Frederic Shapiro

Pediatric Orthopedic Deformities

Volume 2

Developmental Disorders of the Lower Extremity: Hip to Knee to Ankle and Foot

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To my wife, Carol Ann Satler

Preface

Pediatric Orthopedic Deformities**,** *Volume 2:* ***Developmental Disorders of the Lower Extremity – Hip to Knee to Ankle and Foot*** is composed of an Introduction and seven chapters. It focuses on the **hip** with chapters on developmental dysplasia of the hip (DDH), Legg-Calvé-Perthes disease (LCP), coxa vara including slipped capital femoral epiphysis (SCFE), and femoroacetabular impingement (FAI); disorders affecting the **knee**; **rotational and angular deformities of the lower limb** including lesions centered at the diaphyseal-metaphyseal regions; and disorders of the **foot and ankle** including club foot and congenital vertical talus. Volume 1 of *Pediatric Orthopedic Deformities* covered several topics[1], including lower extremity length discrepancies, as well as a detailed overview of the developmental biology of the skeletal system, an overview of how altered biology contributes to causation of deformity, and how the utilization of biologic and mechanical principles leads to correction of those deformities. Understanding epiphyseal and physeal biology is essential owing to its contribution to normal growth and development, to pathologic deformity, and to correction of deformity with growth. Volume 3 will discuss pediatric neuromuscular disorders and the treatment of neuromuscular, congenital, and syndromic scoliosis.

In the Introduction to Volume 2, we have provided a **Definition of Deformity,** a formal list of the 20 **General Principles Regarding Pediatric Orthopedic Deformity** (40 including subdivisions), and management **Implications of the General Principles of Deformity**.

For each deformity in the seven chapters, we provide a definition (terminology), detailed review of the pathoanatomy, experimental biological investigations (where applicable), natural history, review of the evolution of diagnostic and treatment techniques, results achieved with the various approaches, and the current management approaches (in text and tabular form) including detailed descriptions of surgical technique. The book is extensively illustrated to show the range of deformity for the various disorders, the underlying histopathology from human cases (and experimental models where available), imaging findings, and treatment approaches. This broad approach provides an extensive knowledge base regarding differing diagnostic methods, a detailed review of the underlying pathoanatomy of the disorder, the stage in its progression, the range of treatments, and their effectiveness. This combined information for each disorder improves the likelihood that the specific procedure or management approach chosen is applied at the correct time.

The two underlying premises of this volume remain the same as expressed in the preface to Volume 1. These are that ***(i) current orthopedic treatments of deformities of the developing musculoskeletal system are most effective when based on understanding and relating to the underlying pathobiology*** and ***(ii) future treatments are best developed by directly addressing the primary pathobiology.***

[1] Chapters in Volume 1: (1) Developmental Bone Biology; (2) Overview of Deformities; (3) Skeletal Dysplasias; (4) Bone and Joint Deformity in Metabolic, Inflammatory, Neoplastic, Infectious, and Hematologic Disorders; (5) Epiphyseal Growth Plate Fracture-Separations; and (6) Lower Extremity Length Discrepancies. A complete listing of the chapter content subsections can be found on the Springer website [springer.com] listing for *Pediatric Orthopedic Deformities*, Volume 1.

These premises are by no means original; as long ago as 1843, William Little, MD of London, England, stressed repeatedly in his "Course of Lectures on Deformities of the Human Frame" that "…. you can never treat a deformity with advantage to the patient or to your own satisfaction….unless you thoroughly understand the pathology of the case" (Lancet 1843; 41: 382-386). He published his course of 18 lectures on deformities in the *Lancet* in 1843–1844 and collected them in book form in 1853 (Lectures on Deformities of the Human Frame, London, Longman, Brown, Green and Longmans). For each entity discussed throughout our book, **the underlying pathology is described in detail.**

In the current environment, however, these basic premises while generally adhered to verbally are at risk of becoming overwhelmed by the flood of information published in a proliferating number of journals and discussed at innumerable courses. The current concentration on "best practices," "expert opinions," "evidence-based recommendations," "committee recommendations," "consensus reports," "peer-review committees," et cetera all provide meaningful direction for practitioners but risk taking the focus away from more primary studies. Discussion and formulation of "best practices," "evidence-based" approaches, etc. are to be encouraged; they are in fact derived from most of the same reference sources in the various chapters of the book and are included in the information base provided in the book. Bearing these considerations in mind, Volume 2 of *Pediatric Orthopedic Deformities* is designed (as was Volume 1) to provide the pediatric orthopedic surgeon, and those managing pediatric patients with orthopedic deformities, with the detailed knowledge base needed to manage patients independent of simply following consensus profiles. It also provides the detailed pathobiologic background needed to guide the evolving molecular, cellular, and biophysical approaches to managing pediatric orthopedic deformity.

The biologic and biophysical focus of the book provides clear understanding of investigations directed at major sites of clinical deformity. For example:

- Over the past two decades, significant strides have been made in understanding the pathogenesis and effects of avascular necrosis of the femoral head, primarily using experimental piglet models where ischemia is induced by intracapsular circumferential ligation at the base of the femoral neck. Subsequent studies with the model have improved diagnostic methods, led to understanding of both femoral head and secondary acetabular malformation, and helped assess molecular treatment interventions.
- Appreciation of malformations at the femoral head/acetabular interface leading to femoroacetabular impingement (FAI) has had major treatment implications for several disorders, particularly slipped capital femoral epiphysis. There was increasing awareness for several decades that hip osteoarthritis was rarely idiopathic but secondary to childhood hip deformity, even if mild; structural definition of the altered femoral-acetabular relationship, however, clarified the causes and led to the development of corrective interventions.
- Osteochondritis dissecans at the knee and talus are now addressed primarily via arthroscopy. Earlier intervention allows for limiting the damage done and, in many instances, for primary repair; severe involvement can now be addressed by attempting to induce articular cartilage repair by biologic cellular and tissue approaches.

The book is constructed to allow for inclusion of a knowledge base of the underlying pathoanatomy, natural history of the various disorders, and awareness of treatments that have had some effectiveness in the past as well as a detailed presentation of current treatment programs. While using treatments that experts or multicenter committees are recommending can be comforting and tends to raise the consistency of results, awareness of the history of management trends shows that using this approach to management exclusively can be shortsighted. It is a combination of application of knowledge of the underlying pathology of a disorder and appropriate utilization of biomechanical and biological principles in treating the disorder that will ultimately improve the results.

The continual change of management profiles in essentially all pediatric orthopedic disorders over relatively short periods of time cannot be attributed solely to a positive unidirectional flow of improvement. Considerable effort has been made in the book **reviewing the course of management over several decades.** This is not done as a simple historical exercise; rather it ***indicates the evolution of treatments*** pointing out where previous efforts were inadequate. Even where surgical techniques from one era are found not to be required as frequently now, awareness of a technique and its value can be applied fully or partially where newer approaches still leave deformity uncorrected. While some of the older operative procedures are rightly abandoned, others remain of value and need to be understood. Historical review becomes even more meaningful by ***also showing the cyclical nature of many management approaches*** where treatments abandoned as inadequate resurface decades later as treatments of choice. For example:

- Percutaneous tendoAchilles tenotomy for clubfoot deformity, following initial repetitive manipulation to correct the varus/adduction component and followed by lengthy periods of splinting and gentle daily manipulation to maintain the correction, was widely used by Stromeyer in Germany, beginning in the early 1830s, followed very shortly by Little in England, Guérin in France, and many others. This approach had considerable success over several decades and included the procedure that effectively launched the surgical component of pediatric orthopedic surgery. This treatment program subsequently fell into disregard to be followed by several decades of forceful manipulation for clubfoot deformity and a series of nonphysiologic open surgical procedures that, while resulting in apparently straight feet, caused considerable stiffness and deformity necessitating repetitive procedures. Even when Ponseti revived the initial manipulative/casting approach, the almost invariable use of percutaneous tendoAchilles tenotomy for correcting clubfoot equinus, followed by 2–3 years of night splinting, it again took a couple of decades for it to gain its current wide acceptance.
- For symptomatic Osgood-Schlatter disease of the knee nonresponsive to conservative management during the growth years, Makins in 1905 described a good result with a simple surgical procedure at skeletal maturity where the loose "osteocartilaginous nodule" of the tibial tubercle was removed and the soft tissues were re-apposed and sutured to the tibia (Lancet 1905; 166: 213-216). Over the next several decades, and continuing to the present, innumerable operative approaches other than simple loose ossicle removal described by Makins were used. These included bone drilling or autogenous bone grafting to get the ossicle to heal, insertion of ivory pegs at the tibial tubercle site to enhance fusion, excision of the tibial tubercle, longitudinal incision in the patellar tendon to relieve venous hypertension, decompression of tissue in the tubercle by arthroscopy, and (once again) simple removal of the ossicles at open incision. At present, most will now perform the procedure essentially described by Makins (removing loose ossicles) that yields rapid repair. This continuing circularity of management approaches is a feature of several of the conditions discussed in the book.
- Extensive clinical and experimental efforts beginning with Ollier in France in 1867 and prominent from the 1930s to the 1960s were done to stimulate long bone growth for limb length discrepancies by several methods: irritating the periosteum on the shorter side by subperiosteal stripping, elevating it with foreign objects, or cutting it circumferentially. (See Volume 1, Chap. 6, Sect. 6.9.3.) The resulting repair with increased vascularity stimulated the physes of the operated bones to overgrowth, and results were sometimes effective (0.5–2 cm overgrowth), but good responses were irregular and unpredictable and the techniques were abandoned. At present, there is renewed experimentation that cuts the periosteum circumferentially by non-operative means to induce overgrowth for unilateral limb discrepancies.

The questions raised by these examples relate to why, when results were seemingly so good in occasional cases, or at least worked to a certain degree in many, the profession widely abandoned the approaches instead of continuing to refine them with modifications. It is knowledge of the underlying pathoanatomy and the ability to deal with it in appropriate biological and biomechanical (biophysical) ways that sooner or later allows the correct approach to be used.

The **natural history of the various deformities** is described in detail. This too is not provided in a routine or automatic fashion; rather, combined with considerations of the underlying pathoanatomy, the two ***provide major signals regarding the timing for specific interventions as well as indications that observation alone may allow for spontaneous repair.*** Operations for a specific disorder may be "correct" (based on current understanding) in that they are applied for that specific disorder but ***could be considered to have been performed at the wrong time***, *too late* to yield a meaningful long-term result or *too early*, where good evidence exists that spontaneous growth-related correction alone would most likely have caused improvement.

It is the **application of biological and biomechanical treatment principles** that allows for optimal management. This especially applies to understanding the growth mechanism at each region that plays a major role in both causing and correcting pediatric musculoskeletal deformity.

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He thanks Joy Wu, MD, PhD, Department of Medicine/Endocrinology, for appointment in her bone biology laboratory as a Visiting Scholar at Stanford University School of Medicine, Palo Alto, CA. The author is a pediatric orthopedic surgeon who worked at the Boston Children's Hospital, Boston MA USA, clinically in the Department of Orthopaedic Surgery (attending orthopedic surgeon) and doing basic science research in the Orthopaedic Research Laboratory (Laboratory for the Study of Skeletal Disorders). His Harvard Medical School, Boston MA USA, appointments progressed from Research Fellow to Instructor, then Assistant Professor of Orthopaedic Surgery, and ultimately Associate Professor of Orthopaedic Surgery.

Contents

Introduction. Volume 2

Deformity *is a deviation in structure and/or position from normal.*

General Principles Regarding Pediatric Orthopedic Deformity

Several general principles underlie the development and management of musculoskeletal deformity in the pediatric age group. Awareness of this listing of 20 basic principles (40 including subdivisions) provides guidance for the diagnosis of deformity, following patients, timing treatments, and continuing assessments until skeletal maturity.

1. The shape of individual bones and the alignment between adjacent bones and regions often change normally with growth especially in the first few years after birth. It is important to be aware of developmental patterns that are normal physiologic variants and not pathologic deformities. For example, (a) 40° anteversion of the femoral head and neck at birth is common and will decrease with normal growth to 12–15° in late adolescence, while 40° anteversion in an adolescent regardless of cause will not correct spontaneously and can be clinically problematic for the future; (b) bow leg positioning (genu varum) of 30° is almost always a normal, self-correcting physiological position in a 14-month-old but is highly likely to be pathologic in the adolescent; and (c) kyphosis is the normal sagittal plane position at cervical, thoracic, and lumbosacral regions in the newborn spine; the cervical region begins to develop its normal lordosis at 3–6 months of age (as the infant crawls holding the head upright), while lumbar lordosis develops after 1 year of age (with walking in the standing position); both cervical and lumbar kyphosis in juvenile and adolescent years are true deformities with negative clinical consequences.
2. There is a range of normal angles and rotations within individual bones and between adjacent bones and regions. Variations within these normal ranges can be relatively wide and should not be interpreted as deformities.
3. Deformity may be evident by clinical observation alone (such as moderate to severe scoliosis or clubfoot deformity), by plain radiography (such as coxa vara of the hip or varus of the distal femur and valgus of the proximal tibia causing knee joint obliquity), or by more sensitive imaging modalities such as ultrasound, magnetic resonance (MR) imaging, or computerized tomographic (CT) scanning.
4. Gene mutations cause intramolecular and intermolecular malalignments (molecular deformation) leading to abnormal tissue patterning and development and eventual musculoskeletal deformation. While these abnormalities are not commonly considered to be deformities, they do deform the involved molecules and are increasingly viewed as intracellular and extracellular deforming forces; examples affecting the skeletal system are signaling molecules of the Notch group (affecting early patterning) and structural molecules like collagen. Common examples of gene mutations causing significant clinical deformation in the musculoskeletal realm include skeletal dysplasias, osteogenesis imperfecta, peripheral neuropathies (Charcot-Marie-Tooth disease), congenital scoliosis, and muscle disorders (Duchenne muscular dystrophy). In children with deformities, it is now

necessary to consider abnormalities of chromosomes, such as Down syndrome with an extra copy of chromosome 21 (Trisomy 21) or genetic point mutations, deletions, or insertions on specific molecules as primary causative deformities; examples include collagen abnormalities leading to osteogenesis imperfecta, dystrophin abnormalities leading to Duchenne and Becker muscular dystrophy, peripheral myelin protein 22 (PMP22) abnormalities leading to Charcot-Marie-Tooth peripheral neuropathy, and delta-like 3 (*Dll3*) mutations of the Notch family leading to congenital scoliosis with rib malformations.

5. A pathologic deformity during the growing years may proceed along one of the three pathways: it may correct spontaneously with growth, it may remain unchanged with growth, or it may worsen with growth.
6. Each of the main mechanisms of spontaneous correction of a bone deformity in a growing child can be seen with remodeling following a physeal, metaphyseal, or diaphyseal fracture that heals with angulation or malrotation but an intact functional physis. Repair mechanisms involve (a) differential physeal growth tilting and rotating the physis to the normal plane, (b) periosteal new bone formation on the concavity of the deformity, and (c) bone resorption on the convexity. These mechanisms are more effective the greater the number of years of growth remaining, the closer the angular deformity is to the physis, the more the physis involved contributes to the growth of the bone, and the presence of deformity in the plane of motion of the adjacent joint.
7. If persistence of a deformity or its worsening with growth is likely, the deformity must be assessed as problematic or non-problematic over the short, intermediate, and long (adult years) time frames. Problems resulting from deformity include some or all of pain, abnormal function such as gait disturbance, associated organ impingement (cardiac, pulmonary), or appearance. Also to be considered for any deformity is what degree of deformity is associated with what amount of clinical problem.
8. There is a constant interplay of forces during the growing years between the soft tissues [muscle, tendon, ligament, intervertebral disc] and the developing bones [composed of cartilage and bone tissue]. Any abnormality in either the soft tissue or the developing bone due to imperfect structure or function can lead to deformity.
    - 8.1. For any deformity it is essential to determine whether it is flexible or rigid. If it is flexible, there is a need to know if it fully straightens, or at a joint fully reduces, with change of position or passive manipulation or if it only partially corrects. Conversely, if rigid there is need to determine whether it is completely rigid or if manipulation partially straightens or reduces the deformity. The more rigid the deformity, the more extensive the surgery needed to correct it and, in the case of the spine, the less correction attainable.
    - 8.2. Primary soft tissue deformities, including neuromuscular disorders, are initially associated with normal cartilage model/bone model development. The longer the deformity persists and the more severe the deformity, the greater the likelihood that asymmetric pressure on the skeletal elements involved and the lack of normal functional stress will lead to structural deformity of cartilage and bone (and intervertebral joints in the spine).
        - 8.2.1. Excess muscle pull may be due to localized muscle weakness (with the adjacent normal muscles continuing to function) or to localized muscle over-reaction due to spasticity (that overpowers the adjacent normal muscles). Either occurrence leads to malposition of joints and distal structures or disturbances of normal spinal alignment. The malposition or abnormal alignment may correct with muscle relaxation or muscle-tendon transfer to balance strength (e.g., across a joint) but will recur with continuing asymmetric activity and eventually lead to cartilage and bone deformity with growth.
        - 8.2.2. Ligamentous laxity leads to malpositioned joints that are initially present only in the upright position or with weight-bearing but correct to normal anatomic positioning with the recumbent position or non-weight-bearing. If left uncor-

rected in the presence of continuing growth, the malposition can progressively become rigid, not actively or passively correctable, and even associated with misshapen bones.

8.2.3. Asymmetric soft tissue tightness can lead to rigid joints (contractures) and distal structure deformation that with continuing malposition eventually leads to developing bone deformity with growth.

8.3. Bone/cartilage deformity has varying relations to the orientation of adjacent cartilage surfaces.

8.3.1. Angular bone deformity centered at metaphyseal or diaphyseal regions leads to asymmetric joint alignment (obliquity) at one or both ends of an individual bone with a predisposition to abnormal intra-articular pressure and articular cartilage osteoarthritis.

8.3.2. Asymmetric growth plate function due to focal physeal abnormality leads to angular growth of the bone and joint obliquity distal and sometimes proximal to the involved physis that predisposes to abnormal intra-articular stresses.

8.3.3. In rare instances there is bidirectional angular deformity (or bowing) within a bone owing to curvatures within the metaphyseal-diaphyseal regions. This can occur in patients with osteogenesis imperfecta or rickets. For example, a valgus angulation of the tibia centered at the proximal metaphysis can reverse direction by curvature in the mid-diaphysis leading to varus angulation centered at the distal metaphysis. In some instances there can be normal articular surface alignment persisting at both ends, while in others the angular deformity also tilts the physis, epiphysis, and articular surfaces into an oblique plane.

8.4. Correction of deformity due to soft tissue abnormalities will often allow for spontaneous growth-related correction of mild to moderate cartilage model/bone deformities.

8.5. Neuromuscular deformities with both soft tissue and cartilage model/bone deformation usually require soft tissue correction (balancing of muscle groups) and might need bone correction by osteotomy or asymmetric physeal stapling.

8.6. Repositioning of dislocated or partially dislocated (subluxed) joints by non-operative or operative means often allows mild to moderate cartilage model/bone deformities to correct with growth. The younger the patient is and the more years there are to skeletal maturation, the greater the likelihood for cartilage/bone correction with growth after repositioning.

8.7. Normal function or improved function posttreatment allows the normal stresses on immature tissues to further correct the remaining deformity by differential growth toward a normal position.

8.8. Deformities whether congenital or acquired of intra-articular soft tissue structures, such as knee menisci and cruciate ligaments and the hip labrum, if allowed to persist can damage articular cartilage. The damage can occur directly by causing abnormal contact or indirectly by causing joint instability. These deformities can be suspected by clinical history and examination but are diagnosed definitively by direct arthroscopic visualization or by MR imaging.

9. Trauma to a developing bone can lead to deformity in several ways.

9.1. Acute diaphyseal or metaphyseal fractures that heal with angulation, malrotation, translation, or length discrepancy represent deformities that, with growth, may correct partially or fully, persist unchanged, or (rarely) worsen and require surgical intervention for correction.

9.2. Acute physeal (growth plate) fractures (or fracture-separations) can heal with no subsequent growth sequela or with partial or complete growth plate fusion predisposing to angulation and/or shortening with growth.

9.3. Chronic repetitive stresses on growth plates can lead to growth damage and premature growth plate fusion. Chronic repetitive stresses on tendon insertions in growing bones can lead to non-displaced tendon-cartilage-bone interface avulsions with painful tendinitis and insertion site swelling.

9.4. Articular cartilage damage either by displaced osteochondral fractures or linear oblique intra-articular fractures with persisting surface irregularity or gaps predisposes to arthritic changes.

10. Bone deformities are not passively correctable. They may, however, spontaneously remodel with growth, be correctable with growth with the use of serial casting or bracing, or eventually require surgical intervention by metaphyseal/diaphyseal osteotomy, asymmetric physeal stapling, or vertebral body tethering for correction.
11. Once a deformity is identified, the presence of a specific disease or an underlying causative disorder must be considered. Treatment can differ significantly for the same deformity: (a) in a patient who is otherwise normal and in one who has an underlying disorder and (b) between patients with different specific diseases.
12. It is important to distinguish between a primary deformity and a secondary or compensatory deformity. The primary (or initial) deformity is at the site of pathologic abnormality and tends to be at least partially fixed or rigid. The secondary (or compensatory) deformity tends to be fully flexible initially and for a longer period of time since it represents a process within normal adjacent regions designed to maintain stability, balance, and alignment. A long-standing secondary deformity may become rigid with time. In the spine, if the secondary curves above and below the primary curve remain flexible, trunk balance compensation occurs (curve in the opposite direction), and the head above and pelvis below remain level; if the primary curve above involves the entire cervical spine, compensation cannot occur and the head is tilted, and if the primary curve below involves the entire lumbar spine, compensation cannot occur and the pelvis is tilted (pelvic obliquity).
13. A static joint deformity is present at all times and in all positions (e.g., supine, standing) owing to its associated bone or soft tissue rigidity. A dynamic joint deformity is present only with muscle activity since it is due to muscle imbalance. Dynamic deformities commonly occur with gait or attempted upper extremity activity with associated asymmetric muscle under- or overactivity.
14. Some deformities are sufficiently mild that no treatment is warranted since they cause no current problems and there is little convincing evidence that they will cause problems in the future.
15. Treatments for some childhood deformities may not be warranted even if they are moderate to severe in view of the severity and progressive nature of the primary underlying pathologic process such as a neurodegenerative disorder even though treatment of the same deformity in an otherwise normal or only mildly involved child would be warranted.
16. Deformity even if marked may be a secondary compensatory deformity necessary to maintain function such that its correction to a normal anatomic position may significantly decrease function. Marked lumbar lordosis in a patient with neuromuscular disease with gluteus maximus muscle weakness can allow a person to continue walking, whereas spinal bracing or fusion straightening the spine can make the patient anatomically normal but unable to walk. Compensatory deformities are common in the spine above and below regions of primary scoliosis or kyphosis.
17. Treatment in childhood is warranted for deformities that will not correct on their own if they are already symptomatic, causing discomfort or altered function, or if they are asymptomatic but there is scientific evidence that they will become symptomatic with time, even if that will be during the adult years.
18. Once a deformity is considered treatable, two general approaches follow. The first is to treat any existing primary underlying disorder by medical means, for example, infection with antibiotics, hemophilic arthropathy with factor replacement, rickets with vitamin D,

and spastic cerebral palsy with muscle relaxant therapy. In the future, this principle will also apply to direct treatments for gene and molecular deformation. The second is to treat the deformity itself by orthopedic means.

19. Orthopedic treatment can be non-operative or operative.
    19.1. The variety of non-operative therapies is great and includes rest, range of motion and stretching exercises, pharmacologic agents for pain relief and muscle relaxation, and serial casting or bracing to stretch tightened soft tissues and position the deformed part straight to allow normal cartilage and bone growth to occur.
    19.2. The ranges of operative therapy are also great. Some operations cure deformity permanently with one intervention. Other procedures correct one aspect of the deformity and then rely on spontaneous growth or repositioning for full correction of adjacent structures. Some deformities need a series of operations over a short or longer period of time in the growing years since (a) the underlying cause may persist, (b) the damage done to the growing structure may itself lead to recurrence, or (c) soft tissue and bone procedures may be needed for correction but are best done at different times in the growth period.
20. Once a deformity in childhood has been corrected, it may follow one of the three pathways during the remaining years of growth: (a) the region involved may remain anatomically normal, and no further management is ever needed; (b) the deformity may recur due either to the fact that it had not been fully corrected or the underlying disorder that caused it persists; and (c) the deformity may overcorrect leading to deformation in the opposite direction due to such factors as a new pattern of muscle imbalance, asymmetric growth plate function, or continuing growth correction resulting in overgrowth. This variability leads to the advisability for following patients with a pediatric orthopedic deformity until skeletal maturity.

Implications of the General Principles of Deformity

1. **Close relationship between skeletal growth and pediatric orthopedic deformity can be either beneficial or detrimental.** Some deformed positions are actually ***physiologic*** and *correct spontaneously* with growth; ***pathologic deformities*** can *correct, remain the same, or worsen* with growth; and ***deformities that have apparently been corrected*** can *remain straight*, *worsen with recurrence* (due to unrecognized under-correction or persistence of the underlying disorder), or even *overcorrect* (due to altered muscle or bone formation balance) with growth.
2. **Several concepts must be assessed when managing pediatric orthopedic deformities.** These include a ***range of biologic variation*** within a bone and the angular relationship between adjacent bones; ***primary versus secondary/compensatory*** deformity; ***rigid versus flexible*** deformity; ***static*** deformity (present regardless of position) ***versus dynamic*** deformity (present with muscle function); ***interplay between soft tissues*** *(muscle, tendon, ligament, intervertebral disc)* ***and growing bone/cartilage models***; and ***relationship between primary disease and secondary orthopedic deformity***, such as rickets and bowed femur/tibia; hemophilia and knee, elbow, and ankle synovitis; and septic arthritis of the hip causing avascular necrosis of the femoral head.
3. **Presence of deformity can lead to variable approaches to management.** ***Major surgical intervention*** may be warranted even with minimal deformity based on *natural history studies of invariable worsening with time*; the ***same deformity seen in different patients may warrant differing approaches***: *surgical correction in an otherwise normal person* versus *no surgery in one with a progressive neurodegenerative disorder*; and ***surgery to correct major secondary compensatory deformities may be contraindicated*** such as *spinal fusion for lumbar lordosis in an ambulatory neuromuscular patient* that *worsens function while achieving anatomic straightening.*

Developmental Dysplasia of the Hip

1

1.1 Terminology

Developmental dysplasia of the hip is a general term referring to a spectrum of deformities, usually diagnosed in the neonatal period, in which the structural relationship of the proximal femur to the acetabulum is intermittently or continuously abnormal. The spectrum includes (i) a subluxatable or dislocatable hip associated with capsular laxity in which the head of the femur moves partially or totally out of the acetabulum with extension and adduction and back into it with flexion and abduction, (ii) a subluxated hip in which there is a partial but persisting loss of the normal relationship of the head of the femur to the acetabulum in extension with the head more lateral than normal in the acetabulum and the acetabulum more shallow than normal with its lateral roof angled outwardly and upwardly, and (iii) a dislocated hip in which there is a complete and persisting loss of any femoral head-acetabular relationship, regardless of the position of the hip. Developmental dysplasia of the hip (DDH), as currently defined, is not associated with clinically evident connective tissue, neuromuscular, or other diseases. The single most important initial pathoanatomic change appears to be a capsular laxity which renders the hip unstable at birth with all subsequent abnormalities being secondary phenomena which develop an increasing variation from the norm the longer a hip is allowed to grow with any persisting malposition. The terminology used to describe this condition has always been variable and imprecise primarily due to the imperfect understanding of the pathoanatomy and timing of its initial occurrence.

Congenital dislocation of the hip (CDH) was used previously to describe the entity, although some used the term congenital dysplasia of the hip to encompass the entire spectrum of the disorder. Dunn defined congenital dislocation of the hip as an "anomaly of the hip joint, present at birth, in which the head of the femur is, or may be, partially or completely dislocated from the acetabulum" [1]. The entity is now referred to as developmental dysplasia of the hip (DDH). Developmental has replaced congenital since (i) it focuses on abnormalities in development which predispose to the condition and which 'worsen in the absence of normal hip positioning and (ii) it is not definite that all dysplastic hips were structurally abnormal and/or detectable at the time of initial postnatal examination. Dysplasia is a vague general term referring to a poorly defined disease process. Delayed, and thus imperfect, development of the acetabulum and of the proximal femur is referred to as a dysplastic process. Acetabular dysplasia and proximal femoral dysplasia themselves are either primary disorders and/or disorders which occur secondary to growth in the presence of undetected and untreated developmental hip disease.

Developmental dysplasia of the hip therefore encompasses a spectrum of hip abnormality. These include (i) an initial subluxatable or dislocatable hip in which the femoral head is located in a normal relation to the acetabulum in certain positions (generally flexion and abduction) but has a partial or complete loss of continuity in other positions; this situation can spontaneously correct itself within a few days of birth or it can progress if untreated to persisting deformity, (ii) a subluxation of the hip which refers to a partial loss of continuity between the femoral head and acetabulum where the abnormal relationship is present throughout the entire range of movement, and (iii) a dislocated hip with complete loss of continuity between joint surfaces at all times regardless of the position of the hip. Some refer to an unstable hip detected clinically on initial screening in the newborn nursery as having "neonatal hip instability" (NHI). Terminological distinctions are not merely a semantic issue; imprecise use of terms implies imprecise understanding of the underlying pathoanatomy that can lead to investigations and treatments which are not fully appropriate.

1.1.1 Change in Terminology

Klisic wrote a brief report in 1989 strongly supporting the use of DDH (which he defined as developmental displacement of the hip) to refer to the entire entity of hip dysplasia, subluxation, and dislocation [2]. He felt that the widely used

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term CDH (congenital dislocation of the hip) was inaccurate since it suggested a gross prenatal malposition demanding orthopedic correction. In reality the term DDH was preferable since it indicated a dynamic disorder capable as the child developed of getting better or worse. Klisic acknowledged the role of Michele who had used the term developmental hip dislocation as the title of his chapter on hip dysplasia in his book *Iliopsoas: Development of Anomalies in Man* in 1962 [3]. Michele recognized that a small number of dislocations (~2%) were congenital originating as embryologic defects in the germplasm but that the vast majority (~98%) occurred in an otherwise normal fetus at 6–9 months of uterine life due to a failure of stimulus to normal growth leading to what was in effect a "developmental dislocation." He felt that "congenital" should refer only to the atypical teratological cases, while the typical acquired environmental-anthropological cases should be referred to as "developmental" dislocations.

The term DDH is now widely accepted, but CDH or CDH/DDH will be used in discussing articles written using the CDH terminology.

1.2 Development of the Hip: Embryonic and Fetal Periods

1.2.1 Earliest Developmental Biology of the Hip. Chick Embryo Studies

As long ago as 1883, Johnson outlined the earliest development of the pelvic girdle, hip region, and hind limb in the chick embryo [4]. "The future cartilage is only just distinguishable from its surroundings of indifferent (undifferentiated) mesoblastic cells." "We can clearly distinguish three elements in the girdle meeting in the broad acetabular region, which passes on without a break into the femur….. the cartilage of the femur is continuous with that of the girdle, as are the three elements of the girdle with one another (ilium, ischium, pubis)." Only after structural development of the femoral-acetabular components was relatively well established "the femur begins to be separated from the girdle by an intervening tract of tissue" (meaning the cellular interzone which then is removed by the joint cavitation process).

Chevallier using chick and quail embryonic transplants demonstrated that the bones of the pelvic girdle originated from the somatopleural mesoderm which was shown to be regionalized as early as 2 days incubation, even prior to somatic segmentation [5]. The eventual cartilage centers of the three bones composing the acetabulum were preformed as a uniform mesenchymal condensation at 5 days but separate centers for the ilium, ischium, and pubis formed at 8 days which was shown to be regionalized as early as 2 days incubation.

Malashichev et al. performed two studies on the early embryogenesis of the pelvic region and its genetic components [6, 7]. One study demonstrated that ectodermal signals occurred at pre-limb bud stages for pelvis formation and that the regulation of ilium development differs from that for pubis and ischium [6]. *Emx2* was shown to be required for formation of the ilium but not the other two components. When the ectoderm over the somatopleure was removed, there were severe defects in the pelvic skeleton, but the defects differed depending on the time of intervention. The diverse pelvic elements appeared in temporal sequence of the ilium, pubis, and ischium. *Emx2* was expressed in regions giving rise to the ilium and *Pax1* in regions for the pubis, but these were restricted to times prior to chondrogenesis. In a second study [7], the entire pelvic girdle originated from the somatopleure with no somitic cell contribution to the pelvic skeleton. Ectodermal signals controlled development of the pelvis however, especially pubis and ischium. *Pax1* and *Alx4* modulated normal ischial and pubic development. It became evident that while *Emx2* expression helped direct formation of the ilium, signals from both ectoderm and somites were needed to complete development of the ilium. The chick pelvis thus originates from lateral plate mesoderm, but its development requires signals from overlying ectoderm and (for the ilium) paraxial mesoderm/somites.

Nowlan and Sharpe assessed chick embryo hip joint morphology not only by histology but also by a more sensitive technique of direct 3D capture using optical projection tomography (OPT) assessing tissue-specific markers allowing for earlier evolving structural assessments [8]. This enabled them to determine that major anatomical features of the developing hip were present a full day prior to joint cavitation. This included demonstration that rotation of the pelvis with respect to the femur in advance of cavitation (which allows for the effects of motion).

1.2.2 General Aspects of Human Hip Development

The embryonic period in the human refers to the first 8 weeks of development during which time each of the organs including the cartilaginous models of the long bones and vertebrae have formed. By the end of the embryonic period, the average embryo is of 3 cm crown-rump (CR) length. The fetal period from 8 weeks of age to birth is associated with increase in size and organ differentiation.

Watanabe outlined hip development in 288 hips from 144 embryos and fetuses from 14 to 300 mms CR length ending at 24 weeks gestation [9]. The femoral head diameter around 11 weeks was 2 mms at which time the joint space was formed and the head could be dislocated by cutting the capsule. The femoral head diameter by 24 weeks was around 8 mms. The diameter of the femoral head increases in size in a linear pattern and parallels the growth of the entire body. The femoral head is spherical at the beginning of development and remains so throughout growth. The neck-shaft angle was 130 during fetal development. Femoral anteversion averaged −4° from 10 to 15 weeks, 5° from 15 to 20 weeks, and 11° from 20 to

24 weeks, but there was a wide range of variability at these times of both positive and negative values. At birth femoral anteversion had increased to 35°. Watanabe's study found no examples of full dislocation, but there were 26 dysplastic hip joints characterized by "an overall hypoplasia of the entire hip joint with a shallow acetabulum." The femoral head was always stable with flexion and tended to subluxate with extension. The femoral head and acetabulum had reached infantile shape prior to joint space formation such that dislocation could not occur during the embryonic period.

Strayer studied hip development from human embryos 6.5–237 mms crown-rump length [10]. He concluded, in agreement with other observers, that all elements of the hip joint differentiate in situ in a mass of blastema. The head of the femur is globular (spherical) in shape at all times during its development, and the relative proportions of the blastemal and early cartilage developmental segments of the pelvic bones entering into formation of the acetabulum are the same in early embryos as in fetal stages and postnatal life. The ligamentum teres develops in situ within the joint. Congenital dislocation of the hip cannot occur before the opening of the joint cavity. The synovial lining does not develop as a cellular ingrowth but rather from cells in situ as part of the original blastemal mass. The synovium forms along the line of cleavage that appears between cells as the interzone tissues are liquefied. The acetabulum develops by growth and fusion of processes from the iliac, ischial, and pubic cartilages. Each of these develops around the femoral head, and their fusion initially produces a shallow acetabulum. The portion of the acetabulum to which each pelvic cartilage contributes is approximately the same as those later furnished by the corresponding pelvic bones being 2/5 ischium, 2/5 ilium, and 1/5 pubis. Each of the pelvic cartilages has a centrifugal growth pattern within the blastema. The region that will become the hip joint is composed initially of dense blastemal tissue referred to as the interzonal tissue, while the embryo is growing from 20 to 30 mms in length. Cavity formation begins in the tissue between the cartilage of the acetabulum and the cartilage of the head of the femur. The interzone tissue other than the ligamentum teres becomes looser in texture with time and ultimately is resorbed to leave the joint cavity.

Other Studies The greater trochanter is evident at 30 mms and the femoral neck and lesser trochanter at 34 mms [10]. The hip joint cavity according to Moser appears first in the lateral part of the joint at 34 mms [11]; Haines described an initial cavity at 34 mms [12]. The ligamentum teres develops in situ with Moser describing it as early as 20 mms and Strayer noting it at 23 mms. The glenoid labrum of the hip joint was noted at 30 mms as a transition with the outer region of the acetabular cartilage.

Dimeglio et al. reviewed prenatal hip development stressing the unique interrelationship of the pelvis, femur, and associated muscles on normal structure [13]. They stressed in particular three aspects of growth: (1) enlargement and full development of the acetabulum, (2) harmonious spherical enlargement of the femoral head and its secondary ossification center, and (3) elongation of the femoral neck in the postnatal period.

Detailed observations on the prenatal development of the human hip joint were provided by Gardner and Gray [14] in a study based on 52 human embryos and fetuses ranging from 12 mm crown-rump length (6 weeks) to 370 mm (term) and by Andersen [15] in a study of 30 human embryos-fetuses from 20 mm (7–1/2 weeks) to 121 mm (16 weeks). Their observations are in good agreement and are combined below.

1.2.2.1 Origin of Limb Bud

The lower extremity limb bud is seen in embryos 3–4 mms in length as a small protuberance on the anterior and lateral aspect of the body wall at the level of the lumbar and first sacral segments. The specific tissue differentiation for each bone then follows from blastemal tissue or undifferentiated mesenchymal cells, to precartilage, to cartilage, and then to bone. The region of the future hip joint appears as a group of densely packed undifferentiated cells in the form of a cone with an oblique base applied to the side of the body. The first appearance of the acetabulum is in the 14–15 mm embryo as a line of cells of diminished density proximal to the head of the femur. This region was initially felt to represent an arc of 65–70° which subsequently deepened to enclose a full half circle of 180° as the joint cavity formed. The interzone demonstrates increased cell density by 15–22 mms. Early differentiation of the ligamentum teres and periarticular capsular structures is noted around 23 mms. As development and growth proceed from 23 to 45 mms, the cartilage of the ilium grows out over the head of the femur with the labrum continuous with its outer margin. Increases in the extent of the elements of the acetabulum are responsible for the relative lateral displacement of the labrum. The acetabulum is never flat; from the earliest stages, it extends, together with the labrum, beyond the midway point of the head and always has a concave shape. Differentiation of blastema in the innominate region begins in the ilium just above the acetabulum at the 15 mm stage. This most lateral region lags behind the shaft and head of the femur in differentiation at all stages. The three cartilage centers become vascularized separately and serve to outline the triradiate (or Y) cartilage. Chondrification radiates from the three centers of these regions of the ilium, ischium, and pubis. Endochondral ossification then occurs within the central regions of these cartilage masses: ossification starting at 9 weeks in the ilium, at about the fourth month of gestation in the ischium, and a few weeks later (fifth month) in the pubis [16]. The triradiate cartilage for endochondral growth lies between the bony centers.

1.2.2.2 Acetabular Labrum and Transverse Acetabular Ligament

The acetabular labrum (often referred to in the older literature as the glenoid labrum of the hip joint) is formed at the earliest stages of the formation of the acetabulum as early as

19 mms and appears histologically as a condensation of blastema at the cartilage periphery [10, 14, 17]. By 25 mms it is clearly differentiated. It becomes vascularized at 61 mms. The transverse acetabular ligament also forms during this time period; the site of the transverse ligament of the hip joint is considered by Strayer to be the weakest point of structure. By 28 mms a condensation for the transverse acetabular ligament is seen, and by 30–33 mms the ligament is well defined. The superior labrum covers the widest diameter of the femoral head. The anteroinferior part of the acetabulum, which is known as the acetabular notch, is covered by the transverse acetabular ligament [17]. This ligament is the support for the acetabular labrum as it crosses the notch.

1.2.2.3 Joint Capsule and Synovium

In 12–15 mm embryos, avascular blastemal tissue in the region of the future joint is denser than that of the adjacent anlagen. This density is more pronounced at 17 mms with a definite interzone present. The interzone is more definite by 20 mms, and it is possible to define a three-layered interzone, the middle layer of which is directly continuous with the mesenchymal tissues surrounding the joint except in those areas of capsular condensation. The outer layers of the interzones are directly continuous with the perichondrium of the femoral and acetabular anlagen. The capsule surrounding the joint is defined. Contained within it is a portion of the mesenchyme surrounding the joint that is structurally a part of the interzone. This intra-articular mesenchyme is the first indication of what will become synovial mesenchyme. The intermediate layer of the interzone is continuous with the blastemal synovial mesenchyme, and both are vascularized. The three-layered arrangement of the interzone is more pronounced at 22–25 mms. Early spaces form within the middle layer. By 30–33 mms a clear cavity is present around the periphery of the joint. Even at the time of opening of the joint space, it is not possible morphologically to distinguish between the cells of the inner margin of the capsule that will eventually form the synovial membrane and the capsule itself. The first indication of the fibrous capsule is seen at 20 mms with a condensation appearing as a direct continuation of the perichondrium of the femur and pelvis.

1.2.2.4 Joint Cavity

Joint cavity formation represents a programmed degenerative and mechanical process with no evidence of ingrowth of tissue from the outside to provide a lining for the joint. Early evidences of degeneration are seen at 23 mms with increases in the intercellular spaces in the interzonal cells between the head of the femur, the ligamentum teres, and the acetabulum. At 36–42 mms spaces filled with fluid are formed. Andersen times formation of the joint cavity between 34 and 42 mms [15]. Vascularization of the interzone is an integral part of joint cavitation. Joint cavitation begins in the central area of the joints and then moves toward the periphery [12]. Cavity formation at the hip takes place as an annular rim, limited medially by the head of the femur and laterally by the acetabular/glenoid labrum. The ligamentum teres remains in the middle of the developing joint cavity. In later stages of cavitation, the space is enlarged centrally around the ligamentum teres and peripherally passing beyond the tip of the labrum and surrounding the head in its entirety and also the neck distally to the capsular insertion.

1.2.2.5 Retinacula of Weitbrecht

The extension of the joint space down the neck of the femur leaves as intracapsular structures the perichondrium, the retinacula of Weitbrecht, and the ascending cervical vessels. The retinacula of Weitbrecht are intracapsular flattened band structures of the hip joint present on the interior of the capsule and passing toward the margin of the femoral head. The retinacula are synovial-covered capsular reflections or prolongations [18]. The blood vessels eventually supplying the proximal femur perforate the capsular attachment at the base of the neck and pass along the surface of the neck entering the metaphysis of the neck and the epiphysis of the head through small foramina. Walmsley continues: "From the points where they perforate the capsule these vessels derive and carry inwards indefinite fibrous prolongations of the capsule wall which are covered over or are completely invested by reflections of synovial membrane. These elements constitute the retinacula of Weitbrecht." The fibrous prolongations terminate at varying distances from their origins, while the synovial reflections covering the vessels continue toward the cartilaginous margin of the head. The retinacula are reflections or continuations of the synovial membrane combined with fibrous sheath prolongation of the capsular wall which carry within themselves the blood vessels of the head and neck.

1.2.2.6 Ligamentum Teres

At 22 mms the first suggestion of the ligamentum teres is found. The ligamentum teres is present in 22–25 mm specimens as a region of greater cellularity but is not sharply demarcated from the neighboring interzone. There is never any evidence of a depression in the head to receive the ligamentum. The separation of the ligamentum teres to form a free mass within the joint occurs simultaneously with the opening of the remainder of the cavity which is characterized by peripheral vascularization, degeneration, and splitting between the cells along its margin. The ligamentum teres is well defined in the 30–33 mm fetuses. Blood vessels are first noted within the ligamentum teres at 60 mms. The ligamentum teres originates broadly from each side of the acetabular notch and from the transverse acetabular ligament. It is attached to a depression on the femoral head just below and posterior to the center of the head [11, 17].

1.2.2.7 Extra-articular ligaments

The hip receives additional stabilization from its extra-articular ligaments. Anteriorly and superiorly support is derived from the iliofemoral ligament, referred to as the Y-ligament of Bigelow or the ligament of Bertrand (Fig. 1.1a). Posteriorly, support comes from the ischiofemoral ligament, the lower

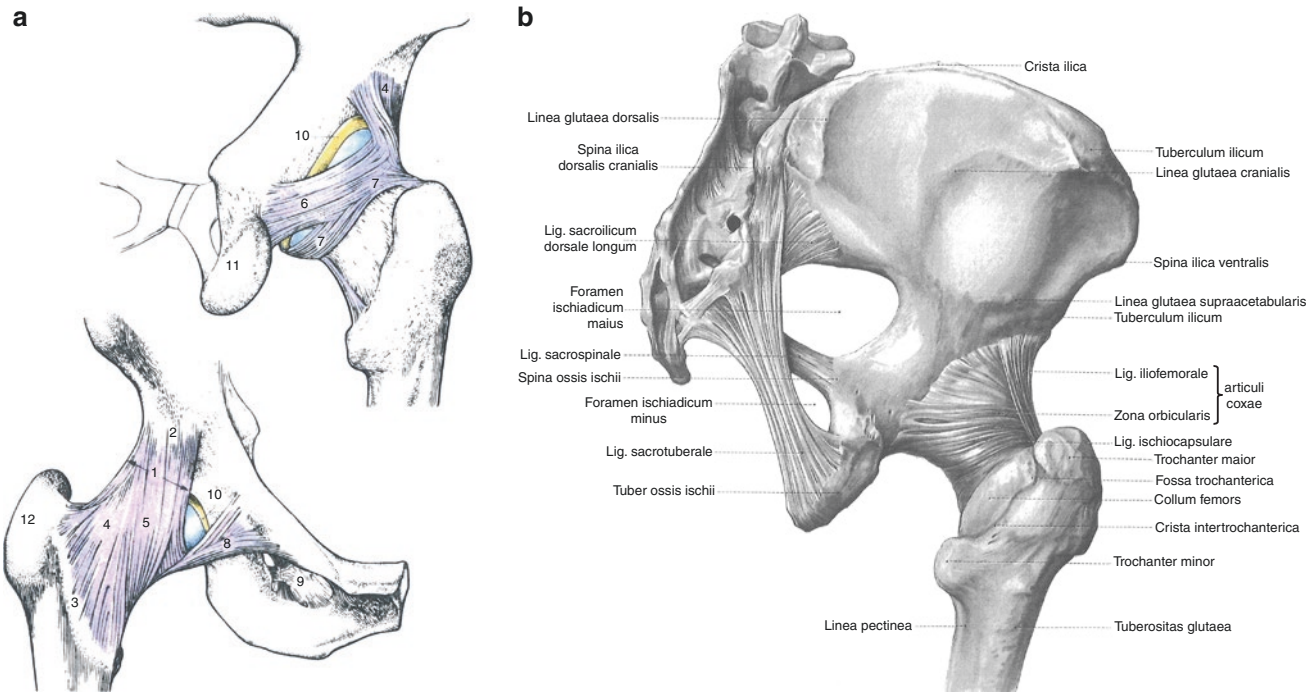


Fig. 1.1 The basic structures of the normal hip in a growing child are outlined in **a**–**j**. (**a**) Anterior view of the hip joint (bottom) demonstrates the iliofemoral ligament (the inverted "Y" ligament of Bigelow). The ligament extends from above the acetabular rim to the intertrochanteric line. The iliofemoral ligament diverges into medial and lateral bands distally (the inverted "Y"). Medially the pubocapsular band is now referred to as the pubofemoral ligament. Posterior view of the hip joint (top) shows the ischiofemoral ligament. The proximal lateral fibers are a continuation from the anterior iliofemoral ligament. The posterior capsule and ligaments insert part way up the neck leaving the distal part uncovered. The lower part of the ischiofemoral ligament is thickened and often referred to as the orbicular band, zone, or ligament. (Figures reprinted from Morris's Human Anatomy (ed. H Morris, J Playfair McMurrich), 4th edition, part 1, Philadelphia, P. Blakiston's Son and Co, 1907). (**b**) Posterior view of the hip joint also showing (above) the superior and posterior part of the iliofemoral ligament as well as the ischiofemoral ligament. The lower margin of the ischiofemoral ligament is almost a discrete structure itself referred to as the orbicular zone or ligament. It is evident on normal hip arthrograms. The synovial protrusion at the lower margin of the orbicular ligament (another arthrographic finding) is shown. The capsule and ligaments of the hip joint insert more distally anteriorly along the intertrochanteric line compared to their posterior insertion that leaves the most distal portion of the neck extracapsular. (Reprinted with permission from Praktische Anatomie by T von Lanz and W Wachsmuth, Springer-Verlag, 1955). (**c**(**i**)) Illustration of the partial pelvis at left from the outer, lateral aspect shows the three component parts of the acetabulum that grow from ileal, ischial, and pubic centers of ossification. The triradiate cartilage is seen linking the three during the growing years. Upper right drawing shows the three bones and triradiate cartilage as they appear on the inner view of the pelvis; lower right drawing shows components in anteroposterior view. (**c**(**ii**)) Growth from components of the triradiate cartilage is shown by directional arrows in the same projections as shown in **c**(**i**) above. The triradiate cartilage lengthens, widens, and deepens the acetabulum with growth. (**c**(**iii**)) Drawing of the acetabular cartilage complex from (a) medial-inner pelvic aspect, (b) posterolateral aspect, and (c) lateral outer aspect. There is cartilage tissue communication between the triradiate cartilage and the hemispheric articular cartilage. (Reprinted with permission from TJ Harrison, *Journal of Anatomy*). (**c**(**iv**)) Drawing shows the functional specificity of the cartilage components of the developing acetabulum and pelvis, all of which appear only as radiolucent regions on plain radiographs. Where two bone regions are adjacent to each other, the triradiate cartilage separating them is physeal, epiphyseal, and physeal from bone to bone. Where a bone region is adjacent to the joint, the cartilage between bone and joint is physeal, epiphyseal, mini-plate, and articular. The full code is listed on the illustration. AC, articular cartilage; EC epiphyseal cartilage; and PC, physeal cartilage. (**d**) At puberty, the depth of the acetabulum is increased by three secondary ossification centers at the periphery of the acetabular cartilage. The os acetabuli (OA) is the epiphysis of the pubis and helps form the anterior wall of the acetabulum. The acetabular epiphysis (AE) is the epiphysis of the ilium and forms a major part of the superior wall of the acetabulum, while a third smaller epiphysis in the ischium is also formed. (Reprinted with permission from Ponseti, *JBJS Am*). (**e**) Three anterolateral views of the pelvis and acetabulum, following removal or displacement of the proximal femur, demonstrate that the acetabulum is spherical, deepened by the acetabular labrum (glenoid in older terminology), and given further support inferiorly and anteriorly by the transverse acetabular ligament across the acetabular (condyloid) notch. Articular cartilage does not cover the entire interior of the acetabular socket, being present in a lunate shape covering primarily the superior, posterior, and lateral aspects of the socket. It is relatively deficient medially where it is replaced or covered by the synovial membrane, the fibro-fatty tissue (pulvinar), and the origin of the ligamentum teres. (Illustrations reprinted with permission from Morris's Human Anatomy 1907). (**f**) Coronal section drawing illustrates the main features of the developing hip. There is lateral extension of the acetabulum by the fibrocartilaginous labrum. The capsule inserts laterally and superiorly above the acetabular labrum and cartilage onto the side of the ilium. This recess is a normal anatomic feature and is outlined in a normal hip arthrogram. A similar attachment of the capsule inferiorly beyond the acetabular labrum occurs. Medially, the floor of the acetabulum is covered by fibro-fatty tissue, the synovial pad, and the origins of the ligamentum teres, leaving the articular cartilage present superiorly, posteriorly, and laterally. The trabecular orientations within the bones outline the direction of bone deposition responding to regions of heightened stress.

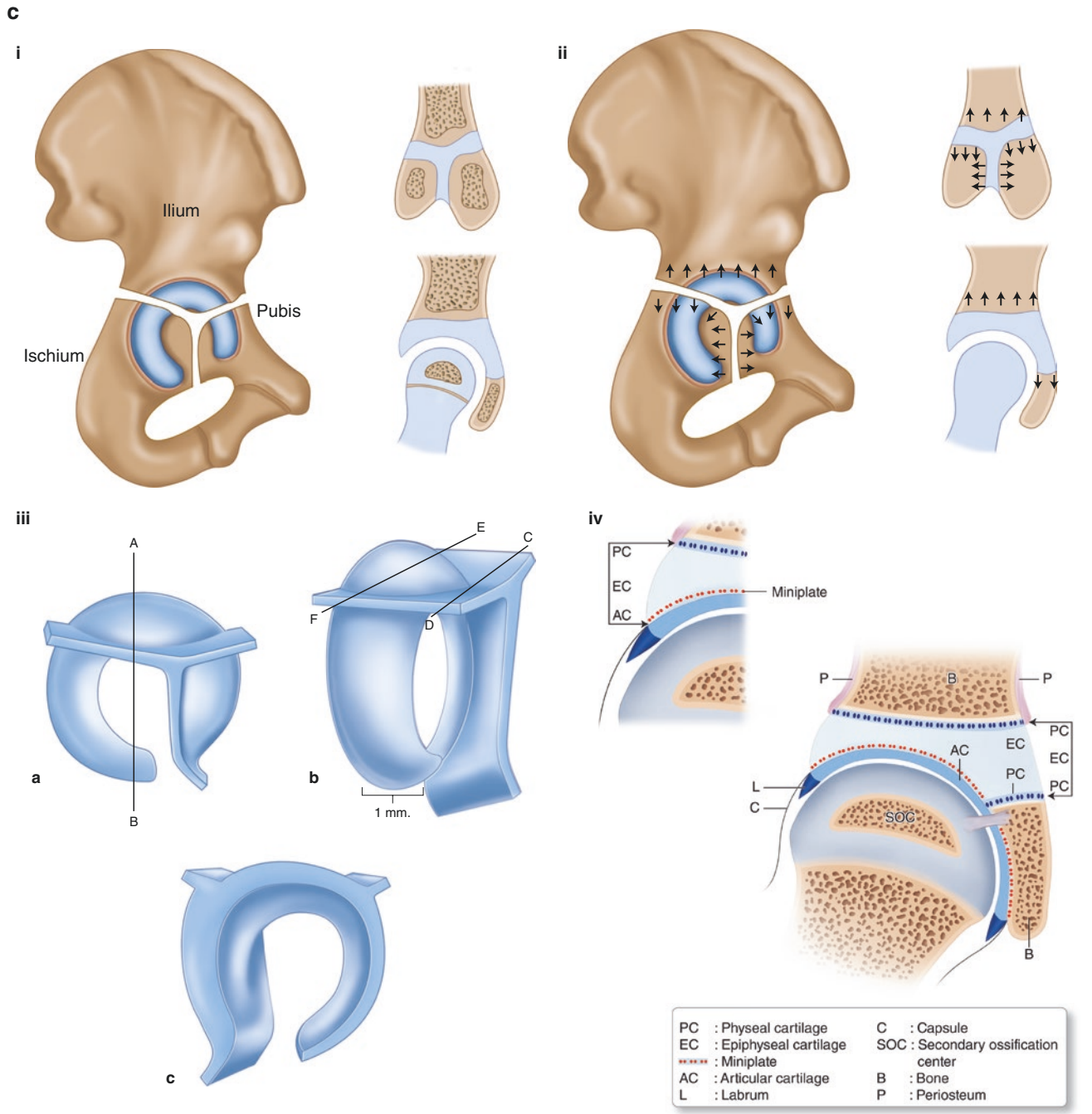
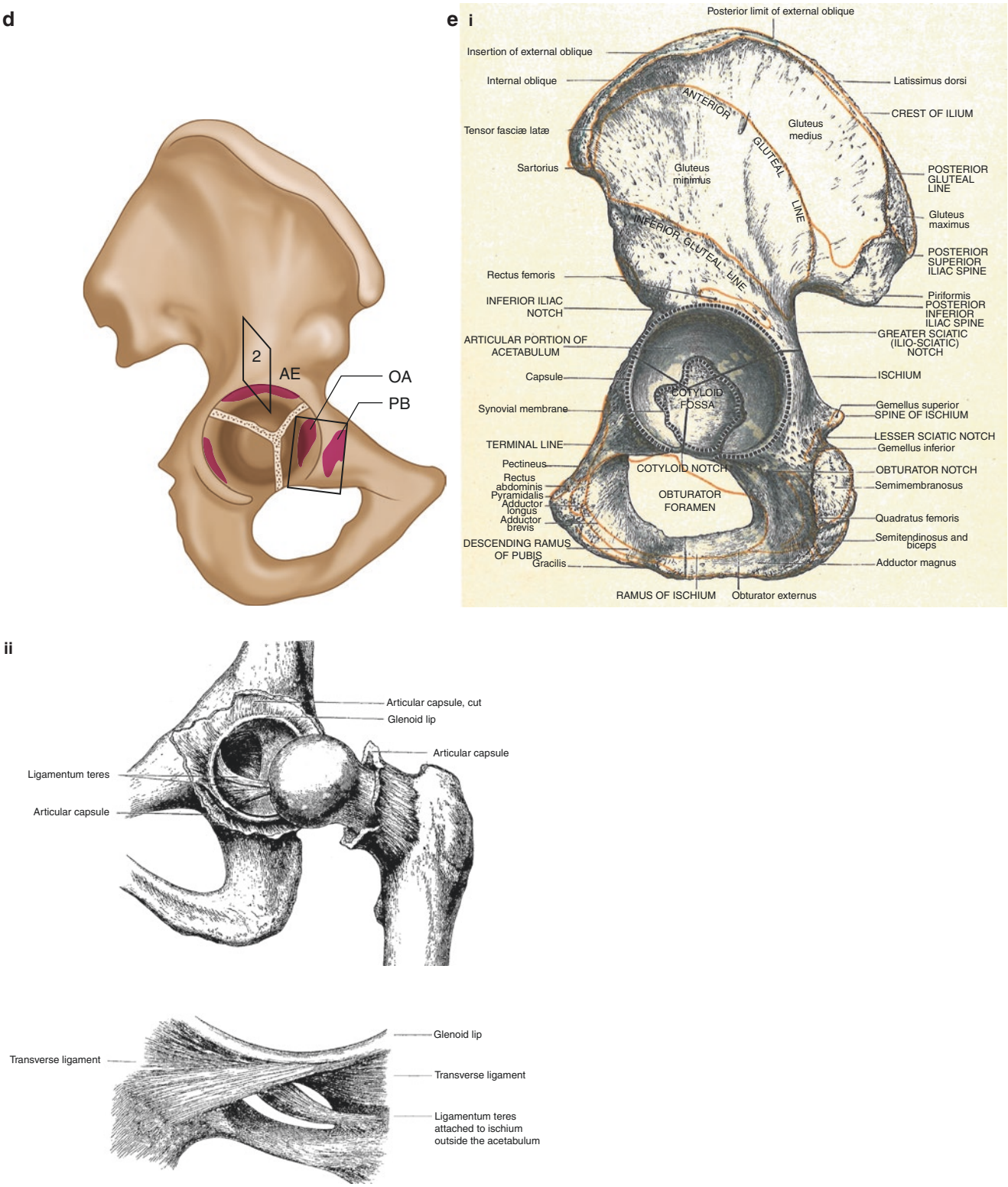


Fig. 1.1 (continued) (Reprinted with permission from Praktische Anatomie by T von Lanz and W Wachsmuth, Springer-Verlag, 1955). (**g**) Anteroposterior pelvic (hip) radiograph from a child at the same age corresponds to the illustration in (**f**). (Reprinted with permission from T von Lanz and W Wachsmuth). (**h**) A series of drawings of proximal femurs shows progressive decrease of the mean head-neck-shaft angles with normal growth from 3 weeks of age (150°) (far left) to 15 years of age and then in adulthood (120°) at far right. (Reprinted with permission from T von Lanz and W Wachsmuth). (**i**) The ranges of anteversion that occur with normal development in the proximal femur are shown. The proximal head and neck of the femur are shaded dark gray, and the distal femur at the condyles (knee) is outlined but clear within. The two femoral segments are drawn as if visualized along the same plane with the proximal part superimposed on the distal. The head-neck axis is the darkest line and the transcondylar axis the lightest. The angle between these lines indicates the extent of anteversion or retroversion of the head/neck in relation to the distal condyles. The middle drawing (c) shows the normal with a mean angle of 12° anteversion. The images above (b) and below (d) are progressing toward the outer ranges of normal, (b) increasing the anteversion to 20°, and (d) decreasing the anteversion to 4°. At top, (a) demonstrates increased anteversion beyond normal to 37°, and, at bottom, (e) demonstrates clear retroversion of −25°. (Reprinted with permission from T von Lanz and W Wachsmuth). (**j**) Diagrammatic representation of the labrum in relation to the articular cartilage of the acetabulum and the lateral edge of acetabular bone. Note the continuous transition zone between articular cartilage and labral fibrocartilage. (Reprinted with permission from Field and Rajakulendran, *JBJS Am*)



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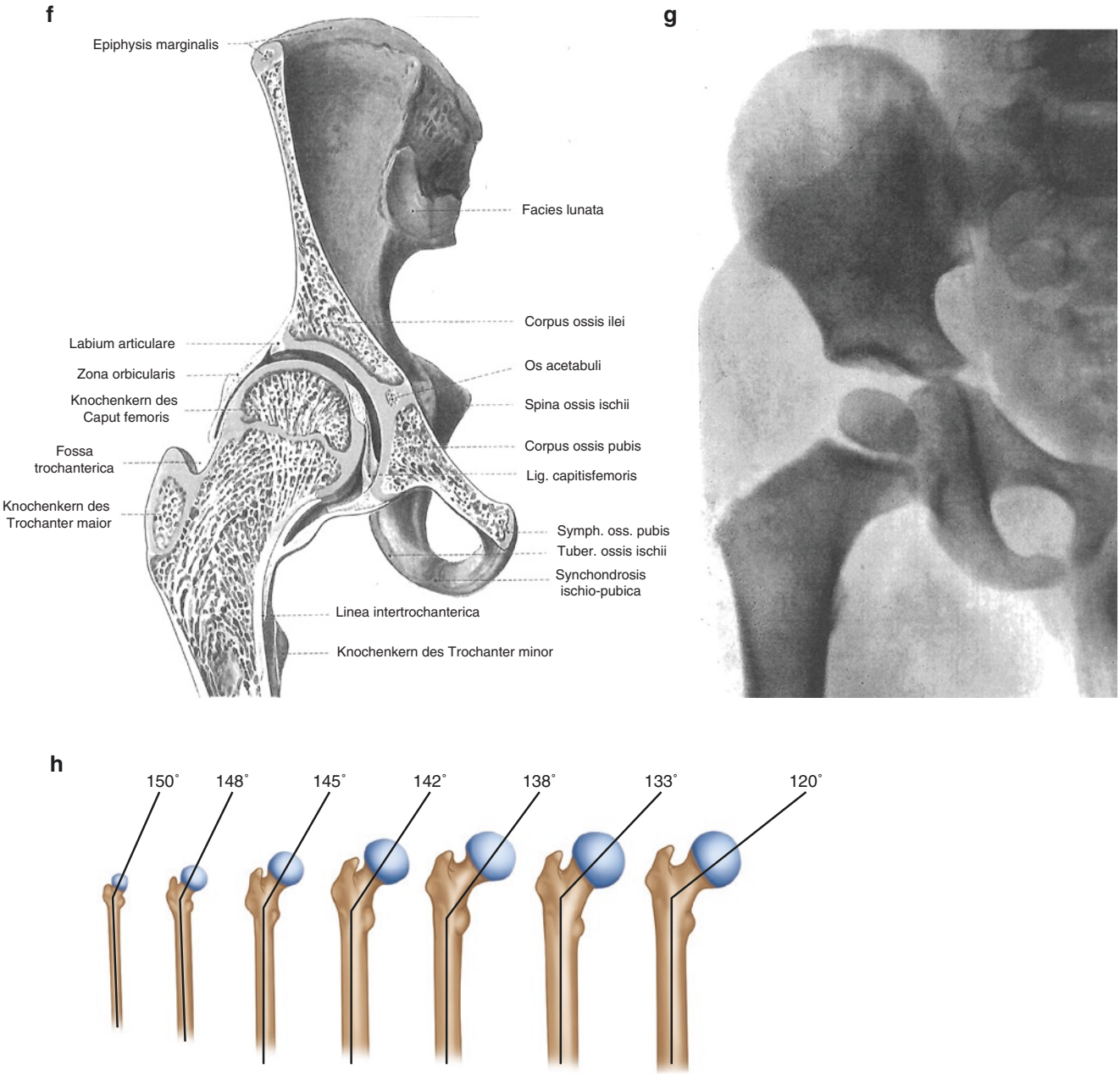

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