TYPES OF PERSISTENT DYSPLASIA IN CONGENITAL DISLOCATION OF THE HIP

S. M. DOYLE, J. R. BOWEN

This article reports five types of persistent bony dysplasia in patients with congenital dislocation of the hip (CDH), suggests the pathogeneses, and discusses the treatment options. We consider the five types to be (1) maldirected acetabulum, (2) capacious acetabulum, (3) false acetabulum, (4) lateralized acetabulum, and (5) femoral deformity. The maldirected acetabulum persists when the acetabulum continues to face forward and laterally. The capacious acetabulum arises from joint instability ; capsular laxity permits the proximal femur to slide within the acetabulum. The false acetabulum results from an ectopic fibrocartilaginous cavity in the pelvis created by the subluxated or dislocated femoral head.44 The lateralized acetabulum occurs with ossification of the cotyloid cavity from longstanding lateral subluxation or dislocation or premature closure of the triradiate cartilage. Femoral deformities include valgus and anteversion of the femoral neck, capital femoral physeal growth arrest, discrepancy between the greater trochanter and the femoral head, and femoral head asphericity. After clinical and radiographic evaluation, we believe that an understanding of the pathogeneses and types of dysplasia will facilitate appropriate treatment programs. Treatments consist of acetabular redirection, acetabular reconstruction, femoral osteotomies, and salvage procedures.

Keywords : hip ; dysplasia ; congenital dislocation.
Mots-clés : hanche ; dysplasie ; luxation congénitale.

INTRODUCTION

The residual bony dysplasia that persists in congenital dislocation of the hip involves the femoral head, acetabulum, or both. In dysplasia, the hip joint forms normally in embryonic organogenesis, but deforms progressively throughout fetal development (44). Dysplasia refers to maldevelopment of the hip joint, which alters the congruency of the femoral head and acetabulum. The incongruency may range from subluxation to complete dislocation (15). The purpose of this chapter is to analyze hip dysplasia and suggest treatments. We classify dysplasia into five types : (1) maldirected acetabulum, (2) capacious acetabulum, (3) false acetabulum, (4) lateralized acetabulum, and (5) femoral deformity.

NORMAL DEVELOPMENT AND GROWTH OF THE HIP

At four weeks gestation, the lower limb buds emerge. In the proximal and central portion of the lower limb bud, there is a condensation of uniform, densely packed cells that will develop into the cartilage model of the hip. By eight weeks, the cartilage model divides creating an acetabulum and femoral head ; and by twelve weeks, the musculoskeletal components of the thigh, the articular cartilage, and the hip joint are well formed (44). The acetabulum continues to enlarge throughout gestation by means of labral and triradiate growth (42). At birth, the normal femoral head is deeply seated in the acetabulum. Further growth of the hip joint is interdependent between the femoral head and acetabulum. Whereas interstitial growth within the triradiate cartilage is

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responsible for increasing the hip joint diameter, labral growth is responsible for increasing the acetabular depth (27, 46). The orientation of the acetabulum in the final three months of intrauterine life and at birth is more anteverted than in adult life ; this stems from the flexed and abducted position of the hips (18). Growth of the proximal femur occurs from a chondrophysis that develops into a confluent physis with three regions : the proximal femoral physis, the greater trochanter, and the femoral neck isthmus (35). These ossification centers of the hip fuse by sixteen to eighteen years of life (27).

DYSPLASIA OF THE HIP

In dysplasia, the hip joint is thought to form normally in embryonic organogenesis, but is thought to deform progressively throughout fetal development. Dysplasia refers to maldevelopment of the hip joint that alters the congruency of the femoral head and acetabulum (28, 44). The interdependence of the femoral head and acetabulum, required for normal hip joint development, is violated. Prior to discussion of our proposed five types of dysplasia (maldirected acetabulum, capacious acetabulum, false acetabulum, lateralized acetabulum, and femoral deformity), we will consider the primary and secondary etiologic factors of dysplasia.

The primary factors of hip dysplasia are determined genetically (positive family history). The incidence of dysplasia increases with patients who have a positive family history, an ethnic background, such as Lapps and Native Americans, female gender, ligamentous laxity, or syndromes, such as Ehlers-Danlos or Larsen. Some newborns with a dislocated hip have persistent dysplasia, in spite of timely successful reduction. This observation supports a genetically transmitted or primary cause of congenital hip dislocation (22, 44, 47, 50, 51).

The environment determines the secondary factors of hip dysplasia. Examples of this include hip malpositioning and avascular necrosis. Hip malpositioning is influenced by post natal swaddling positions or intrauterine compression caused by oligohydramnios, multiple fetuses, first pregnancy, and breech position (5, 44, 47, 50). Several studies support that acetabular dysplasia is a secondary adaptive defect, and is the result, not the cause of congenital dislocation of the hip. Still born babies with congenital dislocation of the hip have minimal dysplasia and concentrically reduced hips demonstrate a reversal of acetabular dysplasia (12, 20, 36, 44).

Primary and secondary factors together may produce hip dysplasia. Hip instability may be due to excessive capsular laxity or insufficiency (12). This ligamentous laxity may be hereditary or mechanical. Generalized familial joint laxity is a recognized risk factor in CDH that is inherited as a dominant trait with incomplete penetrance (52). Experiments performed in puppies demonstrate mechanical instability. When the capsule and ligamentum teres were excised during growth or the capsule was stretched, hip dislocation or dysplasia resulted. Incomplete hip reduction after attempted treatment of CDH by open or closed procedures may also cause residual hip dysplasia, by violating the interdependent growth of the femoral head and acetabulum (20, 37).

TYPES OF DYSPLASIA

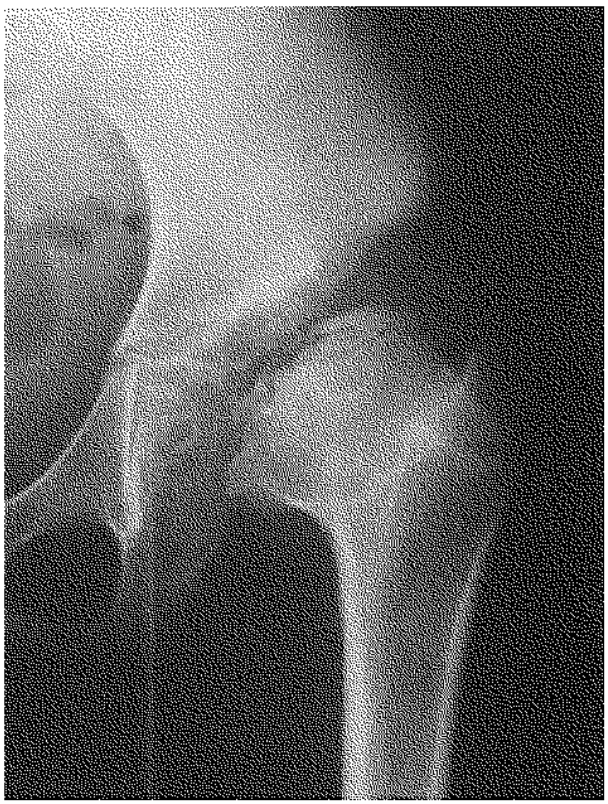
We classify dysplasia into five types : (1) maldirected acetabulum, (2) capacious acetabulum, (3) false acetabulum, (4) lateralized acetabulum, and (5) femoral deformity. The maldirected acetabulum persists when the acetabulum continues to face forward and laterally (fig. 1). With hip dislocation, there is a lack of corrective forces exerted by the proximal femur to redirect the acetabulum to its proper downward facing position (16). If the acetabulum remains in this maldirected orientation, after the hip joint is reduced, the femoral head will be inadequately covered anteriorly and laterally with hip extension and adduction (31). In addition, growth stimulation diminishes with the lack of concentric pressure between the femoral head and acetabulum, and the acetabulum becomes permanently thickened, shallow, and oblique (44).

The capacious acetabulum arises from joint instability. Impairment of labral growth reduces

Fig. 1. — Maldirected acetabulum : This form of dysplasia persists when the acetabulum continues to face forward and laterally. Note the lack of anterior femoral head coverage.

the depth of the acetabulum (27, 46). Whereas the normal configuration of the acetabulum is like a hemispherical cup, the abnormal shape of the capacious acetabulum is like a shallow saucer (fig. 2). Capsular laxity permits the femoral head to slide within the acetabulum, which reinforces the existence of an enlarged, open acetabulum that is incongruous with the femoral head. Persistence of this disproportionate relationship of the hip joint produces further instability (12, 44).

The false acetabulum is an ectopic fibrocartilaginous cavity in the pelvis created by the subluxated or dislocated femoral head. The mechanical stimulation from the superiorly displaced femoral head on the lateral ilium produces fibrocartilaginous tissue generated by fibroblasts, which forms into the false acetabulum. When the hip is subluxated or dislocated and can no longer be reduced, there are several obstacles to reduction. These obstacles include the inverted limbus, ligamentum teres, fibrofatty pulvinar, and iliopsoas tendon. When the transverse acetabular ligament is stretched superiorly with the capsule, it hypertrophies and obstructs the inferior acetabulum. Then pelvifemoral muscles become shortened and contracted, which provides another barrier to hip joint reduction (21, 44).



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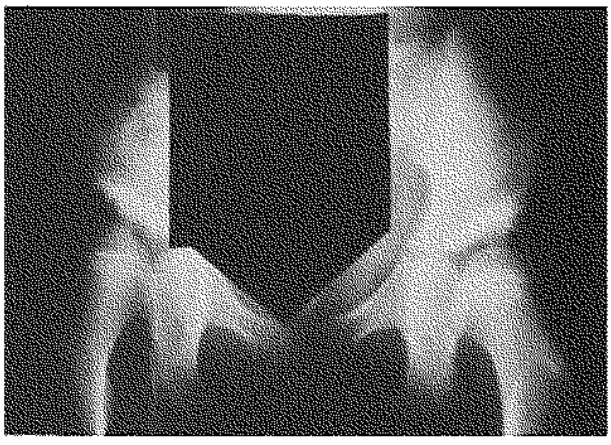

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Fig. 2a & b. — Capacious acetabulum : This form of dysplasia results from an impairment of labral growth, which reduces acetabular depth. The shape of the acetabulum is like a shallow saucer.

The lateralized acetabulum occurs with widening of the medial acetabulum and ossification of the cotyloid cavity from longstanding lateral subluxation or premature closure of the triradiate cartilage. Incomplete reduction of a dysplastic hip with persistent lateral subluxation allows thickening of the medial acetabular wall demonstrated radiographically by a wide teardrop. There is a loss of normal contour between the femoral head and acetabulum due to the lateral position of the femoral head. The chronicity of the femoral head position laterally may lead to hypertrophy of the soft tissue structures lining the cotyloid cavity of the acetabulum. The medial wall ossifies reducing the depth of the acetabulum, which insures persistence of the lateral position of the femoral head (fig. 3). Premature triradiate closure decreases the width and diameter of the acetabulum (27, 28). Premature triradiate closure may result from infection of the hip or iatrogenic injury to the triradiate cartilage during reconstructive treatment (7, 13, 15).

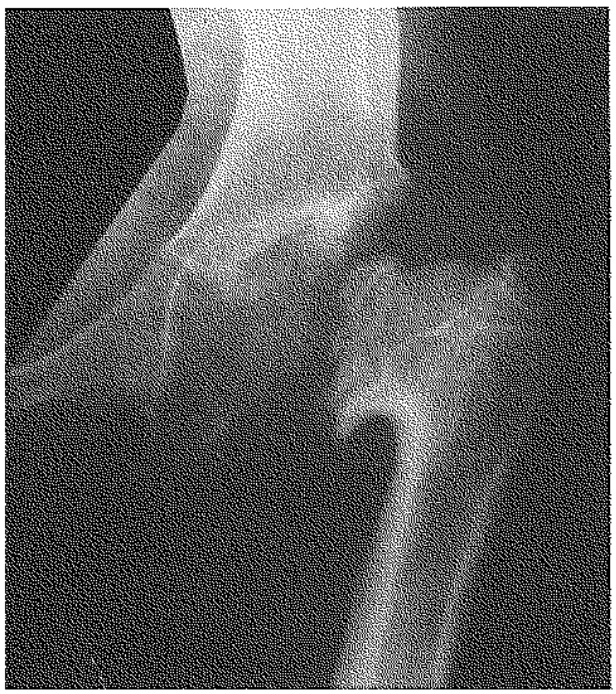

Fig. 3. — Lateralized acetabulum : This form of dysplasia occurs with widening of the medial acetabulum and ossification of the cotyloid cavity from longstanding lateral subluxation or premature closure of the triradiate cartilage.

We consider femoral deformity as a type of dysplasia. Deformities which stem from the proximal femur may be one of the following : valgus and anteversion of the femoral neck, capital femoral physeal growth arrest, discrepancy between the greater trochanter and the femoral head, or femoral head asphericity (fig. 4a and 4b) (13). Increased femoral anteversion and coxa valga may be present to a varying degree in CDH. The resulting persistence of anteversion and coxa valga is a significant factor in instability of hip joint reduction (48). Disturbance in proximal femoral growth from avascular necrosis may occur after treatment efforts. Kalamchi introduced a classification system to evaluate the different types of vascular changes involving the femoral head and capital femoral physis and to predict the natural history of avascular necrosis. Capital femoral physeal growth arrest may be located in the lateral or central region of the physis. Damage to the lateral physis causes a "tether" and results in valgus deformity of the head and neck. The valgus tilt of the femoral head may lead to inadequate acetabular coverage. Central physeal arrest causes shortening of the femoral neck. The relative overgrowth of the greater trochanter results in a discrepancy between the greater trochanter and the femoral head leading to hip abductor weakness and limb length inequality. Femoral head asphericity occurs with damage to the entire head and physis.13 There is femoral head irregularity, flattening, and coxa magna. Incongruity, hence instability, between the femoral head and acetabulum ensues (15).

EVALUATION OF DYSPLASIA

Roentgenograms, arthrograms, ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI) are used to evaluate hip dysplasia. Roentgenograms are helpful in diagnosing bony dysplasia after the femoral ossific nucleus appears, between 4 and 7 months of age. An anteroposterior (AP) radiograph of the pelvis with the hips in approximately 25° flexion is best to negate the anterior pelvic tilt that occurs with neonatal hip flexion contracture (45). After walking age, dysplasia is identified on the standing AP pelvic

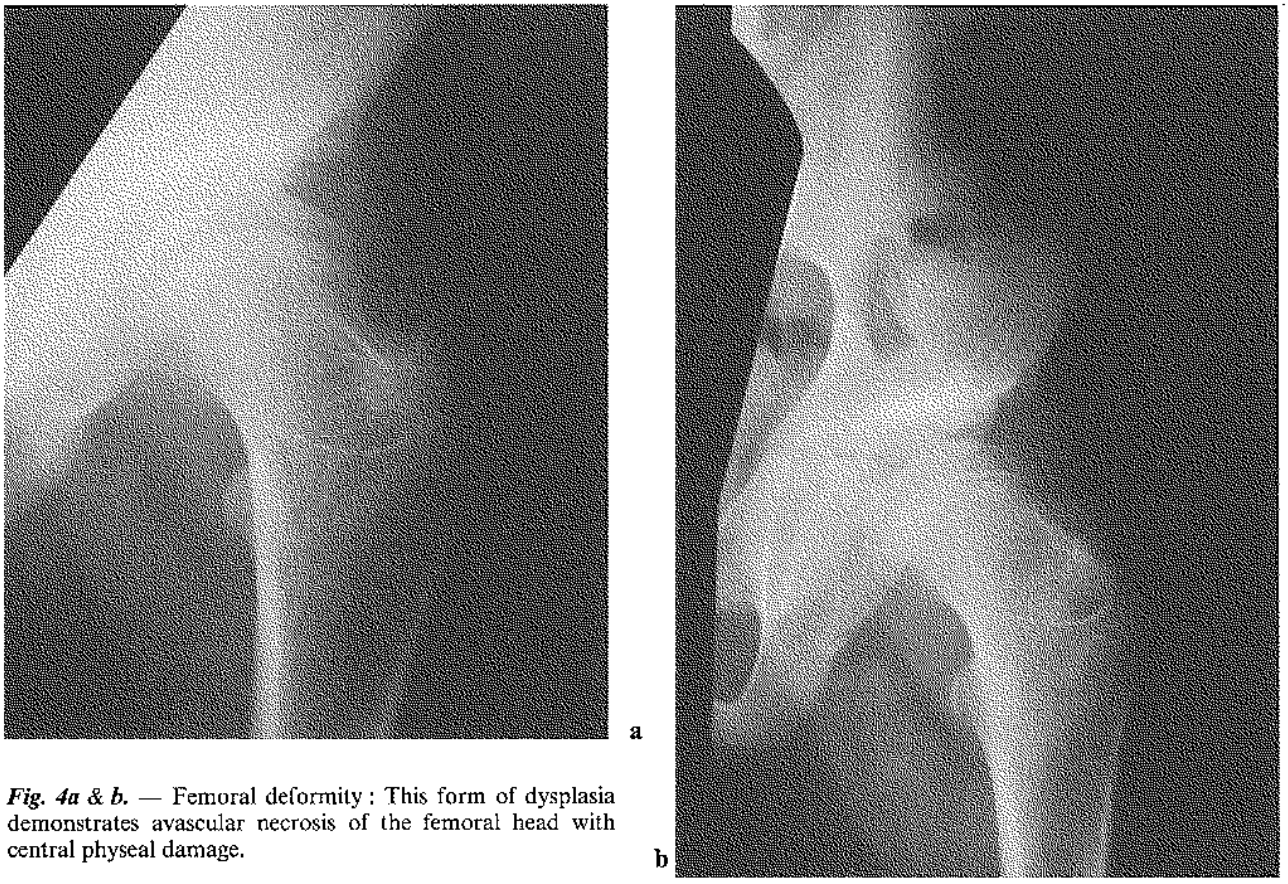

Fig. 4a & b. — Femoral deformity : This form of dysplasia demonstrates avascular necrosis of the femoral head with central physeal damage.

radiograph by the following characteristics : increased acetabular index, decreased center edge angle, increased Sharp's angle, decreased femoral head coverage, distortion of the tear drop, and possibly delayed appearance of the femoral ossific nucleus (29, 34, 47). Whereas the acetabular index is used to measure acetabular development in the child younger than 8 years, the center edge angle is used in older children and adults (44, 49). Once the triradiate cartilage is fused, the acetabular angle of Sharp may be used to measure acetabular dysplasia. Employed for the appropriate age, these measurements help to interpret an abnormal bony configuration of the acetabulum such as that seen in the maldirected, capacious, false, or lateralized acetabulum. Decreased femoral head coverage may be seen in any type of dysplasia. Distortion of the teardrop and increased ossification in the cotyloid cavity, combined with an increased Sharp's angle and decreased coverage of the femoral head, may depict a lateralized acetabulum. The standing false profile, a 65° posteriorly rotated view of the pelvis, helps to identify anterior acetabular lip dysplasia that may be seen in the maldirected or capacious acetabulum (45). Avascular necrosis may be identified as a complication of closed treatment of CDH on the AP pelvic radiograph. Salter's criteria for diagnosing avascular necrosis include : (1) failure of appearance of the femoral head ossific nucleus during one year or longer after reduction, (2) failure of growth in an existing ossific nucleus during one year or longer after reduction, (3) broadening of the femoral neck during one year after reduction, (4) increased radiographic density of the femoral head followed by the radiographic appearance of fragmentation, and (5) residual deformity of the femoral head and neck when reossification is

complete. Changes in femoral growth and configuration may disrupt the congruity of the hip joint required for normal development (29).

Arthrography is a dynamic means of evaluating instability, inadequacy of reduction, and cartilaginous deformities that cause persistent dysplasia. Utilized for patients at any age, arthrography enables the examiner to appreciate incongruity of the hip joint in motion. It also helps to detect irregularities of articular surfaces, not ascertained by other radiographic studies (6, 32). Severin studied the features of the infant hip using arthrography to evaluate hip joint development after closed reduction and to differentiate residual abnormalities of the hip. Results were classified into 6 groups : well developed hip joints, moderate deformity of the femoral head, neck, or acetabulum, dysplasia, subluxation, femoral head articulation with secondary acetabulum, and redislocation (33). Due to the invasiveness of the procedure, arthrography is more likely to be employed as an adjunct to surgical treatment of dysplasia, rather than as a primary method of evaluating the hip joint.

Ultrasound is gaining wide acceptance for evaluation of the newborn hip because it can distinguish cartilage and soft tissue structures that are undetectable by roentgenograms. Since the femoral head ossific nucleus does not appear until approximately 4 months of age, ultrasound is the method of choice for identifying hip position from birth through early infancy. After femoral head ossification, ultrasound becomes a less reliable tool for evaluating the hip joint (4). Ultrasound also is used to assess acetabular configuration. The "dynamic minimum standard exam", devised by Graf and Harcke, uses the coronal and transverse views to establish acetabular landmarks and femoral head location with the hip at rest and with stress during the Ortolani and Barlow maneuvers. Whether treatment for hip reduction is closed (Pavlik harness, overhead traction, or manual reduction) or open, ultrasound is an excellent means of monitoring hip position, stability, and morphologic development after treatment in the infant. Ultrasound will recognize a lack of concentric reduction in the infant hip that may lead to cartilage deformity and persistent dysplasia. The reliability and sensitivity of ultrasound to determine bony dysplasia is not fully established (4, 9, 10, 45).

Computed tomography (CT) and 3D CT is most useful for evaluating bony dysplasia in older patients, however, it does not demonstrate cartilaginous components of the hip joint. It is the best method for evaluation of hip concentricity after closed or open reduction when the patient is in a spica cast (40). Contrary to plain radiographs, CT shows the orientation of the acetabulum, which is useful for identifying the maldirected and capacious acetabuli. In addition, CT shows posterior and anterior dislocation or subluxation of the femoral head and the accompanying false acetabulum (1). When the cotyloid cavity ossifies, as is the case with the lateralized acetabulum, CT provides an excellent assessment of the femoral head and acetabular articulation. Though femoral anteversion can be estimated by physical examination, CT measures it more precisely (26). 3D CT is recommended for demonstrating acetabular morphology to plan and select the appropriate osteotomy for treatment of residual dysplasia.

Magnetic resonance imaging (MRI) illustrates cartilaginous and, to a limited extent, bony components of the hip joint, which helps to assess dysplasia in young children. In the past, the need for patient sedation and expense have precluded its routine use in the evaluation of CDH. Recently, however, support for the use of MRI over CT in evaluation of the hip joint after surgical reduction has been proposed. After operative reduction in CDH, MRI accurately depicts acetabular anatomy and confirms adequacy of reduction without patient exposure to ionizing radiation, as is the case with CT (19). In addition, MRI may be used in conjunction with radiographs to elucidate vascularity of the femoral head in avascular necrosis (9).

TREATMENT OF DYSPLASIA

After clinical and radiographic evaluation, we believe that an understanding of the types of acetabular dysplasia will facilitate the treatment program. The treatment of dysplasia ranges from bracing to surgery. Surgery consists of acetabular

redirection, acetabular reconstruction, femoral osteotomies, and salvage procedures. We divide the treatment of persistent dysplasia into 3 phases : (1) initial, (2) reconstructive, and (3) salvage.

In the initial phase, treatment is aimed at correcting factors that cause persistent dysplasia, such as instability, incongruity, and predictive growth disturbance. In the newborn to 3 month old child, an unstable hip may be recognized by clinical examination (Ortolani positive and/or Barlow positive) and confirmed by ultrasound. The types of dysplasia usually encountered in this phase are the maldirected, capacious, and false acetabuli. If dysplasia is detected, a Pavlik harness or other retentive hip positioning device, such as the Von Rosen splint, Craig splint, Denis Browne hip abduction splint, or Frejka pillow may be applied to maintain the hip joint in a congruent position (24, 44). The brace is employed until the hip is stable via clinical examination and ultrasound (10). Complications with brace utilization consist of avascular necrosis of either femoral head (excessive abduction), femoral nerve palsy or inferior subluxation (excessive flexion), instability of the knee joint (tight harness), and skin breakdown (11, 44). In the infant failing orthotic treatment or the older child, 4 months to 2 years, treatment consists frequently of skin traction, adductor tenotomy, open or closed hip reduction, followed by placement in a spica cast. After closed reduction, the spica cast period is roughly 6 months with cast changes every 6 to 7 weeks. With open reduction, the spica cast period is only 6 weeks. Following spica cast immobilization, subsequent to closed or open hip reduction the patient may be placed in a flexion-abduction orthosis until there is resolution of the acetabular dysplasia. Complications of this treatment include resubluxation or dislocation, due to excessive capsular laxity and/or excessive femoral anteversion, and avascular necrosis (15, 44).

The reconstructive phase involves treatment of dysplasia that has persisted in spite of the corrective efforts attempted in the initial phase of treatment. The goal is to achieve and maintain a concentrically reduced hip joint with adequate acetabular coverage. Patient age and type of dysplasia are the two factors needed to determine which reconstructive procedure should be performed. The Salter innominate osteotomy and variations of the triple innominate osteotomy help to correct dysplasia caused by the maldirected acetabulum. The Salter innominate osteotomy is used in children as young as 18 months to approximately 6 years. This transverse osteotomy of the ilium, located just superior to the acetabulum, is opened anterolaterally by hinging and rotating the acetabular segment on the pubic symphysis. The advantages of using this procedure to correct the maldirected acetabulum are two fold. First, it covers the femoral head with a hyaline cartilage lined acetabulum, which provides appropriate nutrition and load bearing to the joint ; and second, it does not disrupt acetabular growth at the triradiate cartilage or the lateral lip (28, 30, 44). Complications of the Salter procedure include infection, sciatic and femoral nerve palsies, Steinmann pin migration or breakage, medial or posterior displacement of the distal segment, postoperative hip joint stiffness, lateral and upward resubluxation and redislocation, and avascular necrosis (23, 29, 44). In children 7 years or older, the triple innominate osteotomy may be adopted to correct the maldirected acetabulum. Unlike the single innominate osteotomy of Salter, this triple osteotomy of all 3 bones of the pelvis allows greater mobility of the acetabular segment to cover the femoral head. This procedure is utilized in older children because the pubic symphysis ossifies and can not act as the hinge through which rotation and tilting of the acetabular segment can be achieved in the Salter osteotomy (41). Advantages of this procedure are similar to those of the Salter osteotomy. Complications include infection, pressure necrosis of the skin above the displaced anterior superior iliac spine, resubluxation and dislocation, and avascular necrosis. Depending upon the surgeon's preference, the double innominate osteotomy of Sutherland, the modified Salter osteotomy of Kalamchi, the Ganz periacetabular osteotomy, or other variants of acetabular redirection osteotomy may be utilized for similar indications.

Pericapsular innominate osteotomies, such as the Pemberton and Dega acetabuloplasties, help to correct the capacious acetabulum. The Pem-

berton osteotomy depends upon an open triradiate cartilage that will be used as a hinge for rotation ; hence, it can be performed in patients between 2 and 8 years of age. The Pemberton is best used for anterior and lateral coverage. The osteotomy begins between the anterosuperior and anteroinferior iliac spines and extends posteriorly around the acetabulum to the posterior rim of the triradiate cartilage, where it terminates (25). The Dega is best used for lateral and posterior coverage. The osteotomy, located 10 mm superior and parallel to the acetabular roof, extends from the iliopectineal prominence to the posterior limb of the triradiate cartilage posteriorly. Both of these osteotomies create a fulcrum close to the hip joint, which allows a greater degree of correction of acetabular dysplasia and coverage of the femoral head than the innominate osteotomies (43, 44). Penetration through the hip joint articular cartilage or triradiate cartilage may lead to chondrolysis and acetabular growth arrest, respectively. Premature triradiate closure produced by the treatment of the capacious acetabulum may in turn create a lateralized acetabulum. Like the other acetabuloplasties, avascular necrosis of the proximal femur may occur as a complication of treatment (15, 29).

Reconstructive treatment of the false acetabulum is usually performed in conjunction with open reduction of the hip joint and femoral shortening. Lateral and upward displacement of the femoral head may be treated by placement of the patient in preoperative skin traction (14). Obstacles in the true acetabulum, inverted limbus, ligamentum teres, fibrofatty pulvinar, and psoas tendon, need to be removed during open reduction of the hip. Following open reduction an appropriate acetabuloplasty is selected to provide adequate femoral head coverage in the true acetabulum.

A variety of reconstructive procedures are available for treatment of dysplasia due to femoral head deformity. Femoral head deformities include valgus and anteversion of the femoral neck, capital femoral growth arrest, discrepancy between the greater trochanter and the femoral head, and femoral head asphericity. For persistent valgus and anteversion, in the child over 3 years, the Wagner or Lloyd Roberts intertrochanteric oblique osteotomy may be performed to increase hip stability and provide a concentric reduction of the joint, required for normal development. Both of these osteotomies are performed at the intertrochanteric level above the lesser trochanter and the iliopsoas and gluteus maximus insertions ; this will prevent lateral rotation of the hip (anteversion) due to pull of these muscles (43, 44). One must distinguish true from apparent coxa valga ; excessive femoral anteversion may mimic coxa valga on an AP pelvic radiograph. A disadvantage of these varus producing osteotomies is that they result in limb shortening. Capital femoral growth arrest, discrepancy between the greater trochanter and femoral head, and femoral head asphericity are all manifestations of avascular necrosis as described by Kalamchi (13). When there is damage to the lateral physis of the femoral head (Kalamchi group II), there is progressive valgus and shortening of the femoral neck, which leads to limb length discrepancy and relative greater trochanteric overgrowth (fig. 5). Recommended treatment for these deformities is distal and lateral greater trochanteric transfer to restore abductor length and contralateral distal femoral epiphyseodesis to

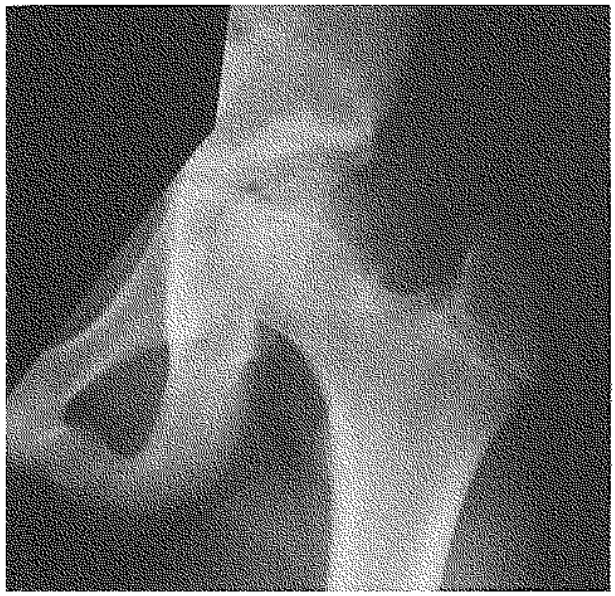

Fig. 5. — Femoral deformity : This demonstrates avascular necrosis of the femoral head with lateral physeal damage. This superolateral growth arrest tethers the femoral head into valgus, which results in a short neck and relative greater trochanteric overgrowth.

equalized limb lengths. With central physeal damage (Kalamchi group III) there is shortening of the femoral neck, relative overgrowth of the greater trochanter, and functional coxa vara. Treatment of central physeal arrest is aimed at restoring hip abductor strength by performing a greater trochanteric apophyseodesis. This procedure is most effective in restoring hip mechanics if it is performed in children less than 6 years (17, 43, 44).

Adolescent and young adult patients with a lateralized acetabulum and/or femoral head asphericity due to avascular necrosis (Kalamchi group IV) or longstanding hip incongruity (coxa magna) are treated often with salvage procedures (8, 15). The salvage procedures include the Chiari medial displacement innominate osteotomy, the Shelf procedure, and rarely hip arthrodesis. In the adolescent and young adult with a lateralized acetabulum, there is an irreducible lateral subluxation or dislocation of the hip, which leads to hip joint incongruity, instability, and subsequent arthritis. Hip joint incongruity and instability are seen also in dysplasia due to an avascular or enlarged femoral head. The Chiari and the Shelf acetabular augmentation provide coverage of the femoral head by enlarging the capacity of the deficient acetabulum, stabilize the hip by preventing superolateral migration of the femoral head, and increase the surface area of the weight bearing portion of the acetabulum (43, 44). The Chiari osteotomy is a form of capsular arthroplasty that enables coverage of the laterally displaced femoral head. After osteotomy of the ilium is performed between the anterior inferior iliac spine and the greater sciatic notch immediately superior to the origin of the hip capsule, the inferior segment of bone is displaced medially. Complications of this osteotomy consist of the following : sciatic nerve palsy (1% incidence), hip arthrosis, narrowing of the pelvic outlet (which may obstruct vaginal delivery of a full-term baby), ipsilateral limb shortening as a result of medial and upward displacement of the acetabulum, and non-union (2, 3). The Shelf procedure extends the roof of the acetabulum laterally, anteriorly, and, posteriorly, by placing iliac crest bone strips in a slot, created along the width of the acetabulum immediately superior to the hip capsule (38, 39). The Shelf procedure does not displace the acetabulum medially ; hence, the hip center of gravity remains lateral, which is biomechanically unsound (43, 44). Hip arthrodesis is a treatment option for the young adult with severe degenerative changes in the hip joint, but in our practice it is seldom performed.

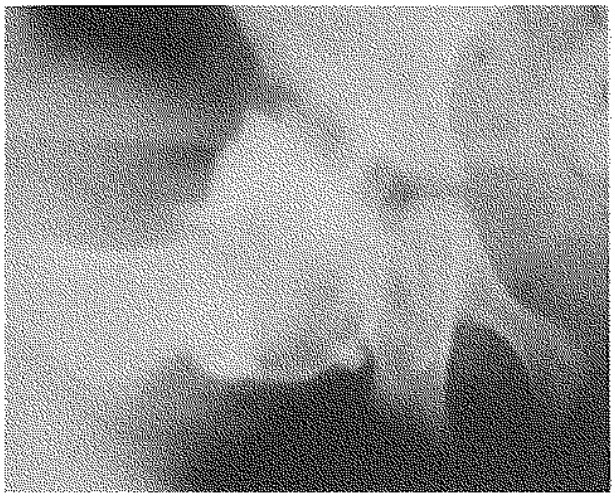

Fig. 6. — Arthrogram : This demonstrates the usefulness of arthrography in evaluating the joint space in a capacious acctabulum.

CONCLUSION

Conscientious long term follow-up is essential in the management of patients with CDH. Persistent dysplasia is potential sequelae in the treatment of CDH and should be recognized early to execute appropriate intervention in a timely manner. We have reported five types of persistent bony dysplasia in patients with CDH. The maldirected acetabulum, capacious acetabulum, false acetabulum, lateralized acetabulum, and femoral deformity are forms of dysplasia seen in young patients that, if left untreated, may manifest later as painful arthritis in adult patients. Subsequent to clinical and radiographic evaluation, a thorough understanding of the pathogeneses and types of dysplasia helps to facilitate a suitable treatment plan.

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SAMENVATTING

S. M. DOYLE, J. R. BOWEN. Types van persisterende dysplasie van de heup.

Dit artikel beschrijft 5 types van persisterende botdysplasie bij patiënten met (sub)luxatie van de heup : 1) maloriëntatie van het acetabulum, 2) te groot acetabulum, 3) vals acetabulum, 4) gelateraliseerd acetabulum en 5) femorale deformatie. Type 1 is wanneer het acetabulum naar voor en lateraal gericht blijft, type 2 ontstaat door de instabiliteit van het gewricht : capsulaire laxiteit veroorlooft de femurkop om te glijden. Type 3 ontstaat door een ectopische lokalisatie door een ge(sub)luxeerde heupkop. Type 4 ontstaat door ossificatie van de cotylaire ruimte door langbestaande laterale subluxatie of premature sluiting van het triradiate kraakbeen. De femurdeformatie bestaat uit valgus en anteversie van de femurhals, groeistop van de capitale groeischijf, discrepantie tussen femurkop en grote trochanter en niet-sfericiteit van de kop. Na klinisch en radiologisch onderzoek, inzicht in de pathogenese zal leiden tot een aangepast therapeutisch beleid bestaande uit acetabulaire re-oriëntatie, acetabulum reconstrucite, femurosteotomie en salvage procedures.

RÉSUMÉ

S. M. DOYLE, J. R. BOWEN. Les types de dysplasie résiduelle dans la luxation congénitale de la hanche.

Cet article présente 5 types de dysplasie osseuse résiduelle qui se rencontrent chez les patients porteurs d'une luxation congénitale de la hanche, il suggère leur pathogénie et discute les options thérapeutiques. Pour nous, ces 5 types sont (1) un vice d'orientation du cotyle, (2) un cotyle trop spacieux, (3) un faux cotyle, (4) un cotyle latéralisé et (5) une déformation fémorale. Le premier type est caractérisé par la persistance d'une orientation du cotyle en avant et en dehors. Le cotyle trop spacieux résulte d'une instabilité articulaire ; la laxité capsulaire permet au fémur proximal de se déplacer à l'intérieur du cotyle. Le faux cotyle résulte d'une cavité fibro-cartilagineuse ectopique crée dans le bassin par la tête fémorale subluxée ou luxée. Le cotyle latéralisé résulte de l'ossification de la cavité cotyloïdienne à la suite d'une subluxation ou d'une luxation externe de longue durée ou à la suite d'une soudure précoce du cartilage en Y. Les déformations fémorales incluent un valgus et une antéversion du col, un arrêt de croissance du cartilage épiphysaire de la tête fémorale, une disharmonie entre le grand trochanter et la tête fémorale et un défaut de sphéricité de la tête fémorale. Après mise au point clinique et radiographique, nous pensons qu'une bonne compréhension de la pathogénie et des types de dysplasie doit faciliter le choix d'un traitement approprié. Les traitements consistent en réorientation du cotyle, reconstruction du cotyle, ostéotomies fémorales et opérations palliatives.