# 5 Acetabular Dysplasia in the Reduced or Subluxated Hip

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## Introduction

As first proposed by Dr. William Harris in the early 1980s, it has become clear that osteoarthritis (OA) of the hip does not exist as a primary disease, or if it does, it is extraordinarily rare [1]. The extensive experience in total hip replacement surgeries over the last four decades have provided extensive insight into the pathological hip joint processes that lead to hip OA [2]. In particular, mechanical pathologies that cause damage to either the labrum or the chondrolabral junction are often the initiating processes which instigate acetabular and femoral head arthritis (Fig. 5.1) [3]. These key observations have stimulated the field of hip preservation, which focuses on determining the mechanisms that cause damage to these structures and the development of surgical approaches that comprehensively correct these pathomorphologies [4–8]. The three pathological processes that lead to injury and degeneration of the labrum and the chondrolabral junction [1, 2] include: dysplasia, impingement and avascular necrosis. The purpose of this chapter is to review the pathologic processes of acetabular and proximal femoral development that lead to acetabular dysplasia in a located, or subluxated, hip. Emphasis will be placed upon understanding how the developmental milestones of the acetabulum influence the surgeon's decision to operate, and if so, whether to perform an acetabuloplasty, redirectional, salvage or replacement procedure.

> ***"Mechanical pathologies that cause damage to either the labrum or the chondrolabral junction are often the initiating processes which instigate acetabular and femoral head arthritis"***

## Pathophysiology

Normal anatomical development of the hip requires coordinated growth of both the acetabulum and proximal femur that provides essential femoral head coverage and clinical stability by the time the child is skeletally mature [9]. Normal acetabular development results in a congruent hip joint with approximately 15° of acetabular anteversion and sufficient coverage of the femoral head. At skeletal maturity, an intact hip joint is stable during weight bearing and physiologic range of motion, without inappropriate stresses on the chondrolabral junction. Normal proximal femoral development results in a spherical femoral head with approximately

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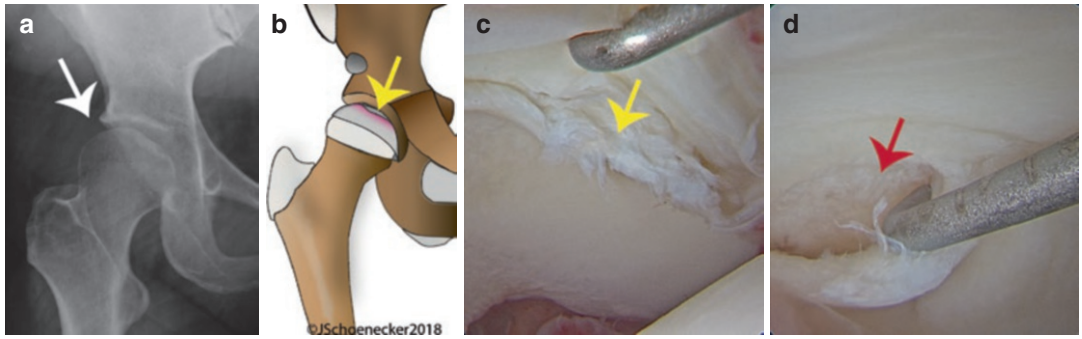


**Fig. 5.1** Prevailing theory of osteoarthritis of the hip. Mechanical pathology leads to labral pathology and hip osteoarthritis. (**a**) Radiographs of a mildly dysplastic hip in 34-year old female with right hip pain (white arrow represents position of patient chondrolabral junction). (**b**, **c**) Years of improper loading of the hip results in tearing of the labrum or the chondrolabral junction (yellow arrow). (**d**) Progression of the tear, or increased pathologic loading leads to arthritis of the femoral head or the acetabulum (red arrow)

15° of femoral version and the tip of the greater trochanter at the level of the center of the femoral head. This normal hip development allows for joint congruency during physiologic activities, with physiologic motion of 90° of flexion, 15° of internal rotation at 90° of flexion and a 90° arc of rotation with equal internal/external rotation (45°) in the prone position. Proper femoral neck offset and alignment of the greater and lesser trochanters assures both normal muscle tension and force vectors for hip rotation while avoiding extracapsular impingement on the pelvis during physiologic joint motion. Adequate acetabular coverage, proper acetabular and proximal femoral version and 15° of external tibial torsion, results in a stable base to support the torso over the limb with a foot-forward gait during single leg stance.

Hip dysplasia with a located, or subluxed, femoroacetabular joint is associated with inappropriate development of either the acetabulum, proximal femur or both. Either alone or in combinations, the resulting pathomorphologies cause damage to the labrum/chondrolabral junction and articular cartilage, leading to premature degeneration of the hip joint [4–8]. Acetabular undercoverage of the femoral head produces instability of the femoroacetabular joint. In addition to damaging intracapsular structures of the hip, intracapsular/extracapsular impingement of the femur on the acetabulum or pelvis causes restricted motion and pain [10]. During ambulation, hip dysplasia may result in excessive stress on the labrum and/or chondrolabral junction (Fig. 5.2), abnormal abductor muscle tension (with gait disturbance), and malalignment of the limb leading to distal joint pathologies. With these in mind, surgical approaches have been designed to preserve the hip joint, addressing the pathological mechanical problems presented by these processes. The goals of hip joint preservation surgery is to restore stable acetabular coverage of the femoral head and achieve a near normal range of motion, without femoral acetabular/pelvic impingement. Proximal femoral dysplasia may occur from primary developmental pathologies, such as proximal femoral focal deficiency, coxa valga/vara or excessive version. It can also develop secondarily, from diseases such as Legg-Calve-Perthes disease, slipped capital femoral epiphysis or avascular necrosis. These conditions are presented in detail in other chapters. Acetabular dysplasia may occur primarily from failure of development or secondarily from improper loading by the proximal femur. The morphologic characteristics are dependent upon the stage of acetabular development at the time of insult.

Acetabular development (Fig. 5.3) is a dynamic process of endochondral ossification involving the cartilaginous anlage that includes two essential growth centers: the triradiate

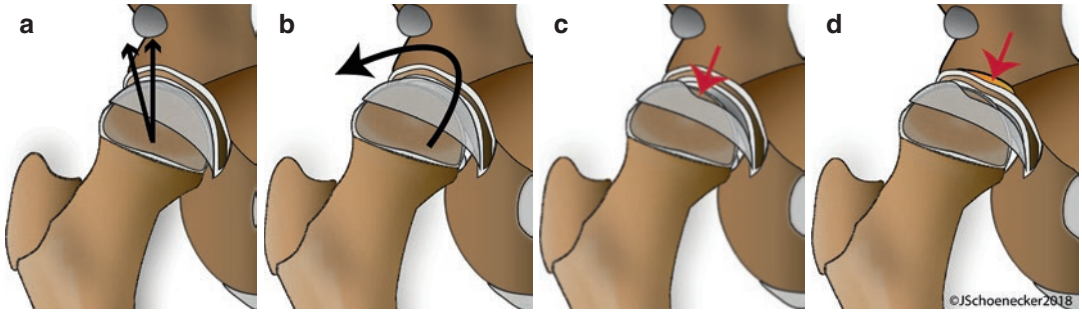


**Fig. 5.2** Micro-instability of the dysplastic hip damages the chondrolabral junction. Hip dysplasia with a located, or subluxed, femoroacetabular joint refers to inappropriate development of either the acetabulum, proximal femur and or both. (**a**) Angle arrows represent insufficient lateral center edge angle (LCEA) of the ossifying acetabular epiphysis. (**b**) The resulting pathomorphologies leads to micro-instability of the femoroacetabular joint during daily activities. (**c**) This instability ultimately leads to damage of the (**c**) labrum/chondrolabral junction (red arrow) or even (**d**) fracture of the acetabular epiphysis (red arrow). Ultimately, this damage leads to premature degeneration of the hip joint

cartilage and the acetabular epiphysis (os acetabuli) [11–13]. During the first 4 years of life the majority of acetabular development occurs via biomechanical molding, with sequential ossification of the acetabular cartilaginous anlage and radial growth of the acetabulum (by the triradiate cartilage). The shape of the acetabular cartilaginous anlage is primarily influenced through direct contact (articulation) with the femoral head. The femoral head must be stably reduced in the true acetabulum for optimal acetabular growth and development. The cartilaginous anlage is considerably plastic during the cartilaginous phase and becomes much less plastic later on, following vascular invasion and subsequent ossification. The triradiate cartilage is responsible for growth of acetabular width, which must match the growth of the femoral epiphysis. In a normal hip, the majority of the cartilaginous anlage has ossified by 4 years of age [12, 13]. After 4 years of age, triradiate cartilage growth continues to widen the developing acetabulum to accommodate a larger proximal femoral epiphysis [12, 13]. Triradiate cartilage growth is typically complete by 12 years of age in girls and 14 years of age in boys [12, 13]. Starting around age 4 years, and continuing to skeletal maturity, an increase in acetabular depth occurs secondary to growth of the acetabular epiphysis. This growth is essential in providing adequate coverage of a hip in response to the increased size of the femoral epiphysis [12, 13].

Acetabular dysplasia can therefore be broken down to three categories: (1) improper shape and/or delay in ossification of the cartilaginous anlage, (2) damage to the triradiate cartilage, or (3) problems of shape and/or delayed ossification of the acetabular epiphysis. Problems of shape and/or delay in ossification of the cartilaginous anlage occur early in life. As the cartilaginous anlage is considerably plastic, malformation most commonly occurs as a result of eccentric loading of the proximal femur. This results in an altered hip center which is typically superiorly migrated, observed as a "break in Shelton's line" on a weight bearing AP pelvis with femoral version neutralized (Fig. 5.4). Severe subluxation may cause more significant morphologic changes to the cartilaginous anlage and resemble a dislocated hip on radiographs. Additionally, inappropriate biomechanical loading may also delay the vascular invasion and ossification of the cartilaginous anlage which is observed as an abnormal acetabular index (AI) (Fig. 5.5). Although the cartilaginous anlage has a significant capacity to remodel, it cannot do so without restoration of the appropriate hip center. Additionally, there is no evidence that once an anlage has undergone a morphologic change in the hip center (broken Shelton's line) that it is capable of restoring its normal shape, and hip center,

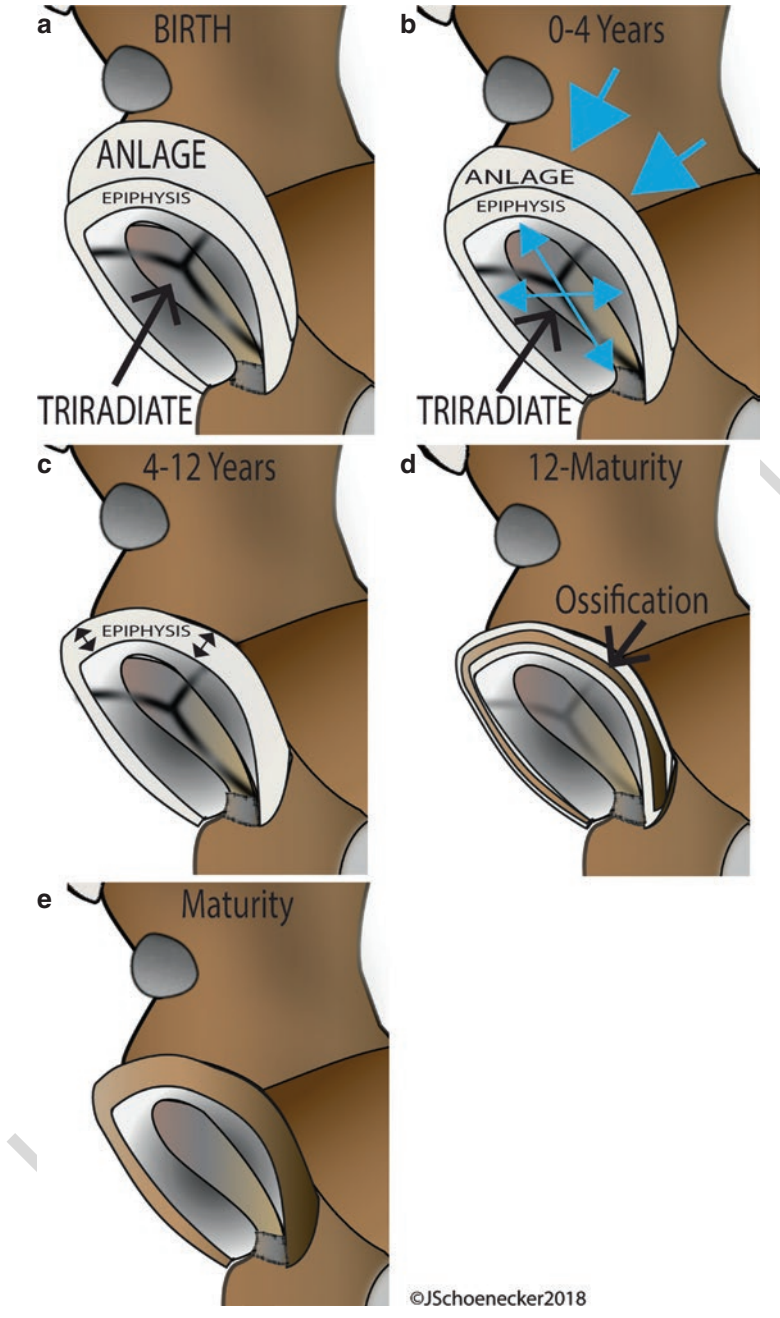


**Fig. 5.3** The dynamic process of acetabular development. (**a**) Acetabular development is a dynamic process of endochondral ossification that occurs through the cartilaginous anlage that includes two essential growth centers: the triradiate cartilage and the acetabular epiphysis (os acetabuli). (**b**) During the first 4 years of life, the greater majority of acetabular development occurs through the biomechanical molding and sequential ossification of the acetabular cartilaginous anlage (big blue arrows) and the radial growth of the acetabulum by the triradiate cartilage (small blue arrows). The triradiate cartilage is responsible for growth of acetabular width, which must match the growth of the femoral epiphysis. (**c**) After 4 years of age, triradiate cartilage growth continues to widen the developing acetabulum to accommodate a larger proximal femoral epiphysis. Starting around the age of 4 and continuing to skeletal maturity, an increase in acetabular depth occurs secondary to growth of the acetabular epiphysis. (**d**) Triradiate cartilage growth is typically complete by 12 years of age in girls and 14 years of age in boys, around which time the acetabular epiphysis initiates its ossification. (**e**) At skeletal maturity, the acetabular epiphysis is completely ossified and acetabular development is complete

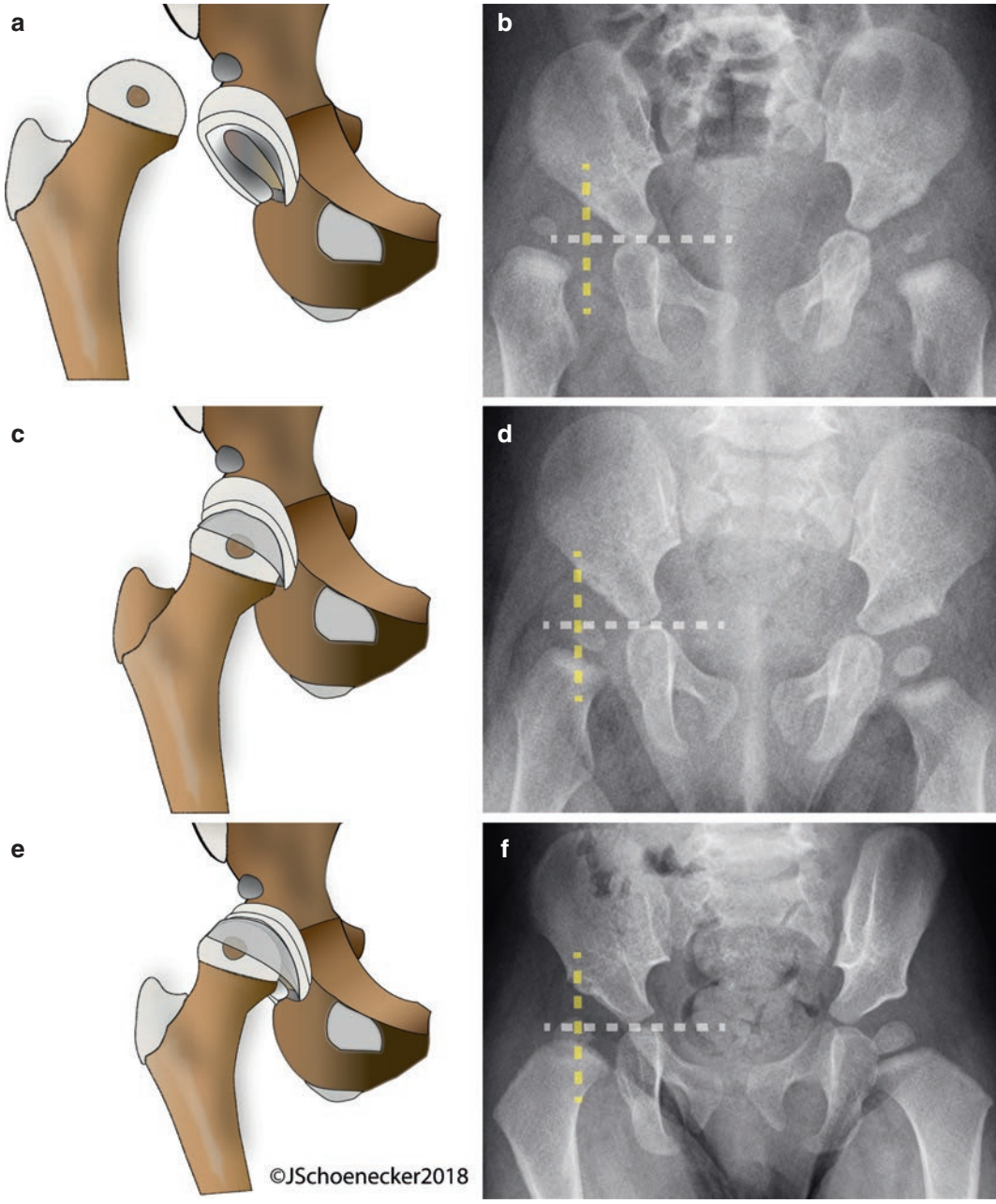


**Fig. 5.4** Deformation of the cartilaginous anlage. Problems of shape and/or delay in ossification of the cartilaginous anlage occur early in life. As the cartilaginous anlage is considerably plastic, malformation most commonly occurs from eccentric loading of the proximal femur. In this case an 8-month old female (**a**) with a dislocated hip observed by (**b**) radiographs with an ossification center in the superior lateral quadrant defined by Hilgenreiner's line (white dashed line) and Perkin's line (yellow dashed line). (**c**) The patient undergoes successful closed reduction with (**d**) restoration of the ossification center to the inferior medial quadrant of Hilgenreiner's and Perkin's line. At this point, although delayed in its ossification, the cartilaginous anlage observed in (**c**) is sufficient to maintain hip congruity. (**e**) As the child grows, if the acetabular anlage fails to ossify and deform, the hip will migrate superior lateral without dislocation. (**f**) This migration is observed on a standing radiograph with migration of the ossification center back towards the superior lateral quadrant formed by Hilgenreiner and Perkin's lines, a break in Shenton's line and increased acetabular index (AI) as compared to the contralateral side. Together, these pathologic changes in the anlage results in an altered hip center, which has not been demonstrated to spontaneously recover

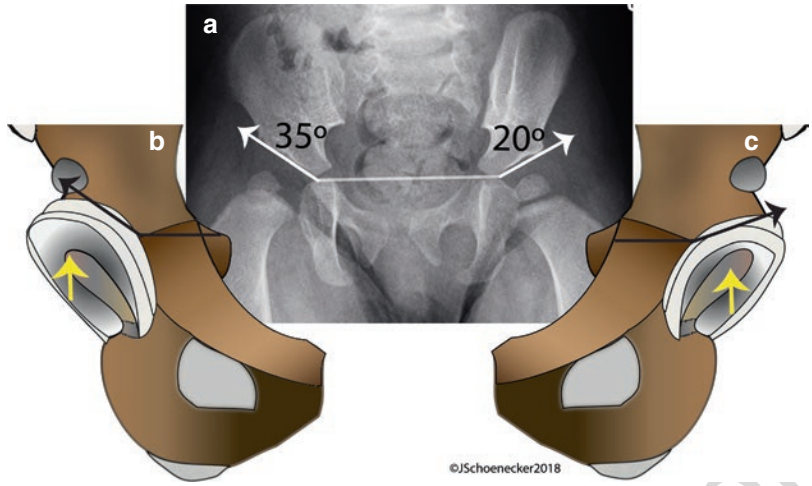


**Fig. 5.5** Acetabular index. The acetabular index (**a**) measures the ossification of the cartilaginous anlage. The normal values per age are presented in Table 5.1. In a normally forming hip (left hip and **c**), the ossification of the anlage results in an index of 20° by the age of 3. This maintains the hip center in the appropriate position (yellow arrow **c**). With pathologic development (right hip and **b**) the anlage may deform leading to a delay in ossification and migration of the hip center laterally (yellow arrow **b**)

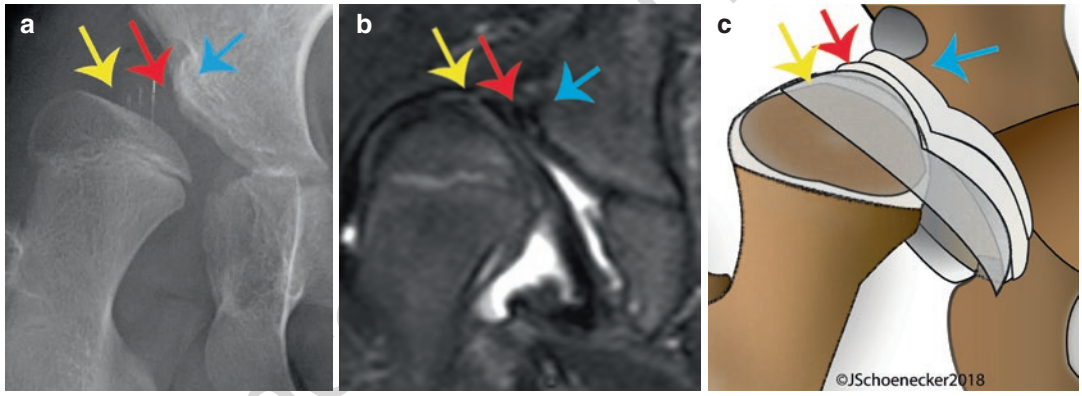


**Fig. 5.6** Severe deformation of the cartilaginous anlage in untreated dysplasia. Although the cartilaginous anlage has a significant capacity to remodel, it cannot do so without restoration of the appropriate hip center. Severe deformity of an untreated subluxed, but not dislocated, hip often leads to permanent deformity of the vital developing structures of the hip. Radiographs (**a**), MRI (**b**) and drawing (**c**) of a 10-year-old boy demonstrates a subluxed hip with lateral migration and break in Shenton's line. This migration places the labrum (yellow arrow) cartilaginous epiphysis at the junction with the labrum (red arrow) in a biomechanically unfavorable position to function. Additionally, the pathologic forces deform and delay the ossification of the cartilaginous anlage (blue arrow)

without surgical intervention. Delay in ossification of the acetabular cartilaginous anlage, observed as a delay in acetabular index normalization, may also occur in idiopathic cases without morphologic change in the anlage. Additionally, if left uncorrected, the acetabular anlage shape and the resultant position of the acetabular epiphysis and labrum, may result in an intra-articular deformity with dual hip centers that present few reconstructive possibilities other than salvage procedures (Fig. 5.6). Conditions affecting triradiate cartilage growth are typically caused by trauma, infection, cancer or iatrogenic (secondary to surgery) [15]. Abnormal triradiate cartilage growth often results

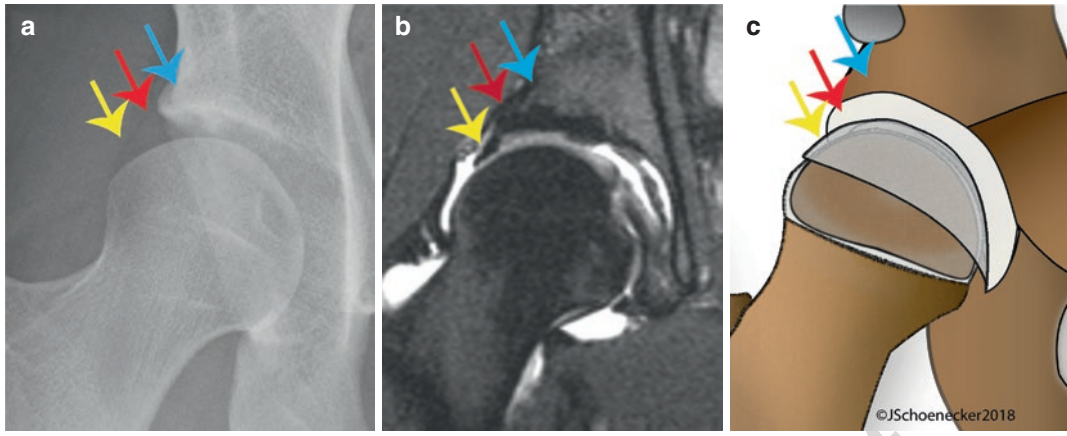


**Fig. 5.7** Late pathology of the acetabular epiphysis: Problems of development, shape or delay of ossification of the acetabular epiphysis occur close to skeletal maturity. They typically present radiographically as an insufficient anterior or lateral center edge angle (**a**) radiographically or (**b**) by MRI. Failure of both cartilaginous growth (red arrow) and ossification (blue arrow) of the anterior and lateral acetabular epiphysis accounts for the majority of late-presenting acetabular dysplasia. These developmental conditions lead to improper loading of the labrum (yellow arrow) and chondrolabral junction

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in the development of a severe pincer type acetabulum. Fortunately, likely secondary to the robust blood supply of the pelvis, premature triradiate cartilage arrest is rare. There are few reports of successfully restoring the normal development of a failed triradiate growth center. Problems of shape or delayed ossification of the acetabular epiphysis occur closer to skeletal maturity. They typically present radiographically as an insufficient anterior or lateral center edge angle radiographically or by MRI. Failure of both cartilaginous growth and ossification of the anterior and lateral acetabular epiphysis accounts for the majority of late-presenting acetabular dysplasia (Fig. 5.7). Primary acetabular dysplasia occurs typically in females, the etiology is unknown.

*Acetabular dysplasia can occur from either: (1) improper shape and/or delay in ossification of the cartilaginous anlage, (2) damage to the triradiate cartilage, or (3) problems of shape and/or delayed ossification of the acetabular epiphysis.*

The main principle in the treatment of acetabular dysplasia involves maintaining hip joint reduction to provide an optimum environment for acetabular and femoral head development. Intervention should be considered to alter an adverse natural history of pathologic acetabular cartilaginous anlage, triradiate cartilage and acetabular epiphysis.

**Essential Pathophysiology Leading to Early Hip Degeneration**

- Primary Dysplasia (conferred by developmental pathology):
  - Acetabular dysplasia (e.g. failure of ossification of the acetabular cartilaginous anlage or epiphysis)
  - Proximal femoral dysplasia (e.g. congenital femoral deficiency, coxa vara/valga, excessive femoral ante/retro-version)
- Secondary Dysplasia (conferred by other diseases):
  - Perthes disease (or other avascular necrosis) with resulting coxa magna,

with late symptomatic impingement and acetabular dysplasia (variable)
- Slipped capital femoral epiphysis (SCFE) associated with metaphyseal CAM deformity and femoroacetabular impingement
- Neuromuscular acetabular dysplasia and coxa valga occurring secondary to spasticity, muscle imbalance, etc. (e.g. cerebral palsy; Charcot-Marie tooth peripheral neuropathy).

## Natural History

The lack of femoral head coverage exists along a spectrum; from under-coverage leading to improper joint loading and subluxation to over-coverage resulting in femoroacetabular impingement (FAI) [16–18]. Both morphologies predispose the hip to damage of the labrum/chondrolabral junction and articular cartilage; potentiating the premature development of OA [10, 16, 18–22]. Nevertheless, the factors which predispose certain hips to eventual degenerative change remain uncertain [23, 24]. Early in life, the fate of a congenital hip dislocation has been well documented. However, the natural history of acetabular dysplasia in the pediatric and young adult patient remains largely undescribed; with the exception of a few studies that have formed the basis of surgical indications for hip preservation [18, 24–28]. Understanding how acetabular morphological characteristics affect the rate of degenerative change in the hip has substantial implications for prognostic assessment and joint preservation patient selection at all ages [29]. The natural history of acetabular dysplasia with subluxation is clear; degenerative joint disease will develop in all patients, usually in the third to fourth decade of life [25, 27, 28]. Additionally, as LCEA decreases, subluxation increases [25]. The natural history of untreated adults with dysplasia is more difficult to predict because patients compensate well and present with dysplasia only as an incidental finding on radiographs or if they have symptoms. However, there is good evidence that dysplasia alone, particularly in females, leads to degenerative joint disease in adults [1, 25, 27, 28]. The question of when hip dysplasia patients will become symptomatic in the absence of treatment was evaluated by Hartofilakidis et al. In their series of 202 dysplastic hips, the average age for onset of symptoms was 34.5 years. In patients with a low and high dislocation, pain from degenerative arthritis associated with a false acetabulum started at an average of 32.5 and 31.2 years, respectively. If there was no false acetabulum, pain onset did not occur until 46.4 years, and was mostly secondary to muscle fatigue [30].

The natural history of hip dysplasia and subluxation in untreated adults can be extrapolated to residual dysplasia and subluxation after treatment in the pediatric patient [1, 27, 28]. In a study of 152 pediatric hips treated with closed reduction and followed for 31 years, the authors reported that dysplastic hips often went on to subluxation and the development of degenerative joint disease [27]. The cause of degenerative changes in dysplastic hips is probably mechanical in nature and related to increased contact stress, especially to the labrum/chondrolabral junction, leading to damages articular cartilage over time. There is a clear association between excessive contact stress and late degenerative joint diseases for other abnormal anatomical morphologies, such as genu varum and genu valgum. This same association seems to occur in dysplastic hips with relation to the development of degenerative joint disease at long-term follow-up [31, 32].

Untreated severe dysplasia of the hip frequently leads to osteoarthrosis [14, 25, 31, 33, 34]. While there is no debate that severe dysplasia of the hip should be treated operatively, objective criteria on which to base the treatment of mild and moderate dysplasia was not available until the mid-1990s and continues to be refined as we learn more about the natural history. Therefore, operative treatment for residual dysplasia of the hip after skeletal maturity assumes that the dysplasia, if left untreated, will lead to secondary osteoarthrosis of the hip [14, 16,

35–41]. Without long-term studies with matched pairs and clearly defined parameters of dysplasia, the natural history of what is now often considered an operative indication may never be realized. In an attempt to provide objective parameters to follow the natural history of acetabular dysplasia, Murphy and colleagues first defined the dysplastic acetabulum using CT to assess the morphological differences between two matched cohorts of females with mean age 20 years [42]. In their study, the first cohort all went on to pelvic osteotomies for symptomatic dysplasia and the control group was obtained from patients who had CT scans obtained for alternate pelvic pathologies. Comparing the cohorts, acetabular anteversion was consistent (mean, 20°) and abduction was moderately increased in the dysplastic group (mean 62° vs. 53° in controls). The most significant difference was in the mean lateral center edge angle with the normal hips measuring 31° and the dysplastic hips measuring 6°. This reduction in femoral head lateral coverage was shown to be part of a more global acetabular deficiency in the dysplastic hip. The normal acetabular volume equated to a hemisphere while the dysplastic only measured one third of a sphere.

***Operative treatment for residual dysplasia of the hip after skeletal maturity assumes that the dysplasia, if left untreated, will lead to secondary osteoarthrosis of the hip.***

From these objective determinates, Murphy et al. [43] then published the first significant natural history study of the skeletally mature dysplastic hip. This study retrospectively evaluated 286 young patients with previous unilateral THA for dysplasia and focused on the contralateral non-operated hip. Ultimately, 115 of the patients developed severe OA in the contralateral hip by 65 years of age. These patients also had statistically greater derangement of all measured radiographic features of dysplasia; including lateral center edge angle (LCEA), acetabular index of depth to width (D/W), vertical distance, lateral distance, peak-to-edge distance, femoral extrusion distance and acetabular index (AI). Key findings were that no patient in whom the hip functioned well until age 65 had a LCEA <16°, D/W <38%, AI >15°, femoral head uncovering >31% or a peak-to-edge distance of 0 mm. While they clearly showed that for patients, whom have a hip replacement for dysplasia in one hip, severe OA will inevitably develop in the contralateral hip if the aforementioned acetabular criteria are not met. However, this investigation lacked a true non-operated control group but did have a comparison group (171 pts) without progression of OA over the same timeframe. Unfortunately, a major limitation to the study was that a substantial portion of the patients that progressed to OA had evidence of mild OA at the time of inclusion thus the outcomes are to some degree measurements of secondary OA progression [43].

Further confounding our understanding of the natural history of acetabular dysplasia, patients often have additional pathologies. For example, many patients have combined acetabular dysplasia with FAI. It is not clear to what extent FAI impacts hip dysplasia and the development of OA. Bardakos et al. showed that mild to moderate OA in hips with a pistol-grip CAM deformity does not progress rapidly in all patients, with one-third of their patients taking at least 10 years to manifest signs of OA with some never showing radiologic signs [24]. Further analysis found two important variables associated with those that did progress; the height of the trochanter relative to the center of the femoral head and the presence of acetabular retroversion. Their conclusion was that a hip with cam impingement is not always destined for end-stage arthritic degeneration [24]. Other studies have found even larger percentages of asymptomatic patients with cam deformity that do not progress to OA. In the study by Hartofilakidis et al., 82.3% of asymptomatic hips with a CAM deformity remained free of OA for a mean of 18.5 years [26]. Clearly the role of FAI in the natural history of hip dysplasia requires more study to determine which hips are at higher risk of progression.

Recently, Wyles et al. published the most comprehensive study of the natural history of OA in patients with hip dysplasia [18]. Similar to Murphy et al., they retrospectively studied 172 young patients (mean, 47 years-old) that had undergone unilateral total hip arthroplasty (THA). While the Murphy et al. study contained

patients with advanced Tönnis grades, all patients in the Wyles et al. study had a Tönnis grade 0 grade for the contralateral hip. These hips were given a structural diagnosis of acetabular dysplasia (48 patients), FAI (74 patients) and normal (40 patients) and were evaluated for OA progression. At a mean follow-up of 20 years, 35 patients underwent contralateral THA; 16 (33%) with acetabular dysplasia, 13 (18%) with FAI and 6 (15%) patients with normal morphology. They showed degenerative changes progressed more rapidly in the acetabular dysplasia group with an increased probability of undergoing a THA at 10- and 20-year follow-up compared to the FAI and normal morphology cohorts. FAI was similar to structurally normal hips in terms of progression to THA but patient with a CAM deformity and concomitant acetabular dysplasia developed OA more rapidly. From their data they created 10- and 20-year prognostic tables predictive of osteoarthritic progression (by Tönnis grade) based on the structural diagnosis and initial Tönnis grade. Furthermore, based on their continuous multistate Markov models, they proposed the following new thresholds for an increased risk for OA progression and thus an indication for hip preservation surgery: femoral head lateralization >8 mm, femoral head extrusion index >0.20, acetabular depth to width index <0.30, LCEA <25° and Tönnis angle >8°. A major limitation of this study is that all patients underwent an index THA and thus the findings cannot be directly correlated to highly active patients.

**Acetabular dysplasia can be classified into mild, moderate and severe forms using several well-established measurements. However, its diagnosis and natural history are complicated by the effects of acetabular and proximal femoral version as well as FAI. While the progression to OA in severe dysplasia is clear, surgical indications in mild and moderate forms continue to be refined.**

## Epidemiology

Acetabular dysplasia is one of the most common causes of pre-arthritic hip pain, hip dysfunction, and secondary OA [1, 2, 4, 44]. The infantile form of acetabular dysplasia is considered a multifactorial disease with genetic, ethnic and environmental risk factors [45, 46]. The intrauterine environmental associations include decreased uterine size with first-born children and breech presentation, both of which restrict fetal leg mobility. The incidence of breech birth is 2–4% in the general population but 17–23% among patients with infantile hip dysplasia [45, 47]. The left hip is more commonly affected because of its fetal position adducted against the sacrum [48]. Although no genetic locus has been identified, a hereditary component of developmental dysplasia of the hip (DDH) is strongly supported on the basis of increased risk in patients with a positive family history and varying rates by ethnicity. Extremely low rates of DDH are seen in the African populations [49, 50] with the African Bantu having an incidence of essentially zero [51]. Much higher rates have been reported in Native Americans [52] and the Sami in Norway [53]. DDH is much more common in females but the exact cause is uncertain; one theory being increased joint laxity during the neonatal period secondary to increased female sensitivity to the maternal hormone relaxin [11, 54]. Another is that females are twice as likely as males to be born breech [9, 54].

The prevalence of asymptomatic dysplasia in the general population varies with ethnicity. Caucasians have a 3–4% prevalence of hip dysplasia, with females having a higher prevalence than males [55–60]. Inoue et al. showed the prevalence in men and women respectively to be 5.1% and 11.6% for the Japanese and 1.7% and 5.6% for the French [56]. Another study by Lau et al. found the prevalence of dysplasia (CEA < 25°) to be similar between Chinese and British men at 4.5% and 3.6% respectively, however the same cohort showed a 50% reduction in OA prevalence in the Chinese group (5.4% vs. 11.0%) [58]. It was not clear why the Chinese

group did not progress to radiographic OA at the same rate. A recent study by Engesaeter et al. looked at 2081 Norwegians (mean age, 19 years) and found the prevalence of a Wiberg center-edge-angle (CEA) less than 20° to be 3.3% (4.3% in women and 2.4% in men) [61]. If the Wiberg CEA threshold was increased to less than 25°, 20% (23% in women and 16% in men) of the cohort had hip dysplasia [61]. This further confirmed the higher prevalence of hip dysplasia in Norway and agreed with previous Nordic Arthroplasty Registry findings [62, 63]. Although the percentage of patients with dysplasia who will ultimately progress to end-stage OA is unknown, it has been documented that 25–50% of primary hip OA is due to acetabular dysplasia [30, 34, 64].

Despite the plethora of studies investigating risk factors for infantile hip dysplasia, there is a scarcity of literature describing patient demographics and disease epidemiology for adolescent and adult patients, with most data coming from single surgeon series reporting on symptomatic acetabular dysplasia [16, 65–68]. In a series of 337 patients undergoing total hip arthroplasty, Clohisy et al. found that 48% less than age 50 had acetabular dysplasia as the predisposing factor for their OA [69]. A recent large multicenter study by Sankar et al. looked at disease epidemiology and patient demographics in dysplastic patients treated with a periacetabular osteotomy (PAO) in 950 consecutive patients [44]. They demonstrated that symptomatic acetabular dysplasia starts 1–3 years before surgical intervention and occurs predominantly in young (average 25.3 years), female, Caucasian patients with a normal BMI (average 24.6). These findings are consistent with other reports in the literature [70–73]. The same study found baseline functional scores to be mean modified Harris Hip Score (mHHS) of 61.8 and a mean UCLA activity score of 6.6. The mean mHHS is slightly lower than published elsewhere (61.8 vs. 66–70) [71, 74] but the mean UCLA activity score is comparable with other authors [75].

Lee et al. further evaluated the differences in symptomatic hip dysplasia treated with PAO based on when the diagnosis was first obtained: infancy, adolescence or adulthood [54]. They found demographic differences between patients diagnosed with hip dysplasia in infancy versus adolescence/adulthood. There were more females with left hip involvement and breech presentation in the infant/DDH population while bilateral disease (61% vs. 45%) was more common in the adolescent population. The same study also looked and family history and found that >50% of all respondents had a family history of hip disease with >40% being first order relatives. first order relatives of adolescent/adult diagnosed patients had a twofold increase in incidence of hip replacement by age 65 compared to infant/DDH first order relatives (50% vs. 22%). However, first order family members of infant/DDH patients were four times more likely to have DDH themselves (59% vs. 16%).

***"Rates of acetabular dysplasia vary widely by gender and cultural origin with several well accepted patient characteristics being predictive of increased risk during infancy. Once a dysplastic hip is present, whether that hip will become symptomatic is directly related to the necessity of treatment. Severity of dislocation and family history along with cultural origin again influence that symptomatology. The periacetabular osteotomy is commonly employed for younger (<40yo) symptomatic patients while a total hip replacement is indicated for older patients (>40yo) with Tönnis 2 changes or greater".***

## Clinical Presentation

Young children with hip dysplasia with or without subluxation typically do not present with any complaints of hip pain or any apparent functional limitations. More likely, children in the first 6–8 years of life will have relatively normal hip function despite radiographic evidence of notable hip dysplasia. However, with growth and increased body mass during and post puberty, older children and adolescents with acetabular

deficiency will variably become symptomatic, particularly in the presence of hip joint subluxation. Signs include functional hip joint associated fatigue and often a subtle, though progressive, gluteus medius weakness limp (Trendelenburg). This early onset weight bearing pain typically is located laterally (trochanteric) and occurs secondary to chronic gluteus medius fatigue. Later, anterolateral groin pain secondary to chondrolabral strain and or injury variably develops. In these relatively older patients, groin pain is characteristically described as an anterior hip centered discomfort often with catching or snapping. Groin pain typically occurs with hip motion such as in pivoting, twisting, running, when arising from a sitting position, and on initiating walking.

On physical examination, the gait of most of the younger children will appear to be normal. On careful observation of older children and or adolescents, there may be an abductor limp and/or a positive Trendelenburg sign on single leg stance examination. The hip abductors may be weak on resistance testing. Passive hip range of motion is initially normal and often increased in all planes, secondary to both relative joint laxity and a variably deficient anterolateral acetabulum. Later as the hip disease progresses, range of motion can become restricted, associated with the onset of painful impingement at the acetabular chondrolabral junction.

## Imaging

Hip joint dysplasia in the child occurs secondary to abnormal growth of either the acetabulum, femoral head, or both, in a non-dislocated hip joint. The shape of the acetabular anlage and its functional capacity to support weight bearing is evaluated with a standing anterior posterior (AP) radiograph. Typically the acetabular development will be deficient anterolaterally. In addition, the absence (or presence) of hip joint subluxation (i.e. Shenton's line intact or not) is assessed. The contour of the anterior and posterior edges of the proximal femurs are visualized on the supine frog lateral radiographs. In older children and adolescents, further visualization of the anterior acetabulum can be seen with the standing false profile lateral radiograph [4]. Persistent hip subluxation after the age of 6 years portends a guarded prognosis for maximal acetabular development for any given hip dysplasia [9]. If subluxation is noted on the standing AP pelvic radiograph, a functional view (AP with hip abducted and internally rotated) is obtained to assess if the femoral head positionally reduces back into the true acetabulum. Knowing that positional reduction of femoral head subluxation is possible, is very helpful in the preoperative planning of joint reconstruction surgery.

**Essential Imaging**

- Plain radiographs: For measurement of AI, LCEA, Tonnis sourcil angle, teardrop morphology, and femoral deformity
- MRI: Used on occasion to assess development of the cartilaginous acetabulum and/or version
- MRI arthrogram (MRA): For assessment of chondrolabral integrity in adolescents
- CT: For either assessment of version (femoral and acetabular) or for concomitant FAI

Normative values for acetabular index (AI) (Coleman 1968; Tönnis 1976), lateral center angle (LCEA) [4, 34], Tonnis sourcil angle [4, 14] are helpful in decision-making and are summarized in Tables 5.1 and 5.2. We also assess the anterior CEA [4, 14] in our older patients. The acetabular tear drop morphology can be useful in assessing acetabular development in late infancy/early

**Table 5.1** Suggested acetabular index (AI) guideline values [14]

| Age | Acetabular index |
|---|---|
| 1 | <25 |
| 3 | <20 |
| 6 | <18 |

**Table 5.2** Lateral center-edge angle (LCEA) guideline values at skeletal maturity

| | LCEA |
|---|---|
| Normal | ≥30 |
| Minimum | ≥25 |
| Poor prognosis | <20 |

childhood (Albinana et al. 1996; Smith et al. 1997). Similarly, Shenton's line, intact or not, is very important in decision making, with chronic subluxation always being a concern for a guarded prognosis [9].

## Non-operative Management

### Childhood (4–10 Years Old)

If sufficient femoral head coverage is present and the child is asymptomatic, one may observe until there is evidence of failure to improve radiographically or until pain develops (Chap. 4, *Developmental Dysplasia of the Hip in Young Children*). Deferring operative treatment after age 6 years (with or without symptoms) is not indicated if hip is subluxated and/or there is persistent acetabular dysplasia in girls ≥8 years or boys ≥9 years.

### Older Child (≥11 Years/Adolescent)

Observe if asymptomatic, but it is essential to follow at least annually. Prognosis relatively guarded in proportion to degree of subluxation and/or acetabular dysplasia.

**Non-operative Pitfalls**

- Inappropriate continued observation in childhood despite subluxation and or lack of progressive improvement in acetabular development (AI) and femoral head coverage (LCEA)
- Loss of opportunity in younger patients (<10 years of age) of correcting dysplasia with simpler procedures (acetabuloplasty and/or Salter innominate osteotomy)
- Failure to continue essential radiographic and clinical monitoring for minimally or asymptomatic adolescents/young adults with hips at risk (acetabular dysplasia and subluxation)

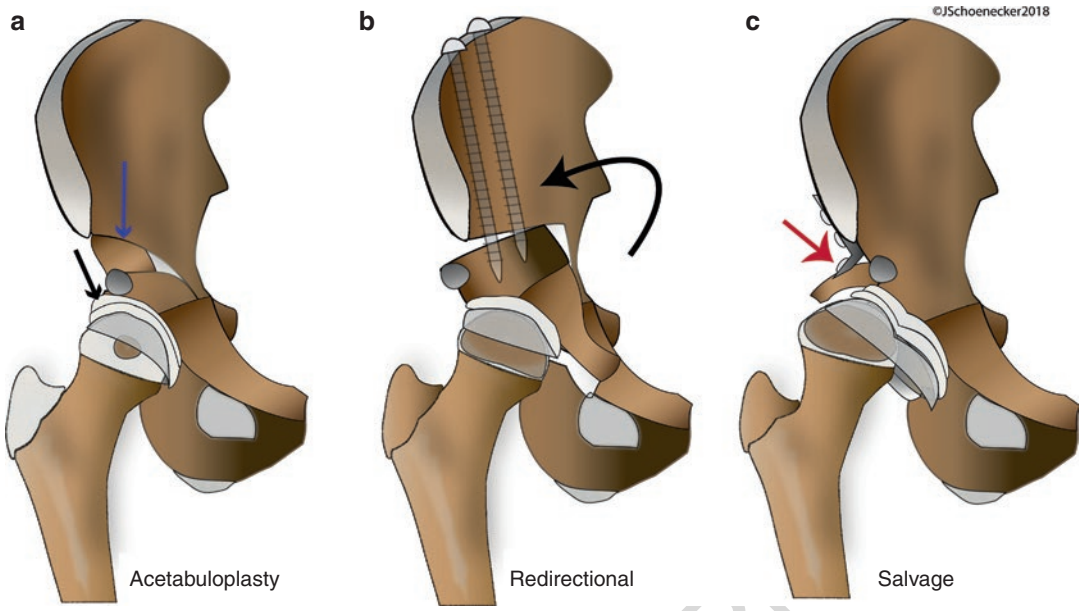
## Operative Management

### Essential Surgical Techniques in Childhood (4–10 Years Old)

Direct surgical correction of acetabular dysplasia can be broken into three different types: (1) acetabuloplasty, (2) redirectional osteotomies, and (3) salvage procedures (Fig. 5.8). The decision as to which procedure is most appropriate in attempting to achieve correction of a specific acetabular dysplasia is dictated by the principal deficiency of development (acetabular anlage, triradiate cartilage or acetabular epiphysis), the age of the patient, and whether the hip is completely reduced. Currently, the incomplete acetabuloplasties [76–80] have become the most popular surgical approach in the correction of residual dysplasia in the skeletal immature pelvis (<11 years).

### Acetabuloplasty

Pathologic shape or delay of the acetabular cartilaginous anlage is often characterized by an acetabulum with a relatively larger arc of curvature then of the femoral epiphysis (Fig. 5.4). The intent of the acetabuloplasty is to correct the pathological increased slope of the superior anterolateral acetabulum (Fig. 5.9). Correcting the acetabular insufficiency (abnormal shape) without damaging the triradiate cartilage redirects the hip center to its natural location, reducing femoral head subluxation. These acetabular osteotomies compress the cartilaginous anlage




AU7 **Fig. 5.8** Types of acetabular corrective surgeries: Direct surgical correction of acetabular dysplasia can be broken into three different types: (**a**) acetabuloplasty, such as the Pemberton osteotomy shown here (**b**) redirectional osteotomies, such as the periacetabular osteotomy (PAO) shown here and (**c**) salvage procedures, such as the acetabular shelf procedure shown here

which restores endochondral ossification providing needed support for functional loading (Fig. 5.10). Acetabuloplasty is best indicated in conditions with remaining acetabular cartilaginous anlage, a relatively capacious acetabulum and or subluxation. Alternatively, satisfactory correction can be achieved with a single innominate (Salter) osteotomy. If the femoral head is relatively large, the Salter redirectional osteotomy can be the preferred technique in attempting to correct acetabular dysplasia in a younger child. Following surgical correction of acetabular dysplasia, an intraoperative arthrogram can be very helpful in assessing femoral head coverage achieved, both laterally and anteriorly (Fig. 5.11).

*"Success is often judged by a radiographically noting a reduction of the acetabular index, a medialized hip center, (from abnormally lateral to more normally medial), and a restored Shenton's line. It is important to be aware of the extent of the acetabular cartilaginous anlage when performing an acetabuloplasty in the correction of acetabular dysplasia. Over correction is very possible which can potentiate late occurring femoral acetabular impingement".*

## Surgical Technique: Pemberton Acetabuloplasty (Figs. 5.9–5.11)

With the patient positioned supine on the operating room table, a small circular roll is placed behind the buttock in back of the affected extremity. The approach to the hip and pelvis is made through a skin excision located lateral to and paralleling the iliac crest extending a couple of centimeters distal to the anterior superior iliac spine. Using the electrocautery, subcutaneous flaps are raised both medially and laterally to the superficial fascia. The fascia overlying the tensor fascia lata muscle is incised longitudinally and dissection directed medially over the tensor and under the sartorius, which protects the lateral femoral cutaneous nerve lying just under the sartorius fascia. Dissection is continued bluntly until the lateral aspect of the rectus femoris tendon and muscle are identified. The external oblique mus-

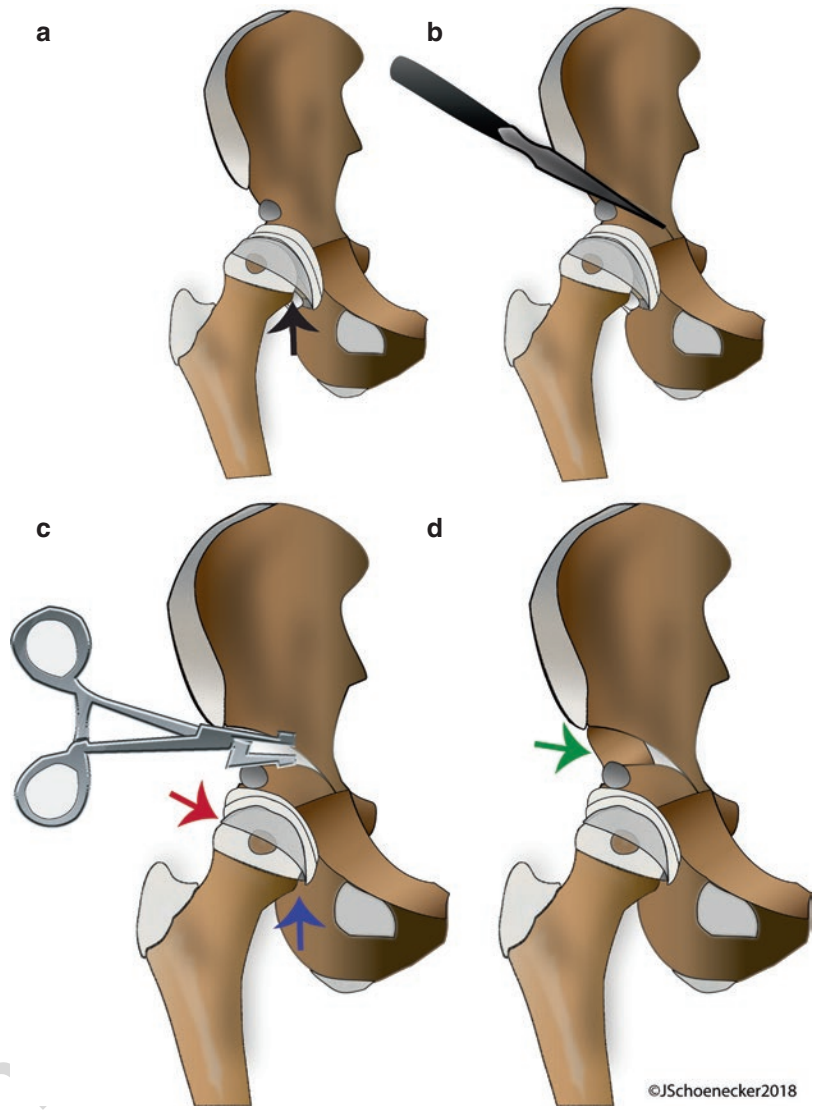



**Fig. 5.9** Acetabuloplasty: (**a**) Pathologic shape or delay of the acetabular cartilaginous anlage is often characterized by an acetabulum with a relatively larger arc of curvature then of the femoral epiphysis and a break in Shenton's Line (black arrow). (**b**) The intent of the acetabuloplasty is to correct the pathological increased slope of the superior anterolateral acetabulum anlage (also see Fig. 5.11 arthrogram). (**c**) Correcting the acetabular insufficiency redirects the hip center to its natural location, here shown with an 'Inge' retractor. The intent is to restore Shenton's line, and the proper position of the chondrolabral junction (red arrow). (**d**) Bone graft (green arrow) is then placed to hold this new position. Healing of the bone graft ensues, often restoring normal hip development (see Fig. 5.10)

cle is reflected of the iliac apophysis from lateral to medial, exposing the apophyseal cartilage which is sharply divided in half from anterior to posterior so as to achieve exposure of the ilium.

The lateral ilium is subperiosteally exposed to the palpable edge of the acetabulum then posteriorly down to the lateral entrance of the sciatic notch (which can be probed with blunt dissecting instrument). The lateral iliac cut is made first. The course of the osteotomy begins anteriorly, mid distance between the anterior superior iliac spine (ASIS) and the anterior inferior iliac spine (AIIS), and extends in a posterior direction. The anterior and lateral line of the cut should be made at least 1½ cm proximal to the edge of the acetabulum. The cut is initiated with a straight narrow osteotome extending in a posterior direction then with a narrow curved osteotome around the acetabulum toward the triradiate cartilage. The C-arm is brought in across from the surgeon and the hip joint and posterior column are visualized with 45° iliac oblique view. As monitored with the C-arm, the course and extent of the curved osteotomy can be precisely located one half way between the acetabulum and the medial edge of the posterior column.

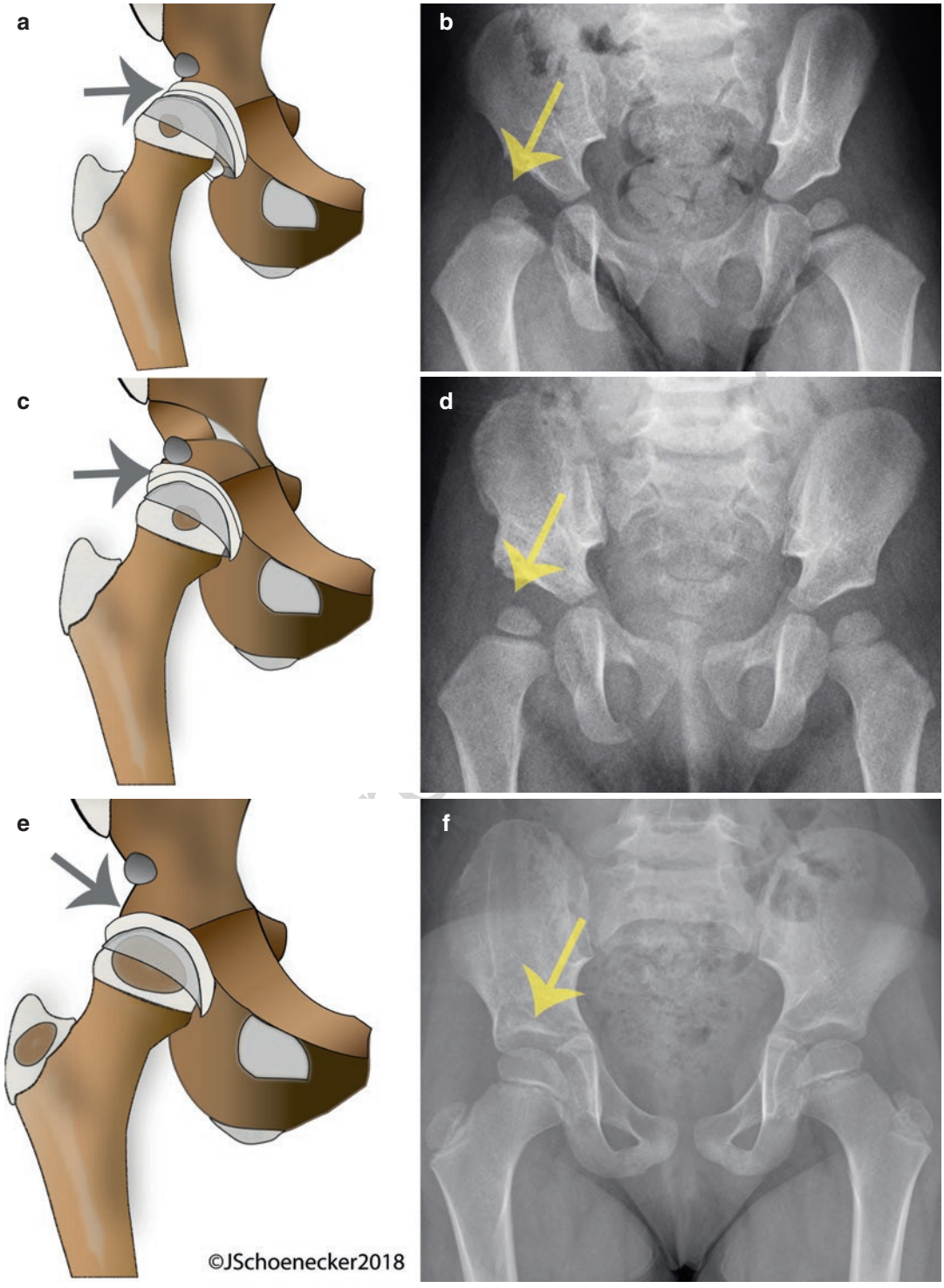



**Fig. 5.10** Restoration of hip development after an acetabuloplasty: (**a**, **b**) Patient from Fig. 5.4 with pathologic cartilaginous anlage (black arrow on drawing and yellow arrow on radiographs). (**c**, **d**) A Pemberton type osteotomy restores Shenton's line and the proper position of the chondrolabral junction, however, note that initially the cartilaginous anlage still appears under-ossified (yellow arrow) as indicated by an increased acetabular anlage at age 2. (**e**, **f**) Four years later, at age 6, the anlage has ossified and the hip position has been maintained without iatrogenic over-coverage of the hip

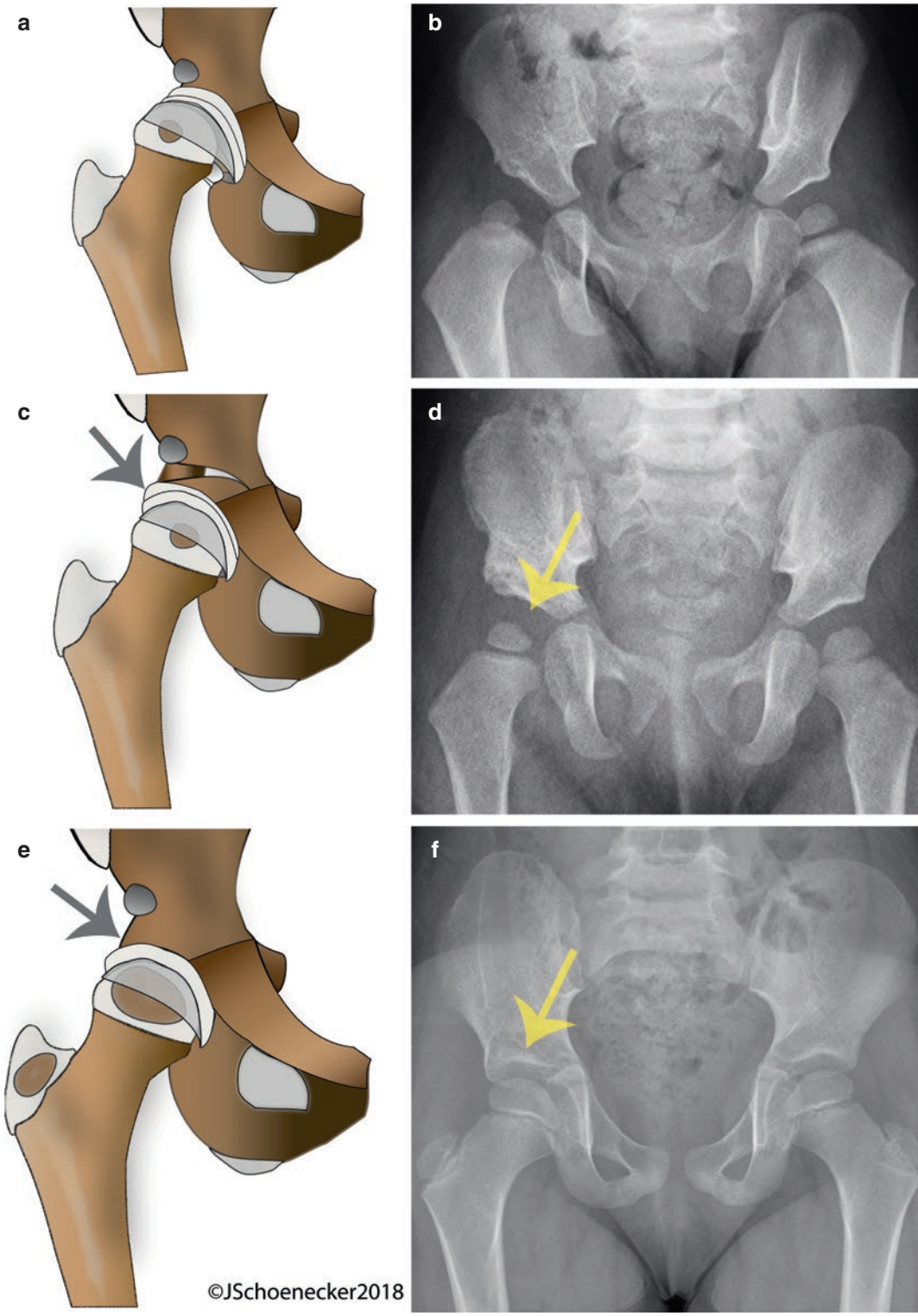



**Fig. 5.11** Measuring correction during an acetabuloplasty: (**a**) Correction of acetabular anlage under-coverage is often monitored by an arthrogram in order to visualize restoration of proper position of the chondrolabral junction (black arrow and "thorn"). (**b**) The gap between the iliac fragments is filled with an allograft (white arrow) and typically inherently stable after graft insertion

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The medial ilium is subperiosteally exposed to and beyond the brim of the pelvis; incising the periosteum facilitates this exposure. A small straight osteotome is then directed through the lateral cut in a medial direction and is visualized cutting through the most anterior 2–3 cm of the medial wall of the ilium. A transverse cut potentiates anterior coverage and a more oblique cut (medial cut made more distal than lateral) greater lateral coverage. The medial cut is continued with a curved osteotome, extending over the pelvic brim, towards but not through the triradiate cartilage, monitored with the C-arm [15]. At this point the osteotomy will be near complete.

> *"As the two fragments are separated with a laminar spreader, displacement of the typical oblique cut (from proximal lateral to distal medial) of the acetabular fragment allows for marked improvement in lateral and anterior femoral head coverage".*

Improved femoral head coverage is monitored by the C-arm using both an AP and false profile lateral views. In the younger child, femoral head coverage is provided by both the bony acetabulum and by the cartilage anlage. An arthrogram can be very helpful showing the true extent of combined bone and cartilage head coverage (Fig. 5.11). Correction achieved should be correlated with range of motion and adjusted to assure there is 90–95° of passive hip flexion following turning down the acetabular fragment. The gap between the fragments is filled with a structural allograft which is typically inherently stable after graft impaction. Optionally, if needed to assure graft stability, a small K-wire may be used in smaller children and/or a 3.5 mm cortical screw in older children, inserted in an antegrade direction starting in the proximal iliac crest and directed across the osteotomy (through the bone graft) and into the ilium medially, just short of the roof of the acetabulum. Bilateral Pemberton osteotomies can be performed if needed. In patients with either excessive femoral anteversion and or coxa valga deformity, a proximal redirectional femoral osteotomy can be concomitantly performed.

The anteriorly prominent spike of bone on the acetabulum fragment is subperiosteally exposed and resected. The apophyseal cartilage is securely repaired with interrupted #1 absorbable sutures, the tensor-sartorius facia reapproximated and the external oblique muscle reattached just lateral to the apophyseal cartilage with running #1 absorbable sutures. The subcutaneous and skin tissues are closed with absorbable sutures. To protect the osteotomy in children less than 4 years of age, a one and half spica cast is placed. In older patients, an abduction pillow is used for 6 weeks. Typically, healing of the osteotomy is sufficient to allow weight bearing as tolerated at 6–8 weeks post operatively.

## Pelvic Redirectional Osteotomies

A pelvic redirectional osteotomy can be single innominate (childhood) or triple pelvic (adolescents/young adults) both being effective in correcting pathological acetabular insufficiency. Salter [39] pioneered operative pelvic redirectional osteotomy correction of congenital acetabular deficiency [39, 81]. The single horizontal osteotomy through the ilium allows for a considerable anterolateral acetabular redirection around the relatively flexible pubic symphysis.

> *"In years past, the Salter osteotomy was the procedure of choice in younger children. It is now used less frequently given the current popularity of acetabuloplasties. However, the Salter osteotomy is still the preferred surgical approach in the correction of residual acetabular dysplasia in the very young child (<5 yrs of age) in which there is relatively little subluxation and an arc of curvature of the acetabulum that is quite similar to the corresponding arc of curvature of the femoral head".*

### Surgical Technique: Single Innominate (Salter) Pelvic Osteotomy

The patient positioning and initial exposure to the ilium is as described above for performing the Pemberton acetabuloplasty. Following division

of the iliac apophysis, both tables of the ilium are dissected subperiosteally until the sciatic notch is encountered, both medially and laterally. Care is taken to avoid penetrating the periosteum as the sciatic notch is dissected subperiosteally. A small curved clamp, such as a Satinsky, is used to pass a # 1 Vicryl suture through the sciatic notch which is then tied to a Gigli saw, so that the saw then carefully passed through the sciatic notch. Crego or similar retractors are inserted into the sciatic notch medially and laterally in order to protect the adjacent soft tissues. An oscillating saw is used to cut the ilium beginning just distal to the ASIS and directed towards the sciatic notch, terminating about 1 cm anterior to it. The Gigli saw is then used to complete the iliac osteotomy so that an angle is created between the posteriorly directed saw cut and the anteriorly directed Gigli cut. Creating such an angle will assist in stabilizing the rotated acetabular fragment against the ilium. The ischiopubic fragment is grasped with a pointed bone forceps just anterior to the sciatic notch osteotomy which effectively helps rotate the acetabulum anteriorly in order to improve anterior and lateral coverage of the femoral epiphysis. The prominent anterior extension of the acetabular fragment is osteotomized (which will be a tricortical bone graft wedge) to be impacted into the gap between the ilium and the acetabular fragment. The osteotomy is transfixed with two threaded Kirschner wires inserted antegrade under AP fluoroscopy, just proximal to the triradiate cartilage. The stability of the osteotomy is assessed manually. The C-arm, with or without a concomitant arthrogram, is used to assure that satisfactory femoral head coverage has been achieved.

Repair of the split apophyseal cartilage is achieved securely with interrupted absorbable sutures. The threaded Kirschner wires are then cut about 1 cm proud from the iliac repaired apophysis in order to facilitate easier removal. Closure of the tensor—Sartorius fascia, reattachment of the external oblique muscle and subcutaneous and skin tissues is as previously described for the Pemberton acetabuloplasty. Following wound dressing application, a single-leg hip spica is applied. The patient is followed up in approximately 2 weeks for clinical and radiographic assessment. The cast is removed at 6 weeks post-operatively and ambulation initiated with a walker until healing is confirmed radiographically.

## Essential Surgical Techniques in Childhood (>11 Years Old to Young Adults)

### Addressing Pathology Subsequent to Ossification of the Acetabular Anlage

Once the acetabular cartilaginous anlage has completely ossified and if the triradiate cartilage is still biologically active, a triple innominate osteotomy should be considered when attempting to achieve satisfactory mobility of the acetabular fragment and, in turn, adequate redirection and correction of acetabular dysplasia (Fig. 5.12). For older children and young adolescents, especially those with significant remaining triradiate growth, greater acetabular mobility is desirable/necessary so as to achieve optimal hip joint stability not only laterally but also anteriorly and posteriorly as needed [14, 76, 77, 82, 83]. With the triple innominate redirectional osteotomy, the surgeon can satisfactorily mobilize the acetabular fragment in attempting to achieve redirection of the acetabulum and optimal hip joint stability.

In the younger child, the surgical exposure during triple innominate osteotomy is extraperiosteal, an essential modification so as to not injure the triradiate cartilage and to facilitate greater mobility with less stress on fixation devices. This technique allows for optimal acetabular redirection (achieving desirable acetabular version) in restoring hip stability; particularly for the younger child with global deficiency (e.g. Down syndrome, Spina Bifida).

### Surgical Technique: Triple Innominate Osteotomy (Fig. 5.12)

The patient is positioned supine on a flat radiolucent table. A roll is placed behind the contralateral knee in order to maintain hip flexion, which helps both to flex the pelvis and to flatten the

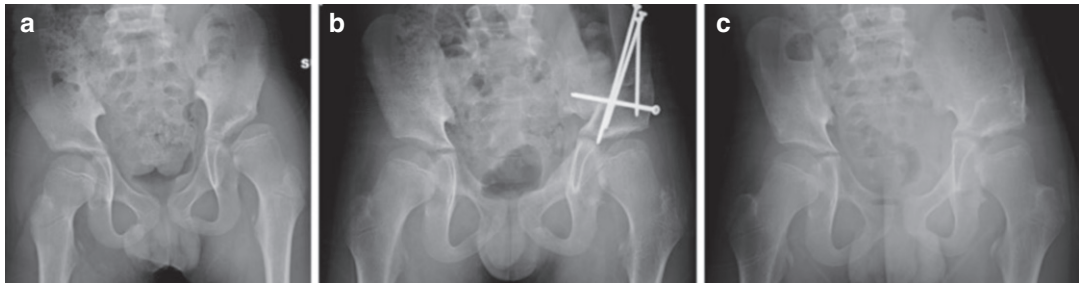



**Fig. 5.12** Triple innominate osteotomy. A 9-year-old male (bone age, 7 years) with a history of viral transverse myelitis secondary to chemotherapy who is functionally a mild diplegic. He is a community ambulator (GMFCS II) with AFO braces and presented with gait deterioration and pain with insufficient lateral and superior coverage of the left hip (**a**). The patient underwent a triple innominate osteotomy (**b**, 3 months post-operatively). (**c**) Two-years later, after hardware removal, the patient had restoration of coverage, increased function and resolution of pain

lumbar lordosis. The initial approach for the triple innominate is as previously described for the Pemberton Acetabuloplasty. Following exposure of the ilium, the interval between the tensor and sartorius is identified. Dissection then proceeds within this interval proximal to the brim of the pelvis. The lateral iliac apophysis is then reflected and the lateral ilium dissected subperiosteally until the sciatic notch is identified. The sciatic notch is also identified medially. The subsequent dissection involves an extraperiosteal exposure of the ischium and pubis while visualizing and protecting the obturator nerve within the retroperitoneum. In order to accomplish this exposure, the fascia of the iliacus is released from the rectus tendon and the hip is flexed in order to relax the iliacus and psoas muscles. This relaxation allows identification of the iliopectineal bursa just medial to the AIIS with blunt dissection medially to expose the pubis. A double-pronged Hohmann retractor is then inserted into the superior pubic ramus medially to retract the iliopsoas while maintaining the hip in flexion to protect the femoral nerve. The interval between the psoas tendon sheath and the hip capsule is developed by opening the iliopectineal bursa medial to the hip capsule in order to access the ischium. Next, with the hip still held in flexion, the iliacus muscle is gently separated from the periosteum of the ilium so that the iliopectineal fascia can be exposed at the attachment to the iliopectineal line. In order to access the retroperitoneum and to protect the obturator nerve, the fascia is then incised and released from the iliopectineal line. The golden coloured retroperitoneal fat is exposed, and the obturator vein and nerve are carefully identified and protected by packing a Ray-Tec sponge extraperiosteally along the quadrilateral surface of the acetabulum. The pubic periosteum can be incised and the remaining iliopectineal fascia released from the iliopectineal line. The true pelvis is then exposed extraperiosteally from the ischial tuberosity to the iliopectineal line.

The pubic osteotomy is performed medial to the pubic limb of the triradiate cartilage using a Gigli saw. In older children the pubis can be dissected subperiosteally; however, in younger children the periosteum is cut with the osteotomy. The anterior pubic periosteum is incised and the root of the pubis carefully dissected subperiosteally using a right-angled clamp. The obturator nerve is protected during this part of the procedure. Alternatively, the entire pubic root can be exposed extraperiosteally. A #1 Vicryl suture is passed through the obturator foramen and used to pass a Gigli saw. The root of the pubis is protected either in an extra- or sub-periosteal fashion and a transverse pubic osteotomy performed using the Gigli saw. Care is taken to orient the osteotomy as perpendicular as possible to the long axis of the pubis.

***"To achieve maximal mobility of the acetabular fragment in children and adolescents, it is very helpful to cut the surrounding periosteum to effectively mobilize the superior pubic ramus fragments after the osteotomy".***

Next the ischial osteotomy is performed using a Ganz osteotome that is passed between the iliopectineal bursa and the hip capsule and positioned at the level of the infracotyloid groove. Using both a 50° cephalad and oblique radiographic (C-arm) guidance, the medial and lateral cortices of the ischium are osteotomized completely, terminating just distal to the ischial spine. The osteotome as visualized on the 50° cephalad view is rotated back and forth in the transverse anatomic plane in order to confirm that the ischium is completely cut. Similar to the pubic cut, the ischial periosteum is cut simultaneously while performing the osteotomy. AP fluoroscopy is used to select the line of the intended iliac osteotomy sufficiently proximal to the acetabulum to allow for later stabilization with screws and/or threaded guide wires. A #1 Vicryl suture is passed through the sciatic notch and used to pass a Gigli saw. The adjacent soft tissue are protected with Hohmann retractors. An oscillating saw is used to divide the ilium from anterior to posterior aiming slightly in a caudal direction and terminating approximately 1 cm anterior to the iliopectineal line. The Gigli saw that has been passed through the sciatic notch is used to complete the iliac osteotomy in order to create a small cephalad directed angle into which the rotated acetabulum can be stabilized. To redirect and control the position of the acetabulum, a 4 mm Schanz screw is inserted into the supra-acetabular ilium and a pointed bone clamp applied to the root of the pubis. The acetabulum is redirected such that the sourcil is oriented horizontally and the anterior-posterior acetabular walls are balanced appropriately across the femoral head (i.e. no "crossover" sign). Provisional fixation is obtained using 2 mm Kirschner wires and an intraoperative radiographs obtained to confirm appropriate reorientation of the acetabulum. On occasion, an intraoperative arthrogram is performed to confirm that the hip is well reduced. The "thorn sign" is used to identify lateral coverage of the head of the femur and overall rotation of the acetabulum. The provisional fixation is then removed and replaced with cortical screws or Steinmann pins. The anterior extension of the acetabular fragment is osteotomized and used as a tricortical graft within the supra-acetabular ilium. The graft is transfixed with one anterior cortical iliac screw. The hip range of motion is assessed for impingement.

Closure is performed in a layered fashion. If the anterior superior iliac spine has been osteotomized, it is reattached with intraosseous O-Vicryl. Repair of the iliac apophysis, closure of the tensor-sartorius interval, reattachment of the external oblique, subcutaneous and skin closure are as previously described. Typically, fixation is sufficient to obviate the need for spica casting, patients are mobilized as appropriate on postoperative day #1. Neurologically disabled children are, as necessary, immobilized in an abduction pillow.

## Surgical Technique: Ganz Periacetabular Osteotomy (PAO)

(Figs. 5.13, 5.14, 5.15, and 5.16)

If the triradiate cartilage appears to be closing or is closed, performing a periacetabular osteotomy (Ganz) achieves both maximal mobility and the potential to redirect the acetabulum into the desired location (Fig. 5.12) [5, 8, 16, 84]. The patient is positioned supine on a radiolucent table. The skin excision again parallels the iliac crest laterally and extends more distally down onto the thigh. Exposure is as described for the Pemberton acetabuloplasty, as is the initial iliofemoral dissection. When reflecting the external oblique muscle from lateral to medial in a skeletally mature patient, the apophysis will be ossified, so the tissue plane is between the external oblique and the periosteum of the iliac crest.

Next, the medial ilium is subperiosteally exposed and an interval is developed between the sartorius and rectus tendon. The ASIS (with the attached sartorius muscle) is osteotomized through an approximately 3 cm oblique cut in a lateral to medial, proximal to distal direction. The interval between the sartorius and the attached ASIS medially and the rectus muscle/tendon complex laterally is developed. Deeper to this, the same interval is further developed between the rectus laterally and the iliacus, iliocapsularis, and iliopsoas muscles medially. With

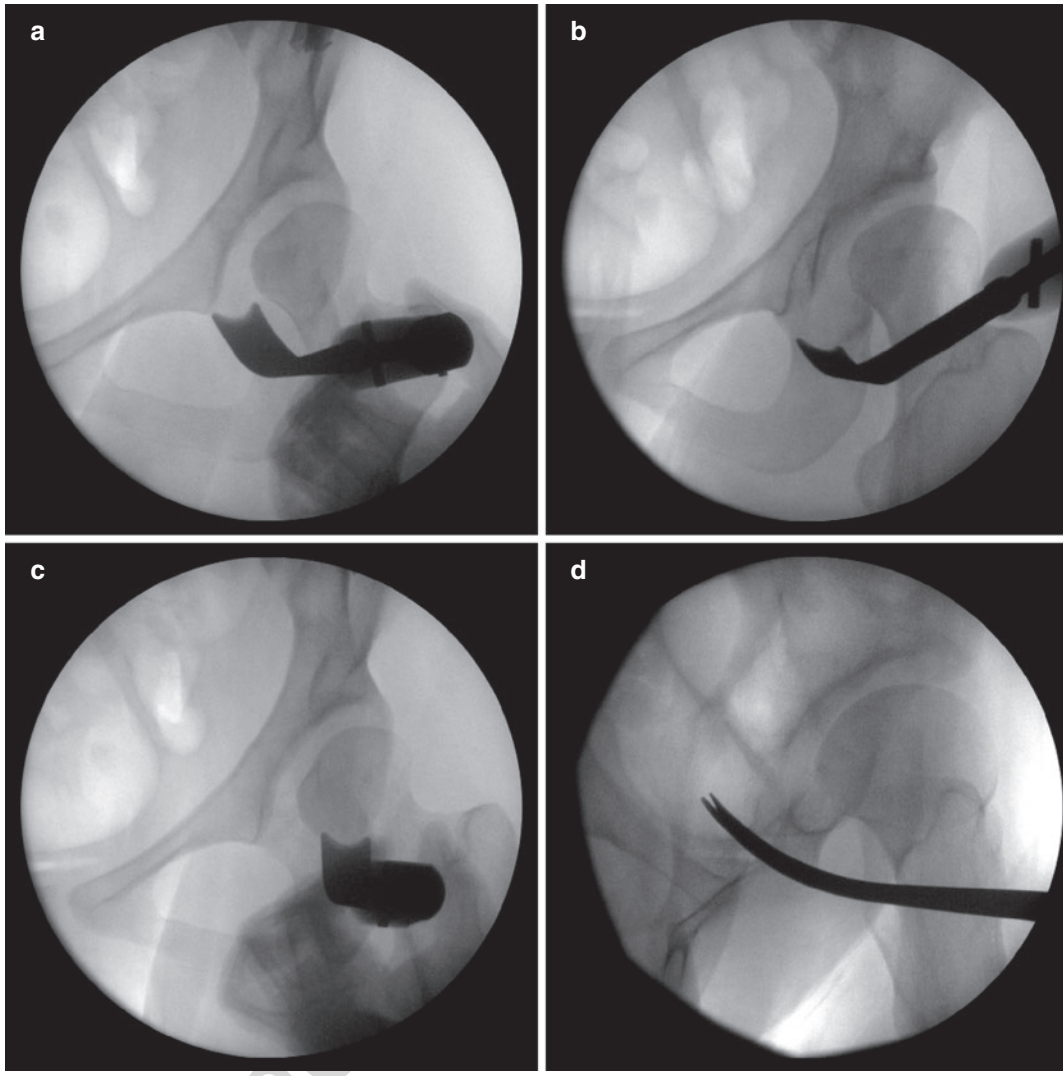



**Fig. 5.13** Periacetabular osteotomy (PAO) Ischial Cuts: The ischium is partially cut (**a**) medially (**b**) centrally and (**c**) laterally in sequence. (**d**) The cuts should extend posteriorly to allow the final posterior column cut (Fig. 5.14) to connect

the hip slightly flexed, the dissection extends in a posterior direction medial to the hip capsule and lateral to the psoas tendon. A long large Mayo type sutures is essential in expanding this interval around the hip capsule posteriorly down to the ischium inferior to the acetabulum. The anterior, medial and lateral cortical surface of the ischium is palpated with the scissors tip. This interval is developed so as to allow for passage of first a hip skid (if possible), then the Ganz angled osteotome inserted down to the ischium with the hip skid protecting the surrounding soft tissues. The ischial cut starts in the infracotyloid groove just inferior to the acetabulum, extending the cut posteriorly around the acetabulum and then proximally, ending near the level of the base of the ischial spine. AP and 45° iliac oblique C-arm views are essential in assuring the correct placement of the osteotome. The medial ischial cortex is cut first, in turn, the middle and lateral cuts are completed. The lateral cut will be shorter (<1½ cm), the

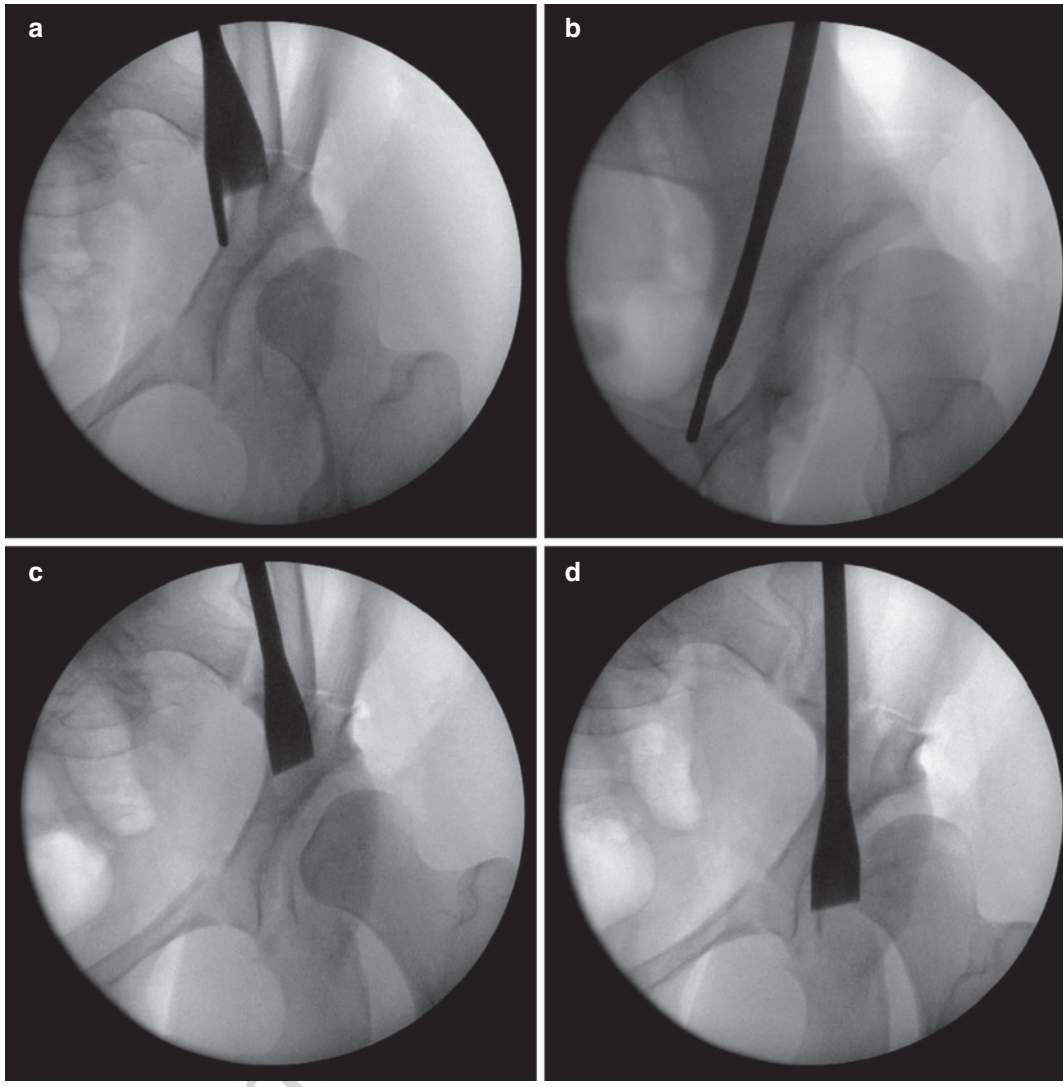



**Fig. 5.14** Periacetabular osteotomy (PAO) Posterior Column Cuts: The final cut of the PAO is to split the posterior column. It is important to complete this cut both medially (**a**, **b**) and laterally (**c**, **d**). (**a**) A "flag" osteotome can be used to follow the false and true pelvis for the medial cut while monitoring the position within the posterior column on the lateral view. (**b**) It should connect with the medial cut of the ischium (see Fig. 5.13a). (**c**) A straight osteotome is used complete the cut—aligning the lateral edge with the lateral edge of the ischium (see Fig. 5.13c)

osteotome directed posteriorly and medially away from the adjacent sciatic nerve. The leg is externally rotated and a foot hand-held to detect any muscle contraction which might suggest sciatic nerve irritation.

The second cut is an osteotomy of the superior pubic ramus. The medial ilium is subperiosteally exposed down to the pelvic brim medially then distally to the obturator foramen. Anteriorly, exposure is extended to and beyond the iliopectineal eminence onto the superior pelvic ramus. A pointed Homan type is driven into the most medial anterior ramus as a retractor for the iliopsoas muscle and neurovascular bundle. Subperiosteal (Crego) type retractors are inserted around the ramus through the obturator foramen proximally (first) and then distally, protecting the obturator nerve. The distal retractor should be

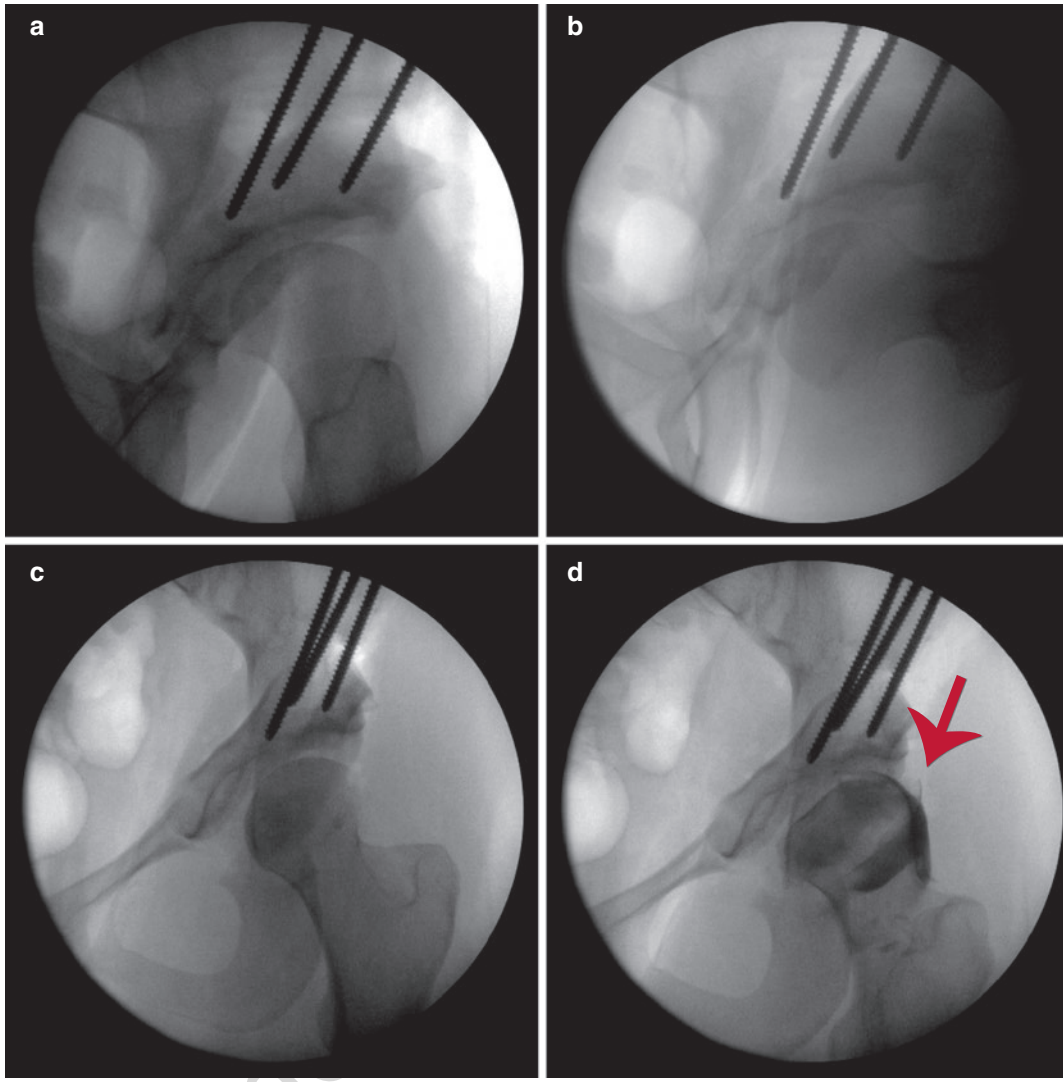



**Fig. 5.15** Periacetabular osteotomy (PAO) Correction: Once free, the acetabular fragment is repositioned to provide anterior coverage (**a**) that permits 90° of hip flexion (**b**). Additionally, (**c**) the fragment should be repositioned to provide significant lateral coverage as judged by an AP radiograph. (**d**) An arthrogram can help judge the position of the un-ossified cartilaginous epiphysis and position of the labrum

slightly medial to the proximal retractor. The superior ramus osteotomy should slope from lateral to medial beginning at a point at least 1 cm medial to the iliopectineal eminence to assure the hip joint is not entered during the completion of the osteotomy.

*"Palpating the adductor muscle for contracture when placing the Crego retractors and/or cutting the superior pubic ramus helps to further protect the obturator nerve".*

To perform the iliac osteotomy followed by the posterior column osteotomy, further exposure is necessary. The quadrilateral plate is subperiosteally exposed down to the sciatic notch and base of the ischial spine. A point is marked on the false pelvis just lateral to the brim of the pelvis which corresponds with the proximal extent of the sciatic notch. To further protect soft tissues, a sub-periosteal retractor is placed along the lateral wall of the ilium in line with the intended course of the iliac osteotomy. The ilium

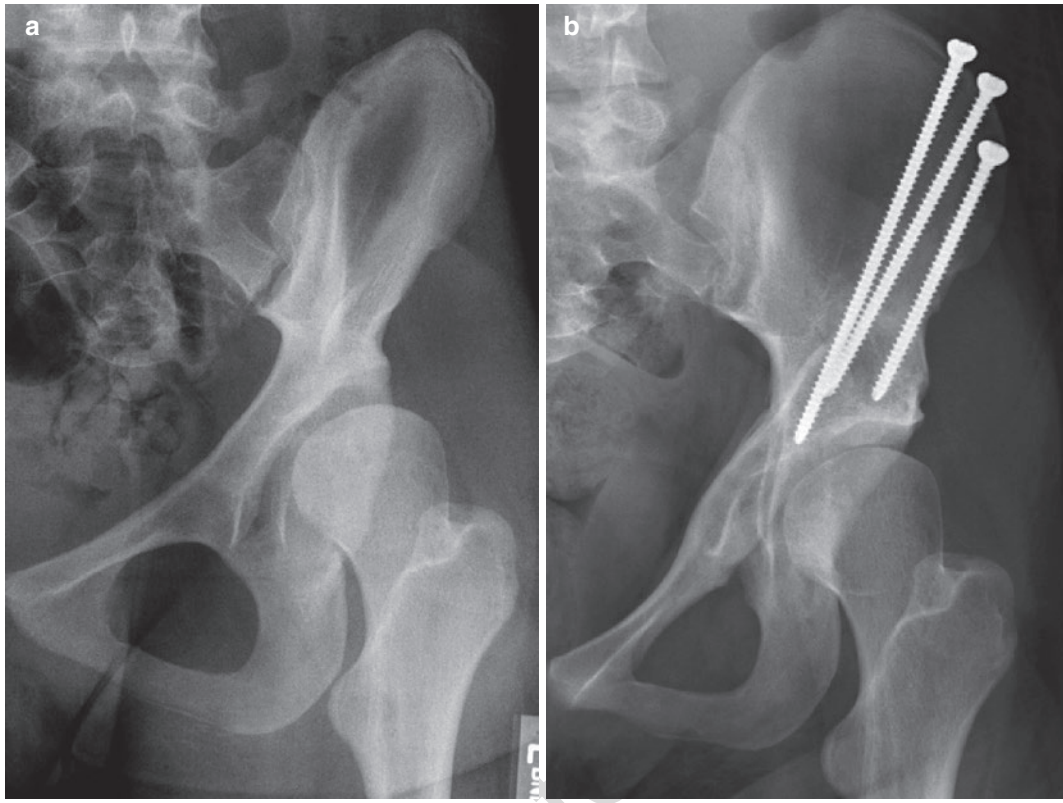



**Fig. 5.16** Periacetabular osteotomy (PAO): (**a**) A 16-year-old female with significant lateral and anterior under-coverage, pain but no labral tear or signs of arthritis underwent a PAO (Figs. 5.13–5.16). (**b**) Two years later the osteotomy is well healed, note the restoration of Shenton's line and hip center. The patient is now pain free returned to sport

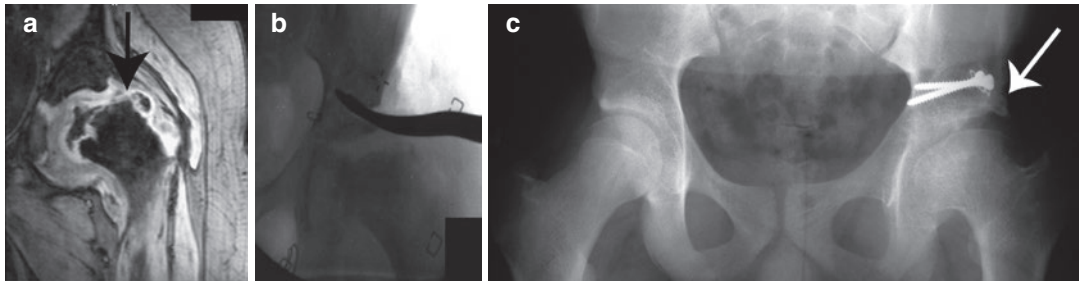
is cut with a power saw to point marked (see above) on false pelvis.

*"The posterior column cut begins at the end of the iliac cut and extends distally to a point near or at the most proximal extent of the initial ischial cut (first cut). The cut should be centered equal distance from the posterior edge of the acetabulum and the edge of the posterior column. The C-arm (45° iliac oblique view) is critical in monitoring the direction of the osteotomy. As the distal portions of the lateral cortex is osteotomized, care must be taken to minimize injury to the sciatic nerve. The hip is extended, abducted and externally rotated and a hand is placed on the foot".*

Having completed all osteotomies, a Shantz screw is inserted into the acetabular fragment just superior to the acetabulum and a T-handle chuck attached. The fragment is carefully mobilized which effectively completes the osteotomies, freeing the fragment from the surrounding intact periosteum. The T-handle is further secured to the acetabular fragment with a curved (lobster claw) bone fragment. Correction desired is typically achieved by adducting, medializing and anterior tilting (extending) of the acetabular fragment. Provisional fixation is achieved with 3/32 K-wires.

*"Correction achieved is assessed with the C-arm. Adjustments are made so as to optimize coverage, medialization and acetabular version. The range of motion is assessed, flexion to 90° and abduction to 30° should be present. If not, the correction obtained should be decreased".*

The acetabular fragment is secured with cortical screws, inserted in the same direction as the previously placed K-wires. Final correction is




AU10 **Fig. 5.17** Salvage procedure: Shelf Arthroplasty. (**a**) A patient with in incongruent femoral head and acetabulum underwent a shelf osteotomy (**b**). Two years later, the patient has healed the shelf and the extracapsular arthroplasty has influenced restoration of the hip shape (white arrow)

again assessed by C-arm and hip motion again assessed. If internal rotation in 90° of flexion is limited (<15–20°) and a head-neck offset deficiency is noted on pre-operative radiographs, an anterior arthrotomy is performed and an anterolateral head-neck osteochondroplasty performed which typically allows for improved internal rotation in flexion [85]. If opened, the capsule is closed. The ASIS and attached sartorius muscle are reattached with #5 permanent suture, placed through the drill holes in the ilium and around the base of the ASIS. The tensor/sartorius fascia is reapproximated and the external oblique muscle sutured to the soft tissue on the anterolateral edge of the ilium. The subcutaneous and skin tissues are closed in layers. Patients are mobilized on post-operative day #1, and early weight bearing is encouraged for patients with good bone quality.

## Irreducible Hip Joint

For those hips unable to achieve a concentric reduction, salvage procedures, rather than redirectional, are indicated. These involve either moving the ilium itself laterally (i.e. Chiari-type) [86–89] or augmenting extracapsular bone with a bone graft (i.e. shelf arthroplasty) to provide femoral head coverage [90, 91].

***"Importantly, for satisfactory outcome of either a pelvic redirectional osteotomy or an acetabuloplasty, it is essential that anatomical reduction of the femoral head into the true acetabulum is possible. In cases in which this is not possible, a salvage type procedure should be considered so as to provide extracapsular stability of the hip joint Fig. 5.17)".***

## Surgical Technique: Chiari Osteotomy

The approach for the Chiari osteotomy is quite similar as to the approach for Bernese PAO and or triple innominate osteotomy. Subperiosteal exposure of both the lateral and medial walls of the ilium is obtained. Medially, the subperiosteal dissection extends to and beyond the brim of the pelvis down to and into the sciatic notch, and distally to the base of the ischial spine. Laterally, the abductor muscle is detached from the iliac crest and subperiosteal dissection extended down to and into the sciatic notch. When exposing laterally, subperiosteal exposure is carried as far distally as possible. In some instances, this may include distally elevating proximally displaced lateral labral chondral tissues (distally, without violating the lateral capsule). Dissecting in the notch with a sponge facilitates exposure. The osteotomy begins anteriorly at a point half way between the ASIS and AIIS and extends posteriorly (similar to the iliac cut of the previously described Ganz PAO) but extends across and through the posterior column, exiting at the sciatic notch. To both optimize the eventual displacement of the fragments and provide enhanced osteotomy stabilization, the osteotomy should be angulated approximately 15° cephalad in the frontal plane. Completion of the osteotomy is performed with a power saw beginning anteriorly and extending towards the sciatic notch. Completion of osteotomy within the notch can be

achieved with a Gigli saw, cutting anteriorly and laterally out of the notch, or with an osteotome cutting into the notch. In doing so, medial and lateral large Hohmann retractors are subperiosteally placed in the notch, serving to protect the gluteal vessels and sciatic nerve.

> *"Once completed, the distal fragment is displaced medially, which variably effects improved superior capsular coverage by the osteotomy surface of the proximal fragment. Effort must be made to assure that sufficient displacement occurs but also that the distal fragment does not displace too posteriorly".*

Improved lateral coverage can be augmented with insertion of a shelf augmentation, which also helps to minimize the variably occurring osseous offset between the two pelvic fragments. The osteotomy is transfixed with K-wires (inserted proximal-distal/lateral-medial) in younger patients (<10 years). For older patients, and/or when no cast immobilization is planned, the osteotomy is fixated with large fragment (4.5 mm) cortical screws and cast immobilization is not required.

Typically, a capsulotomy is not performed. However, if there is considerable subluxation, performing an anterior capsulotomy can help minimize the extent of subsequent hip joint instability. The capsulotomy is performed anteriorly, does not extend superolaterally, but correctly extends through the most medial capsule to the medial edge of the true acetabulum. This potentiates femoral head medialization which in turn makes it possible to perform a more distal Chiari osteotomy cut (which is desired). When repairing the capsulotomy, it is critical to achieve a very competent capsulorrhaphy as the capsule later serves as an interpositional arthroplasty, essential in performing a Chiari displacement osteotomy. The medial and lateral ilial soft tissues are securely repaired both to each other and to the iliac crest with #1 Vicryl suture. The tensor sartorius interval is closed and the external oblique is also reattached to the soft tissues just lateral to the iliac crest. The subcutaneous and skin tissues are closed in layers.

In younger patients, hips are protected with an abduction pillow, and kept non-weightbearing for 5–6 weeks. Younger patients are then mobilized as possible with protected weight bearing using a walker and/or crutches. Older patients whose osteotomies have been fixed with cortical screws are mobilized as possible post operatively. Weight bearing is limited until early bone healing is present (6–8 weeks) and then weight bearing is gradually increased.

## Surgical Technique: Shelf Arthroplasty (Figs. 5.8 and 5.17)

Shelf arthroplasty can at times be very effective in the treatment of problematic hip joint subluxation secondary to various developmental hip joint pathologies, in older children and adolescents. The surgical approach in all cases includes first achieving femoral head reduction and then providing stabilization of what often is a complex hip joint subluxation. For the shelf arthroplasty, it is desirable to use either a screw-plate or screw-washer stabilization of the shelf to optimize immediate post-operative early hip joint stabilization. The combination of surgical reduction as necessary and secured shelf arthroplasty has proven to be very effective for the patient with severe hip subluxation and deformity.

> *"A relatively "fixed" shelf provides immediate/early important stabilization following surgical reduction of the previously subluxated hip in patients with severe acetabular insufficiency secondary to neuromuscular disorders such as cerebral palsy and Charcot-Marie-Tooth Disease. A surgically secured shelf also provides early stability in the containment treatment of Perthes disease".*

The procedure is performed with the patient positioned supine on a flat radiolucent table with a small bump under the buttock and lower back. The hip and pelvis are exposed anteriorly through an iliofemoral approach identical to that described for the Pemberton Acetabuloplasty. If the femoral head is notably subluxated and a capsulotomy and capsulorrhaphy are to be performed, the rectus tendon is transected 1 cm distal to insertion

and the muscle complex reflected distally to achieve complete capsular exposure. A near horizontal capsulotomy is performed, starting 1 cm lateral to the acetabular rim sloping slightly from proximal lateral to distal medial. To facilitate essential medialization of the head into the true acetabulum, the capsulotomy must be extended to the medial rim of the acetabulum. Problematic musculotendinous and ligamentous contractures may preclude obtaining a satisfactory reduction of the femoral head into the true acetabulum and/or achieving satisfactory hip motion after reduction. To deal with this, a proximal femoral osteotomy (PFO) is designed as dictated by the patient pathology. Components of the PFO include shortening of the femur by ~1.5 cm, rotation as indicated to correct excessive anteversion and increasing varus of neck shaft angle to better seat the femoral head into the acetabulum. The PFO is completed through a second incision using a standard lateral approach to the proximal femur. The timing of the PFO during the procedure is based on need. If femoral shortening is required then the PFO should be done prior to attempting open reduction.

Once the head is reduced into the acetabulum and the capsulorrhaphy complete (if necessary), the shelf acetabuloplasty is performed. The anterosuperior aspect of the acetabulum is located utilizing an AP fluoroscopic image. A series of unicortical holes are made just above the acetabulum, using a 3.2 mm drill through the outer table (2 cm deep, directed 20° cephalad)

***"Starting anterior and moving posterior – just above and as close as possible to the superior acetabular – the shelf must anatomically about the hip capsule without penetrating into acetabular articular surface".***

Once the line of drill holes is complete, they are connected from anterior to posterior using small curettes and dental burrs, directing the dissection so the slot being created is immediately adjacent to the capsule and directed proximally. The trough should be deep enough to abut but not penetrate the inner table. After the trough is created, proceed in harvesting the outer table bone graft. An oscillating saw is used to cut off the top of the iliac crest (saved as a source of bone graft) and then a curved osteotome is used to cut three adjacent longitudinal Cortico-Cancellous strips from the outer (or inner table). The strips should be made as long as possible, but be sure to leave enough intact. One should leave room in the distal outer table to accommodate subsequent plate fixation of the shelf. The total width of the combined strips should be roughly the length of the previously constructed trough. Depending on the volume of graft harvested, you may have to fashion additional corticocancellous strips from a tricortical allograft to augment both the thickness and width of the bony shelf.

The shelf is constructed by laying the graft strips in the trough side-by-side starting posterior and moving anterior. Tamp each individual strip deep into the trough abutting immediately on the capsule. Use the initially obtained osteotomized top of the ilium (a tricortical autograft) to press fit and backfill the superior aspect of the trough and reinforce the shelf. The shelf will become more stable as the trough is tightly filled with graft reinforcing its superior surface. A Freer elevator is inserted between the shelf and the capsule and the C-arm is used to assess there is a tight fit between shelf and capsule.

To assure the shelf remains impacted against the capsule, the shelf is secured in place with either a plate and/or abutting screws. A small fragment (distal radius type) T-plate is appropriately bent and secured with 2–3 bicortical screws through the ilium and 2 locking screws through the shelf. The plate is contoured and fixated between the pelvis and graft so as to both hold the composite graft in-place and compress the shelf against the capsule. Alternatively stabilize the shelf by inserting two cortical screws abutting firmly against the proximal surface of the bony shelf in a blocking strategy (washers with points are essential in achieving secure screw head purchase on the lateral edges of the shelf).

***"Once the shelf arthroplasty has been secured. Final C-arm views include an AP (in abduction and adduction) and false profile lateral (in neutral extension and***

*flexion). Assess femoral head coverage and the desired immediate adjacent contact, with the capsule interposed between the head and the shelf. Also, hip motion should be confirmed, with flexion to 90° and abduction to 25–30° mandatory so as to avoid impingement".*

In closing the conjoined rectus tendon (if transected in the approach) repaired with #1 Ethibond. Repair of the apophyseal cartilage, closure of the tensor-sartorius interval, and reattachment of the external oblique and subcutaneous and cutaneous is performed, as previously described. The patient is placed into an A-frame long leg cast bilaterally with the knees slightly flexed. After 5–6 weeks in the A-frame cast the patient is converted to an A-frame brace and three times daily range of motion exercises initiated. At 3 months, the patients can begin weightbearing based on X-ray evidence of graft incorporation, continuing with the A-frame brace at night.

**Operative Pitfalls**

- Failure to use C-arm monitoring while performing an acetabuloplasty risks inadvertently extending the osteotome into the acetabulum and/or the bone graft into and through the tri-radiate cartilage.
- Overcorrective anterolateral coverage, particularly when performing an acetabuloplasty, risks later development of symptomatic FAI.
- Inadequate fixation risks a postoperative loss of reduction following Salter osteotomy; especially in a small pelvis.
- Attempting to achieve improved coverage with a pelvic procedure without first obtaining satisfactory reduction of the femoral head into the true acetabulum risks persistent postoperative subluxation.
- Exacerbating posterior deficiency and the potential for early redislocation by overcorrection can occur with a combination of acetabuloplasty (or single innominate osteotomy) and an overzealous proximal femoral derotational osteotomy that corrects for anteversion.
- Attempting to correct global deficiency associated with myelodysplasia or Down syndrome, with either an acetabuloplasty or single innominate, may fail because of posterior acetabular deficiency. In these cases, a triple innominate osteotomy is typically necessary.
- Lack of familiarity of the modification of the Smith Peterson approach necessary to achieve access to anterior ischial and superior pubic ramus will make it technically near impossible to safely complete either a triple innominate or PAO osteotomy.
- Failure to complete the posterior column osteotomy of an attempted Ganz PAO and mobilize the acetabulum fragment will limit achieving both satisfactory redirection and medialization of the acetabulum.
- Failure to stabilize a triple innominate or Ganz PAO risks early post-operative loss of acetabular reorientation.
- If a Chiari osteotomy is too proximal, relative to the joint capsule, the potential for improved stability provided to the femoral head is notably compromised.
- Allowing excessive posterior displacement of the distal Chiari fragment risks injury to the sciatic nerve.
- Failing to obtain intimate contact of the capsule and femoral head when performing a shelf arthroplasty minimizes any subsequent beneficial supportive function of the shelf.

## Classic Papers

**Harris WH. Etiology of OA of the hip. Instr Coume Lect 1986**. Proposed that OA of the hip typically does not exist as a primary disease, a change in our thinking.

**Klaue K. The acetabular rim syndrome. A clinical presentation of dysplasia of the hip J Bone Joint Surg Br 1991**. Hip pathology that leads to damage to the labrum and/or chondrolateral junction is what typically leads to acetabular and femoral head arthritis.

**Ponsetti IV. Morphology of the acetabulum in congenital dislocation of the hip, gross, histological and roentgenographic studies. J Bone and Joint Am 1976**. Detailed description of the cartilaginous anatomy of the acetabulum and its growth and development.

**Weinstein SL. Natural history of congenital hip dislocation (CDH) and hip dysplasia. Clin Orthop Relat Res 1987**. Extensive description of the normal and abnormal radiological features of hip development and factors, most important in clinical outcome.

**Tonnis D. Congenital dysplasia and dislocation of the Hip in children and Adults. Springer 1987**. The most informative/authoritative text on hip dysplasia (a "must" for hip aficionados).

**Cooperman DR. Acetabulum dysplasia in the adult. Clin Orthop Relat Res. 1983**. Documented that degenerative hip joint disease will eventually develop in all patients with hip subluxation.

**Wiberg G. Studies on dysplastic acetabular and congenital subluxation of the hip joint with special reference to the complication of OA. ACTA Chine Scandinavia 1939**. Very early definitive description of radiographic acetabular deficiency, what was felt to be normal and abnormal and its association with the development of OA.

**Salter RB. Innominate osteotomy in the treatment of congenital dislocation and subluxation of the hip. J Bone Joint Surg Br 1961**. First publication of its kind describing a complete iliac pelvic osteotomy allowing for anterolateral redirection of the congenitally deficient acetabulum.

**Pemberton PA. Pericapsular osteotomy of the ilium for treatment of congenital subluxation and dislocation of the hip. J Bone Joint Am. 1965**. Also a first in the description of redirecting the acetabulum without making a complete cut through the entire ilium, i.e. "acetabuloplasty".

**Wyles EC. The John Charnley Award: Redefining the natural history of osteoarthritis in patients with hip dysplasia and impingement. Clin Orthop Relat Res 2017**. Comprehensive comparison in longitudinal study of frequency of total hip replacement necessary in patients with either FAI, dysplasia or normal morphology.

**Smith-Petersen MN. The Classic: Treatment of malum coxae senilis, old slipped upper femoral epiphysis, intra pelvic protrusion of the acetabulum, and coxa plana by means of acetabuloplasty in 1936 (reprinted in Clin Orthop Relat Res 2009)**. Original description on the technique of what is now the standard anterior approach to the pelvis and hip joint.

**Betz RR. Chiari pelvic osteotomy in children and young adults. J Bone Joint Surg Am 1988**. Indications for and technique of performing a Chiari osteotomy.

**Kuwajima SS. Comparison between Salter's innominate osteotomy and augmented acetabuloplasty in the treatment of patients with severe Legg Calve-Perthes disease. J Pediatr Orthop B 2002**. The author demonstrated the slot-secured shelf arthroplasty in the treatment of problematic Coxa Plana.

**Faciszewski T. Triple innominate osteotomy for acetabular dysplasia. J Pediatr Orthop 1993**. Publication with a substantial number of patients (other than Steel's original article) of successful treatment of residual dysplasia utilizing the Steel triple innominate osteotomies.

**Ganz R. A new periacetabular osteotomy for the treatment of hip dysplasia. Technique and preliminary results. Clin Orthop Relat Res 1988**. "The" article that introduced the Bernese PAO as an alternate to a triple innominate or dial osteotomy in correcting acetabular dysplasia.

**Wells J. Intermediate-term hip survivorship and patient-reported outcomes of periacetabular osteotomy: The Washington University experience. J Bone Joint Surg Am 2018**. Comprehensive review of both local,

national and international outcomes to date of treatment of acetabular dysplasia with the Bernese (Ganz) PAO.

## Key Evidence

The indications for performing either a pelvic redirectional osteotomy or an acetabuloplasty in attempting to correct acetabular dysplasia are dependent upon age and cause of the dysplasia. In the young child (during development of the cartilaginous anlage), indications for surgical correction are dependent on the functional capacity of the acetabulum to support weight bearing and hip range of motion. For these patients, radiographic evidence of acetabular underdevelopment and subluxation/instability (i.e. break in Shenton's line) or symptomatic dysplasia (pain or feeling of instability) is a clear indication for surgical intervention [9]. Given the predictable remodeling potential of the cartilaginous anlage, patients do very well following properly performed interventions [39, 76, 77]. In relatively older patients (following near complete development/ossification of the cartilaginous anlage), the indications are somewhat less clear for the surgical correction of residual acetabular dysplasia (Fig. 5.18). There is a paucity of both natural history and properly controlled interventional studies of hip dysplasia in this age group. In these more mature hips, there will be less potential remodeling following joint preserving surgery. Precise repositioning of the acetabulum is more critical in assuring satisfactory long-term outcome [8, 76, 77]. Most surgeons consider symptoms of dysplasia (pain, feeling of instability, and limping), a positive Trendelenburg sign on exam and radiographic evidence of subluxation as indications for corrective surgery in these more skeletally mature patients. Outcomes of surgical connection of acetabular dysplasia have been very favorable, with a reported 95% 15 year survival rate [92–95]. Younger patients and the preoperative absence of arthritis were predictors of potentially better outcomes. The status of the labral chondral complex and/or head-neck prominence as predictors of outcome is less clear. While techniques have been developed that make it possible to correct both labral chondral pathology and head-neck junction abnormalities



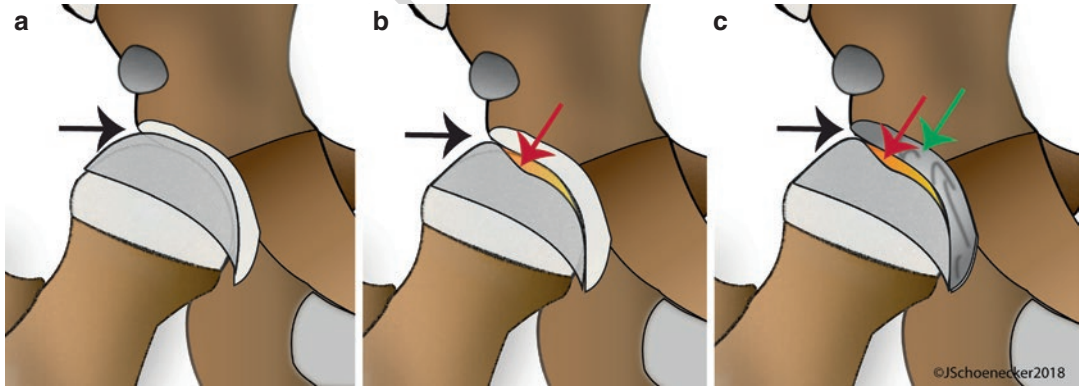



**Fig. 5.18** Current indications of the PAO: Indications for correction of insufficient lateral (black arrow) or anterior coverage (**a**) are continually evolving. Current evidence supports correction of a symptomatic under-covered (see Table 5.1 for values) hip. Current evidence also supports replacement, not preservation, of a (**c**) symptomatic dysplasia with a labral tear (red arrow) and osteoarthritis (green arrow). However, evidence is less clear as to the outcome of correcting incidentally discovered, asymptomatic dysplasia (**a**). Additionally, with the advent of combined arthroscopy, it is unknown if labral repair is required when addressing dysplasia, or, how much a tear is too significant to warrant surgical correction of both the labrum and dysplasia in favor of replacement

concomitant with acetabular reorientation, clinical studies are still ongoing in attempting to show how effective these adjacent procedures are (or are not) in further improving the outcome of acetabular reorientation joint preserving surgery.

**Take Home Message**

- In the last few decades, both our understanding of what leads to hip joint arthritis secondary to acetabular dysplasia and the ability to surgically correct it has changed the practice of our hip surgery.
- It is essential to be knowledgeable of the development of the hip and the processes by which it can fail when selecting a surgical approach in the correction of residual acetabular dysplasia.
- Having an understanding of both the normal and abnormal growth of the acetabulum helps in both the need for timing of and choosing the optimal surgical technique in correcting acetabular deficiency.
- Further observation is indicated for younger children (less than 7 years old) with a progressive decrease of the acetabular index, an intact Shelton's line, normal hip motion and no limp.
- Further observation is not indicated for older children (7 years and older) with an unchanging acetabular index, break in Shelton's line, and a positive Trendelenburg sign. Rather, appropriate surgical correction should be considered.
- For skeletally mature patients, surgical correction of residual acetabular dysplasia is selectively performed typically only for symptomatic patients.
- The clinical and radiographic goals at skeletal maturity, whether by natural history or following surgical intervention, include: lateral and anterior CEA of 25°, less than 20% of the femoral head laterally uncovered, a "stable" hip (i.e. Tonnis angle <10° and Shelton's line intact) and, most importantly, a congruent hip with satisfactory range of motion.

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# Author Queries

Chapter No.: 5 0004302441

| Queries | Details Required | Author's Response |
|---|---|---|
| AU1 | Please check and confirm if the affiliations are presented correctly. | |
| AU2 | As per reference style, name-date references have been changed to numbered references. Please check. | |
| AU3 | Ref. "Ponseti 1978" has been changed to "Ponseti 1978a, b" to match with the reference list. Please check here and in subsequent occurrences. | |
| AU4 | Part label (c) is present in the artwork but not mentioned in the caption of Fig. 5.7. Please check. | |
| AU5 | Refs. "Coleman 1968; Tönnis 1976; Albinana et al. 1996; Smith et al. 1997" are cited in the text but not provided in the reference list. Please check and provide the bibliographic details in the list or delete the citations from the text. | |
| AU6 | Ref. "Faciszewski et al. (1993)" has been changed to "Faciszewski et al. (1993a, b)" to match with the reference list. Please check here and in subsequent occurrences. | |
| AU7 | We have relabeled Fig. 5.8. Kindly check and provide better quality figures if any. | |
| AU8 | Please check the hierarchy of the section headings and confirm if correct. | |
| AU9 | Part labels (c–f) are present in the artwork but not mentioned in the caption of Fig. 5.11. Please check. | |
| AU10 | Part label (c) is present in the artwork but not mentioned in the caption of Fig. 5.17. Please check. | |
| AU11 | Part label (b) is present in the artwork but not mentioned in the caption of Fig. 5.18. Please check. | |
| AU12 | Reference "96" was not cited anywhere in the text. Please provide in text citation or delete the reference from the reference list. | |