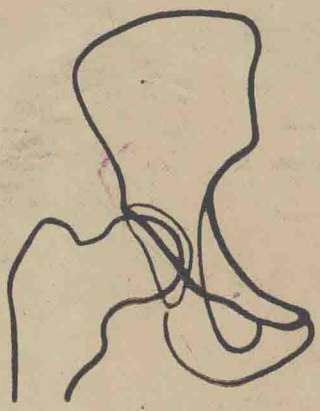
HIP DISORDERS
in Infants and Children



STANLEY M. K. CHUNG

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STANLEY M. K. CHUNG, M.D.

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HIP DISORDERS
in Infants and Children

To my wife Priscilla; my sons Christopher and Anthony; and my mother Gladys Chung.

Preface

This book was written to provide a detailed account of the human hip joint's growth and development and to serve as a practical guide for the diagnosis and treatment of diseases and injuries which affect the hip in the growing child.

Since the literature on some of these diseases is voluminous, no attempt has been made to review all of it. The information presented in this textbook has been gathered from the many individuals and medical centers which have contributed greatly to an understanding of the child's hip, and the author has offered his personal experience and research.

Emphasis will be placed on those disease processes which may eventually lead to the development of adult degenerative hip joint disease. I have excluded the various tumors which seldom affect hip joint function. Poliomyelitis residuals are only uncommonly encountered and are excluded. Since myelomeningocele hip treatment is undergoing significant change, the topic has been omitted.

I hope a clearer understanding of the etiology, early diagnosis, and proper treatment of hip disease in children will contribute to prevention of degenerative joint disease in the patients as adults. However, many more studies are needed to evaluate treatment results and the disease course. Ten to thirty years may pass before the physician can assess the outcome, and the prospective studies with long-term follow-up examination may extend beyond one physician's lifetime. Younger physicians must be encouraged to continue the search for a solution to this problem.

Honolulu, Hawaii STANLEY M. K. CHUNG

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I would like to specify how the following physicians assisted in providing the information in this book. Michael Hardy gave advice on hip anatomy. Joseph Lane, Steven Fischkoff, Joseph Rabson, and Doreen Morrow offered their work on femoral head diameters. Bruce Heppenstall reviewed our patients with chondrolysis at the Children's Seashore House. Juan Cruz Derqui showed me his method for reducing difficult hip dislocations. Karl Chiari demonstrated the Chiari pelvic osteotomy; Dr. Robert Salter demonstrated the innominate osteotomy. Dr. George Lloyd-Roberts explained the use of the Coventry plate, varus osteotomy, and acetabuloplasty. Dr. Yoichi Sugioka assisted me in performing his rotational osteotomy. Dr. Jane Chatten assisted me in obtaining pathologic specimens. Dr. Arthur Hecht provided his mathematical blood flow analysis in Legg-Perthes disease. Drs. Steven Batterman and Toby Hayes offered advice on biomechanics, Frances Gill provided a review of hemophilia, Dr.Kai Lau reviewed the renal osteodystrophy section, Dr. Marie O. Russel reviewed the sickle cell anemia section, Dr. Stanley Plotkin reviewed infectious diseases, Alfred M. Bongiovanni and John Parks brought information on endocrinology, Drs. Phyllis Eveleth and W. M. Krogman covered normal growth and development, Balu Arthryea reviewed the juvenile rheumatoid arthritis and inflammatory bowel disease, and Dr. Ivar Larsen contributed the technique for trochanteric arthroplasty.

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Contents

CHAPTER 1

Normal Hip Development

The developing hip changes at different rates in size, shape, and relationship of parts. This chapter summarizes our laboratory* observations and the present English and foreign literature on human hip development and relevant data from animal experiments. We will describe changes with growth and will give specific measurements, when available, to quantitate changes. Beginning with a discussion of hip differentiation and ossification in the embryo and fetus, this chapter will also discuss the acetabulum, the proximal femur, the geometry of the normal hip, and the vascular supply. Specific information on muscles and nerves, physiology, and biochemistry of the developing hip, for the most part, is not available.

The rapid changes in the embryo, fetus, and newborn compared to the slower changes in older children significantly affect treatment.

This material will give a frame of reference for findings in abnormal hips and help provide a rational basis for treatment. The normal values and terminology are essential for understanding the descriptions in later chapters.

DIFFERENTIATION AND OSSIFICATION

Bone and joint development, which begins in the embryo, is regulated by genes and follows a predictable sequence. Structures differentiated in the embryo grow and mature in the next 28 weeks, in the fetus.

The embryonic age or development may be measured by the crown-rump (C-R) length or by embryonic stage, which is identified by external and internal features. Most investigators use the C-R length method.[2,3,24,25,60,72] The shortcoming of this technique is that embryos of the same C-R length can differ in age and embryos of the same age can differ in length. Staging[61] appears more accurate than C-R length. (Figure 1–1 shows the histologic aspects of hip development during this period.) C-R length is used by most investigators[25,60,72] to determine fetal age.

All elements of the hip differentiate in situ by growth and simple enlargement with minor changes in relationship between structures.[60] Four weeks after ovulation (5 mm C-R), the small lower limb buds begin on the anterior lateral body wall at the lumbar and first sacral segment

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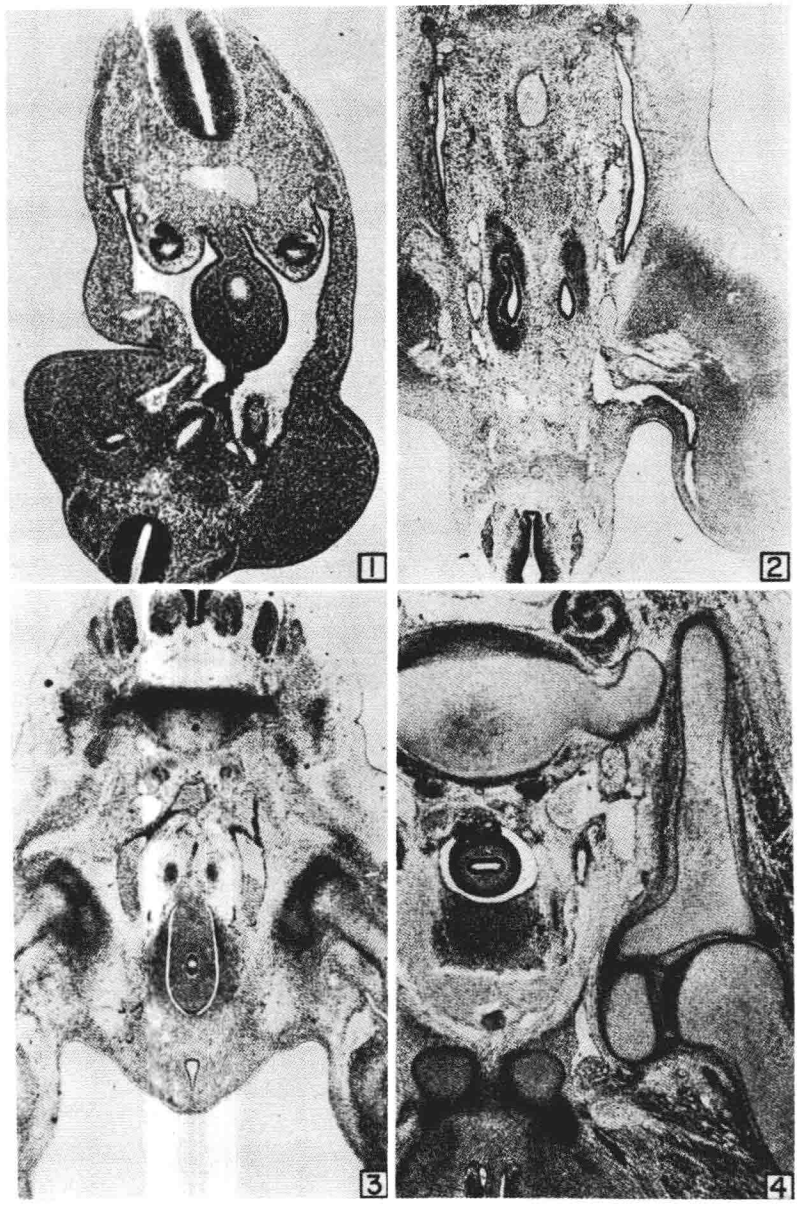

Fig. 1–1. Hip joint histologic features in the embryo. (1) Limb buds in 6.75-mm embryo (×55). (2) Homogeneous skeletal blastema; muscular and osseous elements cannot be distinguished in 10-mm embryo (×30). (3) The globular femoral heads are not congruent with the shallow acetabular depression in 15-mm embryo (×21). (4) The acetabulum, triradiate cartilage region, the acetabular labrum, joint space, and femoral head can be recognized in 30-mm embryo (×30). (From Strayer, L.M.: Embryology of the human hip joint. Clin. Orthop. *74*:221, 1971.)

levels. These buds contain mesenchyme which differentiates to cartilage, bone, synovia, ligaments, muscle, and tendons. Mesenchymal cells form a central mass, the blastema. The central, avascular blastema forms the skeleton.[23] During prenatal life both proximal and distal femur ends are club-shaped. In early stages, the femoral shaft is relatively thick, but later the shaft becomes thinner and longer in relation to the femur ends.[18]

At 6 weeks when the embryo measures 10 mm C-R, the highly cellular blastema condenses to form the cartilage hip model. The primitive chondroblasts then differentiate: their nuclei separate as the cells secrete matrix material into the cytoplasm, and the club-shaped femur forms.[72]

In older embryos (14 to 15 mm C-R), a shallow, saucer-shaped depression appears in the innominate blastema, proximal to the femoral head. This future acetabulum now forms 65 to 70 degrees of an arc.[60] A precartilaginous center occurs in the middle of the femoral shaft at 12 mm.[24]

By 17 mm an interzone is present between the femur and innominate bone anlagen composed of randomly oriented homogeneous cells. At 20 mm the interzone separates into three layers. The two outer layers, continuous with the perichondrium of the femoral and innominate anlagen, are chondrogenous, while the middle layer consists of loosely packed mesenchymal cells.[24]

Both tendons and capsule appear as cellular condensations in the early embryo. Collagen fibers first appear in the iliopsoas tendon at 22 to 25 mm and in the capsule at 28 mm. The ligamentum teres and acetabular labrum appear as increased cell densities at 22 to 25 mm.[24] The primary midshaft femur ossification center appears in embryos 23 to 35 mm C-R[46] while the greater and lesser trochanters appear as slight elevations on the femur.[24]

Hip joint cavitation begins in the late embryonic or early fetal period (C-R 27 to 31 mm).[23] The joint space forms along the femoral head periphery, gradually extending centrally.[72] When the acetabulum separates from the femoral head, it is a deep cavity almost totally enclosing the femoral head. As growth proceeds, the acetabulum depth continues to increase, but the extent to which it encloses the femoral head decreases, reaching a minimum at birth when it represents one-third of a sphere.[52]

The femoral head shape changes during prenatal development. In the embryo the femoral head represents 80% of a sphere, but decreases to 50% at birth. Postnatally the femoral head again becomes more spherical, but not to the same extent as in the embryo. Thus, femoral head coverage decreases during prenatal life from 100% at 12 weeks to a minimum of 65% at birth, after which coverage continues to increase gradually until development ceases.[52]

When studying the effect of neuromuscular blocking agents in chick embryos, the evidence suggests that movement is necessary for joint cavity formation. Decamethonium and crystalline type A botulinum toxin injected into the chorioallantoic circulation inhibits joint cavitation in embryos.[11] The joints fill with vascular tissue and fuse by fibrous or cartilaginous tissue. Intra-articular ligaments fail to develop, and muscle is replaced by fat.[11]

At the end of the embryonic and early fetal period, the primary ossification center appears in the ilium (38 to 39 mm),[46] and the bursa for the obturator internus tendon first appears (30 to 33 mm). However, the time at which the iliopectineal bursa and the trochanteric bursa of the gluteus maximus appears varies and specimens often have no defects in the anterior joint capsule, permitting communication between the hip joint and the iliopectineal bursa.[24]

An early increased cell density (22 to 25

mm) can be identified as acetabular labrum[24] but only clearly forms at 49 to 50 mm.[24,72] Blood vessels enter the acetabular fossa at 30 to 33 mm but femoral head vascularization begins at 40 to 50 mm.[24] Primary ossification centers begin in the ischium at 105 to 124 mm and in the pubis at 161 mm.[46]

ACETABULUM

SURFACE ANATOMY

The acetabulum, a cup-shaped cavity located on the lateral surface of the innominate bone, articulates with the femoral head to form the hip joint. The three components of the acetabulum are: its superior two-fifths composed of ilium, its inferolateral two-fifths of ischium, and its medial one-fifth of pubis. The triradiate (Y) cartilage is located at the acetabular center where these three bones eventually unite (Fig. 1–2).

The central nonarticular acetabular fossa, filled with a haversian fat pad, is separated from the hyaline cartilage articular (lunate) surface by an inner limbus which is raised from the acetabular floor to form the crista articularis (articular ridge). This ridge is highest on the posterior and inferior acetabular surfaces.

The outer articular surface margin is continuous with a tough, fibrocartilage ring (the labrum). There is a notch at the inferolateral acetabular margin where the labrum is attached to the transverse acetabular ligament. The labrum and transverse acetabular ligament form a complete ring around the acetabulum, deepening the cavity.[15] At birth, the labrum resembles that in adults and consists of fibrocartilage.[44]

The smooth slightly concave posteroinferior one-third of the articular surface, called the sustentaculum, supports the inferior femoral head in a sitting or supine position and is reinforced by a small flared ischial bone column. A similar, less defined facet occurs at the anterior articular surface end and is buttressed by the pubic bone where it widens to contribute to the acetabulum.

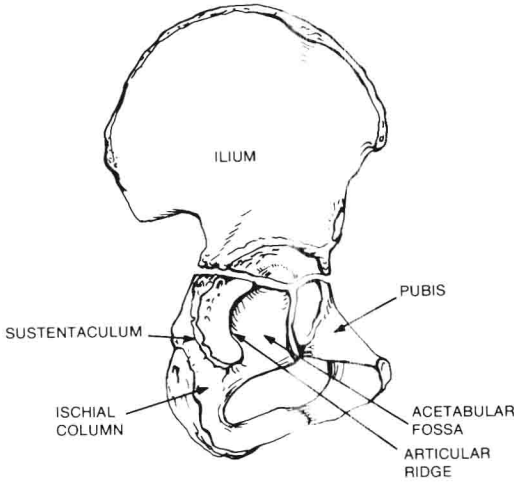

Fig. 1–2. Macerated innominate bone shows acetabular fossa, articular ridge, sustentaculum, and ischial column.

INTERNAL ANATOMY

The acetabular bone is not homogeneous since weight-bearing forces are distributed along different pathways. Two thick bars of bone in the ilium, not noticeable at birth, can be detected at 1 year of age and become pronounced at 3 years.[68] The thicker bar, extending from the auricular ilium surface to the acetabulum, is a three-sided pyramid, its apex at the upper auricular surface and its thick base forming the upper acetabulum. The second pyramidal bone bar extends from the projection on the iliac crest to which the iliotibial band is attached to the upper acetabulum. The bases of these two bars meet in the upper acetabulum.

If the innominate bone is sectioned in a

plane parallel to a line drawn from the upper auricular surface to the acetabulum, two sets of lamellar arches can be seen in the cancellous bone. These arches are inverted with respect to one another and are supported by two thick compact bone buttresses, analogous to the calcar femorale, which reinforces the medial femoral neck. Thus, the strength of these weight-bearing bone bars is not the result of bone thickness alone, but also of its internal structure. This acetabular reinforcement accounts for the relative resistance of the superior acetabulum to fracture.

OSSIFICATION

Although the times at which primary ossification centers appear in the pelvis are established, less is known about acetabulum secondary centers, since few adolescent specimens are available from autopsy and the only centers visualized on anteroposterior radiograms are those located in the triradiate cartilage.

One study finds five centers in the triradiate cartilage and three in the articular acetabular surface.[14] Another author shows four secondary centers in the acetabulum at 10 to 13 years with fusion at 13 to 16 years.[33]

In addition to these secondary acetabular centers, a "radiologic os acetabuli" or "os ad acetabulum" has been described. This elongated or round bone structure, the size of a brown bean, appears at the lateral acetabular margin during adolescence and, in most cases, fuses a few months later.[1,74] The bone separated from the ilium by a narrow space has a well-defined outline but no cortex, and its shadow follows the acetabular rim contour exactly. This normal variant must be distinguished from periarticular calcifications, which have no visible bony structure and may give rise to severe pain, swelling, and hip motion loss.[74]

THE PROXIMAL FEMUR

SURFACE ANATOMY

The proximal femur consists of the femoral head and neck and the greater and lesser trochanters. The femoral head forms about two-thirds of a sphere and joins the neck at the subcapital sulcus. This groove (deepest at the lateral and medial head and neck junctions) contains the intra-articular subsynovial vascular ring. In three of our 17 Latex-perfused specimens the anterior subcapital sulcus was replaced by an articular surface extension of the head to the anterior neck.[6] In dried-adult or macerated adolescent proximal femur specimens, foramina are clearly seen where blood vessels pierce the femoral neck, most often laterally, next most frequently medially. This agrees with the number of arteries penetrating the four proximal femur surfaces.[6]

Neither the femoral head nor the horseshoe-shaped articular acetabular surface is completely spherical. These two hip joint elements come into intimate contact only in the maximum weight-bearing position when the iliofemoral ligament (Y-ligament of Bigelow) is fully stretched by complete hip extension and internal rotation. In all other positions the joint surfaces are unevenly opposed.[22,71] Fresh preparation photographs of normal adult hips reveal that the opposing articular margins coincide with circle circumferences having different radii.[22] Similar relationships probably exist in children but no studies are available to confirm this supposition.

A small depression called the fovea capitis on the medial femoral head surface

is the attachment site of the round ligament (ligamentum teres) which originates from the acetabular notch and the transverse acetabular ligament.

Blood vessels divide in the trochanteric fossa (a space just medial to the greater trochanter) before they either pass through the capsule to the femoral head and neck or proceed laterally to supply the greater trochanter.

The intertrochanteric line, a slightly raised bone ridge, extends from the anterior greater trochanter to the lesser trochanter and is the attachment site of the iliofemoral ligament (Y-ligament of Bigelow). The trochanteric crest occupies a similar position posteriorly between the trochanters and is the distal attachment site for the short external rotators. The piriformis, obturator internus, gemelli, and quadratus femoris attach at the superior crest. The gluteus medius and minimus attach to the superior and lateral greater trochanter, while the psoas attaches to the lesser trochanter.

The femoral neck is covered by synovium, which extends from the subcapital sulcus to the femoral neck base. The synovium extends from the neck base, is reflected upward to the inner capsule surface, extends to the acetabular labrum, and is attached to the acetabular margins. Within the acetabulum, the synovium passes over the transverse acetabular ligament, surrounds the ligamentum teres, and attaches to the haversian fat pad. Prominent synovial folds and fat cover the many intra-articular ring blood vessels on the medial and lateral subcapital sulcus.

The retinacula of Weitbrecht,[70] capsular reflections covered with synovium, can be seen on the medial femoral neck. These structures pass from the peripheral capsule attachment at the femoral neck base to the medial subcapital sulcus and carry blood vessels to the medial femoral neck and head but are not a constant feature.[6]

INTERNAL ANATOMY

The internal architecture of the developing proximal femur includes: (1) the articular cartilage, (2) the secondary ossification centers in the femoral head, and greater and lesser trochanters, (3) the growth plates, (4) the reciprocally interlocking mamillary processes, (5) the perichondrial fibrocartilaginous complex, (6) Ward's and Babcock's triangles, and (7) the calcar femorale (Figs. 1–3, 1–4, 1–5).

In the fetus and newborn, the entire femoral head consists of cartilage. Four to seven months after birth the secondary ossification appears, and bone progressively replaces cartilage. At birth, most of the growth plate is located outside the capsule. During the first months of life the femoral head and greater trochanter growth plates, together with the surrounding perichondrial complex, are continuous. Eventually the two growth plates separate, one directed toward the femoral head and the other toward the greater trochanter, and separate secondary centers later form. The capital growth plate and its proximal metaphysis gradually advance up the femoral neck, and the secondary center fuses with the metaphysis in adolescence (Table 1–1).

From birth to age one year the junction between the growth plate and the metaphysis is relatively smooth, but between ages 14 months and 5 years interlocking bone and cartilage pegs, the mamillary processes, appear, giving the interface a progressively corrugated appearance. These pegs attain their maximum height between age 6 and 13 years and help resist the shear of weight-bearing in the adolescent.[7]

Tissue encircling the growth plate in the long bones of cattle, dog, sheep, rabbit, and human embryos was first described by Ranvier.[53] On longitudinal sections, a circular groove was found in the cartilage at both ossification front margins, encircling the growth plate. Immediately