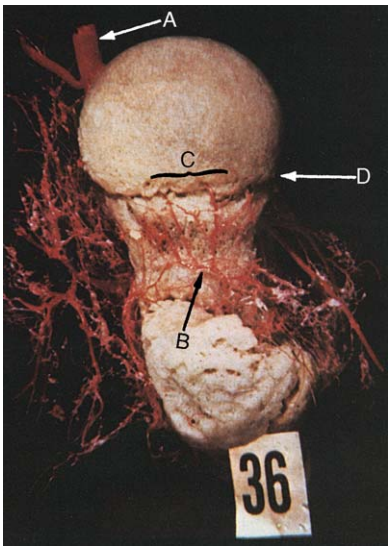

Fig. 7. Superolateral view of proximal part of the left femur from a 14.67-year-old boy perfused with Baton's medium. The femoral artery (*A*), extracapsular ring (*B*), ascending lateral cervical arteries (*C*), and physeal plate (*D*) are demonstrated. (*Adapted from* Chung SM. The arterial supply of the developing proximal end of the human femur. J Bone Joint Surg Am 1976;58(7):964; with permission.)

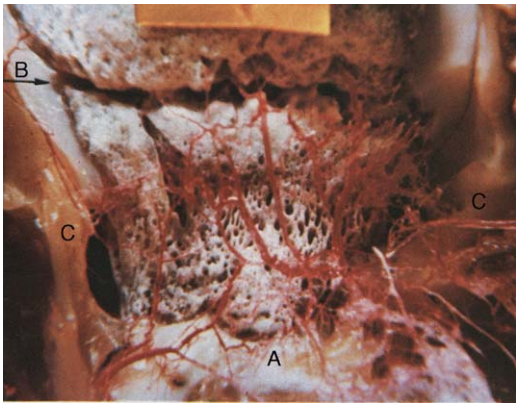

Fig. 8. Close-up of the same specimen as shown in Fig. 7 detailing the lateral ascending cervical arteries. Note the capillaries to the greater trochanter (A). The physeal plate (B) and the capsule (C) are visible as well. (*Adapted from* Chung SM. The arterial supply of the developing proximal end of the human femur. J Bone Joint Surg Am 1976;58(7): 964; with permission.)

The ascending cervical vessels unite more proximally at the junction of the femoral neck and the articular cartilage of the head to form an intra-articular subsynovial arterial ring anastomosis. This ring is sometimes incomplete, more often in males,

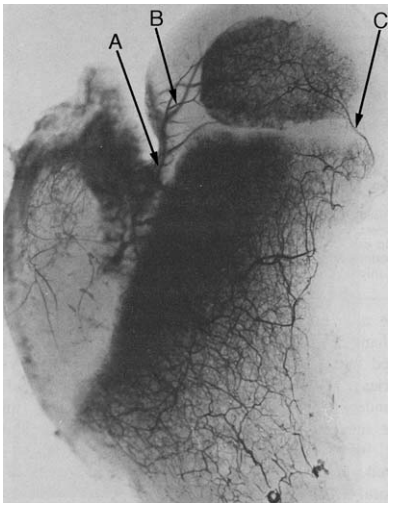

Fig. 9. Anterior half of proximal part of the right femur, perfused with barium sulfate and then divided in the coronal plane, from a 40-month-old white boy. The lateral ascending cervical artery (A) and the epiphyseal branches of the lateral (B) and medial (C) ascending cervical arteries are seen to pass through the perichondrial ring and not the physeal plate. (*Adapted from* Chung SM. The arterial supply of the developing proximal end of the human femur. J Bone Joint Surg Am 1976;58(7):964; with permission.)

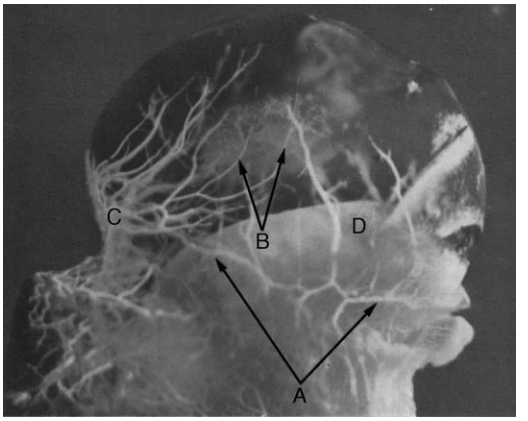

Fig. 10. Anterior view of the right femoral neck, perfused with barium sulfate, from a 9-month-old girl. The intracapsular subsynovial ring (A) is visualized. Multiple vessels are seen to supply the ossification center (B). Numerous branches of the lateral ascending cervical arteries (C) are seen to skirt the edge of the epiphysis and do not cross through the metaphysis (D). (*Adapted from* Chung SM. The arterial supply of the developing proximal end of the human femur. J Bone Joint Surg Am 1976;58(7):967; with permission.)

and is typically more robust on the medial and lateral surfaces of the femoral neck than the anterior and posterior [20]. From here, epiphyseal and metaphyseal branches ensue. The epiphyseal branches cross the physeal plate by skirting the perichondrial ring superficially and then enter the cartilage of the developing femoral head. The metaphyseal branches pierce the femoral neck and travel distally.

It is commonly assumed that the clinical importance of the pelvic and acetabular blood supply may be far less than that of the proximal femur, given the wide range of intraosseous and extraosseous anastomosis. Since the adult blood supply is established by the postnatal course, an understanding of the configuration allows the surgeon to avoid at least a theoretic incidence of acetabular avascular necrosis and growth arrest during pelvic osteotomy. The configuration of the vessels about the acetabulum may be understood if the acetabulum is divided into sectors as in the face of a clock (Fig. 11). At approximately 10 o'clock to 4 o'clock, branches of the superior gluteal artery supply the acetabular dome; at 4 o'clock to 8 o'clock, the posterior branch of the obturator extends nutrient arteries to the inferior acetabular bone; at 8 o'clock to10 o'clock, a branch of inferior gluteal artery provides nutrient acetabular branches [22]. Despite the division into sectors, a rich extraosseous and intraosseous anastomosis exists between

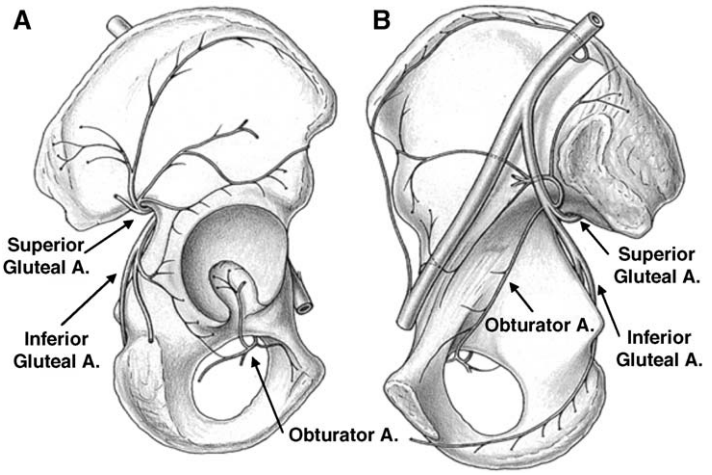

Fig. 11. Lateral (*A*) and medial (*B*) views of the right acetabulum with its associated vascular supply. Note the clockwise arrangement of the superior gluteal artery, obturator artery, and inferior gluteal artery contributions to the acetabulum. (*Adapted from* Beck, et al. The acetabular blood supply: implications for periacetabular osteotomies. Surg Radiol Anat 2003;25:365; with permission.)

these vessels. The redundancy of the vascular supply may explain why avascular necrosis of the acetabulum is rare after pelvic osteotomy, even when done at a young age. For example, bilateral pelvic osteotomies for bladder extrophy repair rarely have untoward results for acetabular growth.

The configuration of the blood vessels about the proximal femur in the child brings to light two important differences between the vascular anatomy of the child and the adult hip. One difference is the presence of a growth plate. From birth to the time of physeal plate closure, the plate is a vascular barrier and no vessels cross it. The ascending cervical vessels access the femoral head by coursing along the perimeter of the growth plate. Note that an extraosseous anastomosis still exists between the intraosseous microcirculation of the head and neck through the subsynovial arterial ring. After physeal plate closure, the metaphyseal vessels penetrate into the epiphysis, and the vascular systems communicate along intraosseous routes [21].

Second, when the cervical arteries penetrated into the cartilaginous femoral head during growth, independent vascular territories were initially defined. In later growth, these territories appear to coalesce into a large anastomotic network. It is speculated that this network is not complete, and occlusion of specific ascending arteries may cause necrosis of previously defined autonomous vascular zones.

Finally, the medial circumflex artery and its terminal end, the lateral portion of the extracapsular arterial ring, provide the majority of the blood supply to the femoral head, neck and greater trochanter [20]. As the child grows, the contribution of the medial circumflex artery assumes greater significance, because the number of cervical arteries contributed by the lateral circumflex artery decrease in development. Chung [20] found an approximately 50% reduction in the number of vessels along the anterior and medial aspect of the femoral neck when comparing children from 0 to 2 years of age and children from 3 to 10 years of age. The number of lateral and posterior ascending cervical arteries, derived from the medial circumflex branch, remained constant. Lauritzen [23] also confirmed that by age 10 the lateral retinacular arteries begin to dominate the blood supply to the head and neck of the femur.

Abnormal development of the child's hip

Developmental dysplasia of the hip

Developmental dysplasia of the hip (DDH) is a disease process that encompasses a spectrum of anatomic hip abnormalities in the newborn ranging from mild dysplastic acetabular change to complete teratologic (antenatal) dislocation. The incidence of DDH is reported as 1 to 1.5 per 1000 live births, but 1 out of 100 newborns may have some evidence of "hip instability" [24].

The causes of DDH are multifactorial, including both genetic and mechanical factors. Genetic factors are illustrated by the increased incidence of the

disease in patients of female gender, a positive family history, or a particular ethnic background, such as North American Indian and Laplander. Mechanical factors are illustrated by the increased risk in patients who experienced breech presentation, oligohydramnios, or other ''crowding'' conditions.

Pivotal to determining etiology is whether or not the acetabular dysplasia characteristic of the disease is a result of primary abnormal acetabular development or secondary to intrauterine hip subluxation or dislocation. A primary etiology would suggest a genetic basis for the disease, implicating a failure in cell-signaling pathways and an intrinsic error in hip growth and development. Acetabular dysplasia secondary to hip subluxation would then support mechanical theories as the primary cause of DDH. In this case, as noted previously, the dislocation may not occur until the joint cavity opens at the seventh week of development and may then result from the failure to keep the head pointed at the acetabulum as a result of extrafetal mechanical pressures favoring dislocation. Dislocation may then occur through the posterior–inferior acetabulum, at the site of the future transverse ligament, the weakest point in the labral structure [1]. The deficiency posteriorly and inferiorly corresponds to the postnatal anatomic abnormality in the hip. Since acetabular depth in the fetus is directly correlated with head size, the fetal acetabulum also becomes shallow [25].

Late diagnosis of DDH and its associated anatomic abnormalities illustrates many of the mechanical growth principles of the child's hip and the necessary interaction between femoral head and acetabulum. Two scenarios may be considered—persistent lateral hip subluxation and dislocation (Fig. 12). In the case of lateral hip subluxation, the pressure on the femoral head becomes concentrated along the medial aspect of the head as the hip hinges along the edge of the acetabulum. Likewise, concentric pressure on the acetabular floor is reduced while it is increased along the lateral edge. Since pressure inhibits appositional growth, the lateral femoral head continues to grow and flattens the head. The acetabular growth cartilage fills the acetabular floor and arrests its lateral growth, forming a progressively more shallow and oblique acetabulum. In the case of complete hip dislocation, both the head and acetabulum have unrestricted growth, yielding a large

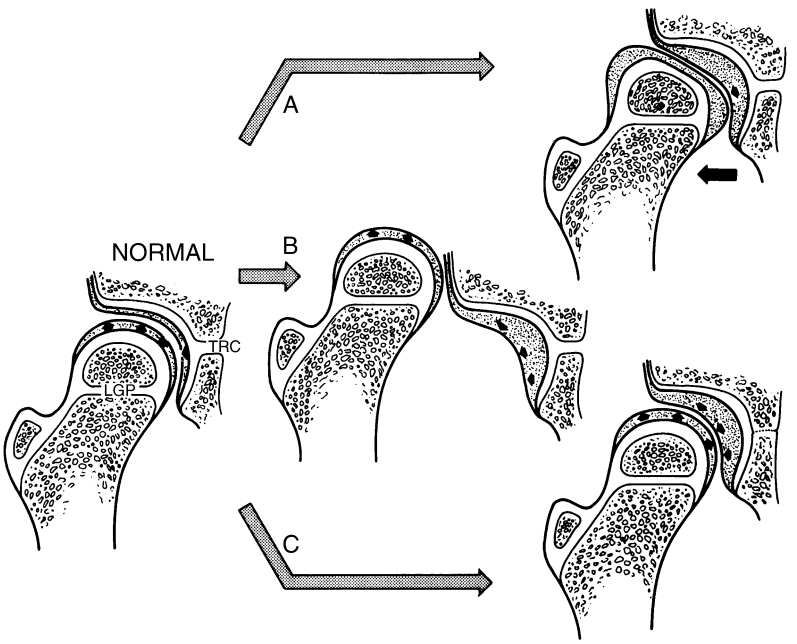

Fig. 12. The normal hip in child development is shown with the femoral head closely associated with the acetabulum to yield a congruent joint. When this relationship is lost, different acetabular deformities result. (*A*) depicts the deformity from lateral hip subluxation, (*B*) depicts the deformity from gross hip dislocation, and (*C*) depicts the deformity from early closure of the triradiate cartilage. (*Adapted from* Siffert RS. Patterns of deformity of the developing hip. Clin Orthop Relat Res 1981;Oct(160):20; with permission.)

head and a shallow acetabulum. With enough time, the femoral head no longer fits the socket and prevents reduction.

The treatment of DDH involves restoring the contact between femoral head and acetabulum. As the child ages, reduction of the dislocated or subluxated hip becomes increasingly difficult, often requiring closed or open procedures under general anesthesia. Restoration of normal acetabular development becomes less likely as the child ages; acetabular morphology is mostly determined by the age of 8 years [16,17].

Operative treatment of DDH often involves open reduction with pelvic and femoral osteotomies and offers another opportunity to apply principles of child hip development. Since the femoral head is typically large and the acetabulum is shallow in a subluxated or dislocated hip, it is thought that pelvic osteotomies that redirect rather than reshape the acetabulum are preferable. A reshaping osteotomy, such as the Pemberton, would decrease the already small acetabular volume and make difficult the maintenance of a reduction. Pelvic osteotomies may also disrupt the continued growth of the acetabulum (see Fig. 5). The operative procedure may cause closure of the triradiate cartilage. As a result, the medial wall of the acetabulum fails to grow and the acetabular articular cartilage cannot expand. A small, shallow acetabulum results, with a progressively subluxating femoral head.

In addition, varus femoral osteotomies for DDH offer an interesting study in proximal femoral growth dynamics. Loss of correction in varus osteotomy is commonly observed and is correlated to the age of the patient, with patients less than 4 years old showing the greatest loss in correction [26]. The remodeling of the varus proximal femur is related to the persistence of the growth plate and the dynamic reorientation of the physis with growth. Further, varus osteotomy may pose a risk to the greater trochanteric growth plate, leading to late deformity. Schofield and Smibert [27] reported an 18.8% reoperation rate for late valgus deformity in 11 of 14 patients in whom the greater trochanteric physis was violated during varus osteotomy.

Legg-Calvé-Perthes disease

Legg-Calvé-Perthes disease (LCPD) describes the apparently idiopathic progressive collapse and deformity of the femoral head in young children. LCPD is most common between the ages of 4 and 8 years, but may be seen in children as young as 2 years and as old as the early teens. It is more common in boys by a ratio of 4:1; and its international annual incidence is 1 in 1200 [24]. The etiology and late hip deformities of LCPD are particularly instructive on the growth and development of the child's hip.

The cause of LCPD is likely multifactorial, but most theories return to the tenuous vascular supply of the proximal femoral epiphysis. The peak incidence of LCPD between 4 to 8 years of age corresponds to specific anatomic vascular development peculiarities, as noted in the previous section from Chung's studies [20]. First, the epiphyseal and metaphyseal branches of the lateral ascending cervical artery, contributing the bulk of the blood supply to the proximal femoral epiphysis, originate from a single vessel that crosses the capsule at the trochanteric notch. Because the space between the trochanter and femoral neck is very narrow in children under eight years old, this single artery is vulnerable to occlusion by compression through hip positioning or unidentified exogenous sources [20]. Next, fewer arteries are present along the anterior and medial femoral neck in specimens from 3- to 10-year-old children than from newborn to 2-year-old children. Finally, the subsynovial intracapsular arterial supply is more often incomplete in males than in females, and may explain why LCPD is more common in boys. Blood supply to the epiphysis from the ligamentum teres likely does not impact the disease course of LCPD, because this supply is not related to age, sex, or race [20].

The pattern of venous drainage of the proximal femur has not been discussed in the vascular development of the proximal femur, but it has been implicated as a causative factor of LCPD. Heikkinen and colleagues noted abnormal venous patterns in 46 of 55 hips during the initial and fragmentation phases of LCPD [28]. Green and Griffin [29] demonstrated impaired patterns of venous outflow in 23 patients who had LCPD when compared with 23 normal hips. Liu and Ho [30] demonstrated delayed venous emptying from the femoral neck in 32 patients who had unilateral LCPD and reproduced lesions histologically similar to LCPD in skeletally immature dogs using an intraosseous injection of silicone into the femoral neck to impair venous outflow.

The altered growth of the femoral head in LCPD illustrates the deformity that may result with alterations in the longitudinal growth plate (LGP) and epiphyseal growth plate of the femoral head. Premature physeal plate closure occurs with LCPD and may be located either centrally or peripherally. In both cases, trochanteric overgrowth occurs. In central growth arrest, however, a short neck results without significant angular deformity. In lateral growth arrest, the femoral head and growth plate are tilted exter-

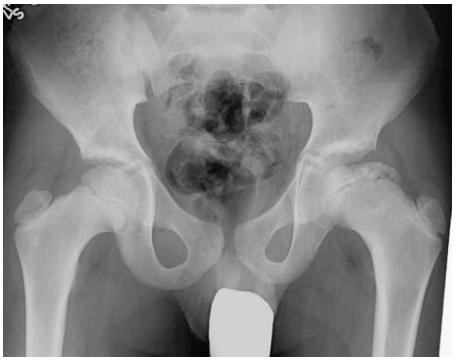

Fig. 13. Anteroposterior pelvic radiograph of a 9-year-old boy who has Legg-Calvé-Perthes disease, demonstrating the late deformity of a wide femoral neck, flattened and overgrown femoral head, and corresponding acetabular irregularity.

nally. Further, the deepest layer of articular cartilage ceases to grow, because it derives its nourishment from the epiphyseal blood vessels. The more superficial layer, which derives its nourishment from the synovial fluid, continues to grow and ossify, leading to a coxa magna. The FNI grows throughout the process and may yield a wide femoral neck (Fig. 13). With progressive deformity of the femoral head and neck, the femoral head is no longer containable; and motion is allowed only in the flexion and abduction plane. The lateral aspect of the acetabulum and the femoral head may be deformed secondarily by hinged abduction.

As in DDH, age and growth potential weigh heavily on the ability to restore a congruent joint. Young children with the disorder often demonstrate complete remodeling of the epiphysis. Even in cases of coxa magna, surgical containment procedures in this patient population can promote congruent development of the femur and acetabulum with respect to each other. On the other hand, children older than 8 years typically have a worse prognosis.

Summary

Child hip development is an ordered pathway of processes that results from the delicate interplay of cellular and mechanical influences. Critical to hip development is cell signaling, intrauterine differentiation and growth, and postnatal growth with maturation of its accompanying blood supply. Central to this process is the necessary contact between femoral head and acetabulum for congruent development. The sequelae of multiple disease processes and their treatments, including DDH and Legg-Calvé-Perthes disease, may be understood in the context of the growth history of this critical joint.

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