

The Classic

Etiology of Congenital Dislocation of the Hip*

Carl E. Badgley MD (1893–1973)
The 11th President of the AAOS 1942

Dr. Carl E. Badgley was born in 1893, the son of a Presbyterian minister [2]. He received his medical degree at the University of Michigan in 1919, and became interested in orthopaedic surgery owing to Drs. Hugh Cabot and LeRoy Abbott. He was appointed as an instructor of surgery in 1920 and was appointed professor and head of the Section of Orthopaedic Surgery in 1932, an appointment he retained until 1963 when he retired.

Dr. Badgley, devoted to his home state, was active in organizing institutions and organizations within Michigan. These included the Rackham Arthritis Research Unit within the hospital devoted exclusively to arthritis research and the Michigan Crippled Children Commission. He was active in the Board of Control of Intercollegiate Athletics. As President of the AAOS in 1942, he faced challenges organizing the 1943 meeting owing to the war years and many parts of the social program, particularly for the spouses, were eliminated [3]. (Travel was limited in part due to rationing of gas and a reduction in some public transportation since the war effort had priority on petroleum products.) Of the 235 members and 461 guests attending the 11th Annual Meeting in 1943, 203 of the men were in the military service. Nonetheless, during his year of Presidency of the AAOS, Instructional Course Lectures (13 courses) were introduced at the 1942 annual meeting (at a cost of \$1.00 per course) and were an immediate success [3]. They were first published the following year (1943) by J.W. Edwards Co., of Ann Arbor, Michigan (who continued to publish the ICL through 1958), under the editorship of a future AAOS President, Dr. Tommy Thomson.

The article we reproduce here details the two major theories of congenital dislocation of the hip: “a primary germinal fault...(and)...a defect of development of environmental



Carl E. Badgley, MD is shown. Photograph is reproduced with permission and ©American Academy of Orthopaedic Surgeons. *Fifty Years of Progress*, 1983.

origin” [1]. As a true scientist, he commented, “The most commonly accepted theory of developmental abnormality is a primary failure of proper formation of the acetabulum, particularly a germinal failure of development of the posterior superior buttress of the ilium...It is difficult to see how an observer, unless influenced by the weight of pre-existing statements and concepts, can authoritatively state a hypothesis as an accepted fact. The author denies dogmatically, for example, that there is scientific evidence of a primary genetic developmental fault of the posterior superior portion of the acetabulum. He does not refute the existence of such a lesion, but contends that no satisfactory evidence has been submitted that this lesion is the primary developmental fault.” How often do we make our judgments based on the “weight of preexisting statements,” rather than compelling observations and data? Also as a true scientist, his thorough review leads to and ends with a hypothesis: “Congenital dislocation and

congenital dysplasia of the hip may be regarded as the result of faulty development, due to environmental factors extrinsic to the hip joint. An inherited fault in the timing of development may produce these extrinsic changes... Heredity can play an important part in altering the growth and time factors." Despite astonishing technical advances, we have the same working hypothesis today and DDH may indeed be related to the timing of genetically controlled events in conjunction with external factors; the details of the genetic factors are being explored with tools not available to Dr. Badgley, but we seem no closer to the larger answer.

Richard A. Brand MD

References

1. Badgley CE. Etiology of congenital dislocation of the hip. *J Bone Joint Surg Am.* 1949;31:341–356.

2. Carl E. Badgley, M.D. 1893–1973. *J Bone Joint Surg Am.* 1973;55:1112–1113.
3. Heck CV. *Fifty Years of Progress: In Recognition of the 50th Anniversary of the American Academy of Orthopaedic Surgeons.* Chicago, IL: American Academy of Orthopaedic Surgeons; 1983.

© The Association of Bone and Joint Surgeons 2008

(The Classic Article is ©1949 by the Journal of Bone and Joint Surgery, Inc. and is reprinted with permission from Badgley CE. Etiology of congenital dislocation of the hip. *J Bone Joint Surg Am.* 1949;31:341–356.)

*Read at the Annual Meeting of The American Orthopaedic Association, Hot Springs, Virginia, June 30, 1947.

Richard A. Brand MD (✉)

Clinical Orthopaedics and Related Research, 1600 Spruce Street, Philadelphia, PA 19103, USA
e-mail: dick.brand@clinorthop.org

Hey Groves has well expressed the present-day concept of congenital dislocation of the hip as "a deformity which is mysterious in its origin, insidious in its course and relentless in its final crippling results". Numerous well-known theories for the development of this condition have been advanced. Extensive anatomical studies of normal foetal skeletons, and investigations of the pathological anatomy of foetal and postnatal material demonstrating true dislocations of the hip, have been reported. Genetic studies of the problem have indicated familial tendencies, with even a strong suggestion of a dominant Mendelian trait. Anthropological studies and knowledge of comparative anatomy have been utilized.

In general, two main theories for the etiology of congenital dislocation of the hip have predominated for many years. Throughout the literature, the main contentions for the various theories are based upon two assumptions,—one that the lesion is the result of a primary germinal fault; the other, that it is due to a defect of development of environmental origin. Recent experimental embryological work tends to show that many lesions, considered the result of a germinal defect, can be produced by environmental changes.

Although exponents of the mechanical theory can present convincing anatomical evidence to support their claim, few authorities accept this theory. The cumulative evidence of sex characteristics, hereditary factors, and geographical and racial incidence, the increasing recognition of an associated hip dysplasia on the so-called normal side, and the frequent occurrence of associated deformities have led to the more popular hypothesis of a primary developmental fault.

The most commonly accepted theory of developmental abnormality is a primary failure of proper formation of the

acetabulum, particularly a germinal failure of development of the posterior superior buttress of the ilium. Murk Jansen, Bruce, Morrison, Hey Groves, Fairbank, and many others, including the present-day followers of the concept of primary acetabular dysplasia as emphasized by Faber and Hart, have believed that the flat socket is the primary developmental fault.

It is difficult to see how an observer, unless influenced by the weight of pre-existing statements and concepts, can authoritatively state a hypothesis as an accepted fact. The author denies dogmatically, for example, that there is scientific evidence of a primary genetic developmental fault of the posterior superior portion of the acetabulum. He does not refute the existence of such a lesion, but contends that no satisfactory evidence has been submitted that this lesion is the primary developmental fault. Similarly, several other authors have stated that, in the mechanism of development of congenital dislocation of the hip, although the acetabulum is defective, the head is within the socket with the hip in the typical intra-uterine position of flexion. On extension of the lower extremity after birth, dislocation may occur suddenly, or gradual displacement may result. With what proof is this statement of the time occurrence for congenital dislocation of the hip made?

There is on record, it is true, evidence of congenital dislocation in a five-month foetus. The teratological dislocation is undoubtedly prenatal in origin, and another type, to be discussed, also occurred prenatally. Rather should one say that the time of occurrence of congenital dislocation of the hip is not known, because of inadequate investigation in early life. Certainly Putti, stressing the features of early diagnosis by his triad of criteria, has demonstrated the possibility of recognizing preluxation of

the hip at a much earlier age, by a process of education of the public and the doctor to look early for this abnormality.

It is time for us to investigate the etiology of dislocation of the hip, free from prejudice, assembling the facts without bias. Conjecture should not be presented as fact.

Mechanical Factors

Mechanical factors producing dislocation of the hip have been recognized in the postnatal period. The position of flexion and adduction has been demonstrated in a number of clinics, including our own, as a dangerous position in hip-joint infection, leading to pathological dislocation. Certain isolated cases of congenital dislocation of the hip have been reported, which have a similar mechanical malposition, known to be a potential cause of dislocation of the hip in postnatal life. Tubby stated that Tridon had reported 121 cases of congenital dislocation of the knee, in twenty of which congenital dislocation of the hip occurred due to intra-uterine mechanical causes.

Through the courtesy of the Ford Hospital, the author has had the opportunity to see such a case of congenital dislocation of the knee, associated with abduction of the hip on the same side (Fig. 1). The opposite hip was adducted and dislocated. The foetal movements were said to be unusual, being felt in only one place above the left iliac crest. Roentgenograms of the mother, taken a week before delivery, clearly demonstrated the fixed deformity of the limbs *in utero*. Such an isolated case is, however, not

a substantiation of the mechanical theory for dislocation of the hip except in unusual circumstances.

The mechanical concept of Le Damany and Dega seems plausible, but does not explain adequately the environmental, genetic, and racial features.

There are many factors opposed to the concept of a primary developmental defect of the acetabulum as the cause for dislocation of the hip. Numerous unsubstantiated explanations for this fault have been offered, such as early arrest of the Y cartilage and, more recently, a primary developmental failure of the posterior superior buttress of the acetabulum.

An intensive review of the etiology, in light of recent methods of embryological experimentation, is necessary. The information available at the present time is inadequate, but may show the possibilities for future study.

Genetic Study

The approach to the problem of inheritance factors in congenital dislocation of the hip is very difficult. Patten has stated that the basic problem of the interplay of heredity and environment, although "both are involved in the causation of anomalous development", makes generalizations unsure as to their relative importance. Warkany and his associates found that feeding defective diets to normal breeding rats produced offspring with skeletal defects. Such findings, which hitherto had always been regarded as the result of a primary intrinsic fault, show the importance

Fig. 1 Child with abduction and flexion of right lower extremity and hyperextension of the tibia on the femur, associated with dislocation of the left hip, probably the result of the dangerous position of adduction in production of pathological dislocation. A roentgenogram of the mother's pelvis, one week before delivery, showed this same deformity *in utero*.



of extrinsic or environmental factors that may simulate a hereditary intrinsic fault. Patten stated that Stockard had produced experimental evidence "that different disturbances applied at the same phase of development would tend to produce the same defects, whereas the same disturbing factor applied at different phases of development produced different defects".

Hart, in an excellent paper on primary genetic dysplasia of the hip, quoted from Faber's work and his own to show that there are latent carriers of the gene for hip dysplasia. A true genetic history cannot be gained from a study limited to hip dislocation. Hart stated that hip dysplasia is due to a dominant gene. An incidence of about 20 per cent. of genetic occurrence in families has been reported by many authors. According to Hart, Faber demonstrated that dysplasia of the hip joint was three times as frequent as was classical dislocation. Therefore, a search for dysplasia of the hip shows a much higher incidence, as indicated by di Prampero's studies.

Patten has pointed out that enough data for genetic studies in man have not yet been accumulated to be definitely informative, that specific mating is not feasible, and that the heterozygous human germ plasma makes definite conclusions as to the importance of intrinsic and extrinsic factors most difficult.

Principles of Development

We are indebted to Weiss for the following observations: Experimental embryology has added definitely to the knowledge of embryonic development. It seems to be established that there is, in general, almost a predestined mosaic pattern for development which, if carried out in a normal environment, will result in a normal development peculiar for the species. Each species, as well as each system of the species, has its own peculiar growth pattern. This growth pattern, inherently designed by intrinsic factors, is dependent, however, upon the environmental conditions, the *extrinsic factors*, under which it proceeds. Temperature, nutrition, and other environmental factors may accelerate or delay the normal growth rate. The predisposition for normal growth is the growth potential which may be interfered with by factors indispensable for its realization; these so-called "growth circumstantial", however, are not responsible for the characteristics of growth. The growth rate may be normal or abnormal. Embryonic growth of a part advances unevenly. Each system has its own time period, influenced and controlled to some extent by the body as a whole. Interference with growth of a part will produce far more abnormality during a phase of rapid growth than during a quiet phase. It has been shown that abnormalities in development do not

depend upon the nature of the exciting factor so much as upon the time period of the disturbance of normal growth.

Perfect timing for the development of the constituent parts is essential. This is well demonstrated elsewhere embryologically in the failure of proper timing in the development of the heart valves, producing congenital heart lesions, and similarly in the time orderliness of the development of the eye. Patten has discussed the development of rachischisis of the spine from faulty timing in growth.

Parts of a joint can develop typically, however, even though they are not in continuity. Experiments in limb-bud transplantation have demonstrated that a typical socket is developed by the shoulder girdle when no humerus is present; and, similarly, that a typical head of the humerus has developed in the absence of a shoulder socket. Nevertheless, the continued embryonic growth of this intrinsically designed head or socket would alter without the environmental factors present to guide its intrinsic design. It is important to remember the fourth dimension, time, in this dynamic growth,—not what has happened, but what will occur as a result of environmental change.

Murray made the following statement: "Summarising, it may be said that the gross form of those kinds of elements which have been mentioned (mainly parts of the limb skeleton) is developed by self-differentiation, that is, under the direction of factors intrinsic in each developing element. These factors are not, however, sufficient for the production of a functional skeleton. In the early stages, when the development of gross form is proceeding, it is doubtless essential that extrinsic forces such as the growth pressure of other elements, etc. shall not deviate far from the normal conditions; it would be absurd to suggest that the intrinsic factors could produce a normal skeleton however unfavourable the extrinsic factors might be. In early stages the intrinsic factors are determinative, the extrinsic factors only important in providing conditions in which the intrinsic factors can act. In later stages, when the gross skeletal model is being refined and perfected, the importance of extrinsic factors increases. It is doubtless the correlation inevitably following upon development and early function in close contact that causes the two components of a joint, to be so perfectly adapted to one another, and evidence has been presented which indicates that various grooves, prominences, etc., of the late embryonic skeleton are probably produced in reaction to extrinsic and presumably mechanical factors."

Hamburger and Waugh studied nerveless or poorly innervated limb buds and concluded that innervation played a minor role in the development of joint formation. Normally developed skeletal design often occurred in these transplants, in spite of isolation from the body and lack of innervation. Continued development of these abnormally

placed and poorly innervated limbs is not recorded, but doubtless would be most inadequate. Hamburger and Waugh concluded that although “the primary development of the limb skeleton is thus shown to be self-differentiation to a high degree, extrinsic factors become of increasing importance in later phases of bone development”. These studies all demonstrate the mosaic pattern for development; the growth potentiality is initially of great importance, but extrinsic factors may later alter the intrinsic design. Hamburger and Waugh quoted Harrison as warning against “the indiscriminate acceptance of the concept that differentiation proceeds universally from an undetermined state to one of rigid mosaic development”.

It is important to recognize that the mosaic pattern may be interrupted or altered. The prospective potency—that is, the full range of developmental performance of which a given part of the germ is capable under any conceivable circumstances—equals its prospective fate, the lineage of each part of the egg through its cell descendants into a definite portion of the adult. One must have the concept of timing, the dynamic concept of a changing structure. The tempo of operation is an important characteristic which each cell has inherited from the egg. Perhaps here the chief influence of heredity, environment, and geographic and racial characteristics is felt. An inherited alteration in the proper timing of growth of the hip system, or environmental delay or overstimulation, might well result in faulty development. To recognize the potentialities of normal or abnormal growth, the concept of the necessity for perfect timing during the various phases of growth of the hip joint must be understood.

Rotation of the Limb Bud

The evolution of posture of the pelvic limbs is characteristic for the human species. This alteration of position is a well-recognized embryological fact of great importance clinically and anatomically. It has been shown by a number of observers, particularly Bardeen and Lewis, that the limb buds in the human embryo undergo a rotation phenomenon. Bailey and Miller stated that the lower limb bud appears at the end of the third week as a small rounded protuberance on the lateral body wall, opposite the sacral flexure. During the fourth week, elongation occurs and the transverse constriction, separating the proximal from the distal portion, develops. During the sixth week, the limb bud is marked off by a bend for the knee.

As the limb buds first elongate, their long axes lie nearly parallel with the long axis of the body. Later they are directed ventrally, nearly at right angles to the body axis. The tibial margins are turned toward the head. To acquire positions relative to the body as found in postnatal life, the extremities must undergo further changes. These consist of

torsion around their bony axes and rotation through an angle of 90 degrees. The right, lower extremity twists to the left. At the same time both extremities swing backward through an angle of 90 degrees, so that they lie parallel with the long axis of the body.

It is clear from the illustrations of selected embryos (from 8 millimeters to 58 millimeters) that the alteration of position of the limb buds starts prior to the separation of the component parts of the hip joint. Even the 15-millimeter embryo shows lessened abduction of the limb bud. Doubtless this alteration is influenced by growth of the embryo in length. It seems obvious that this postural change of the limb bud prior to motion in the hip joint may be a definite factor in the production of the inclination of the neck of the femur, characteristic of the human species. Most of the postural change of the limb buds develops, however, after the separation of the head of the femur from the acetabulum, which appears usually after the 30-millimeter stage of the foetus.

Thus the hip joint is peculiar in its development. Originating in a lateral abducted position to the pelvis and acetabular anlage, the head of the femur and the shaft must adduct to a position parallel with the long axis of the trunk, practically 90 degrees, and must rotate internally approximately 90 degrees at the hip joint to allow the patella and leg to face forward. Much of this rotation occurs during the third month, but is not complete even after birth. Some degree of retention of the foetal position of external rotation is commonly seen months after birth, even continuing at times until adolescence.

Not only are adaptive changes in the acetabulum and the upper end of the femur necessitated by this rotation phenomenon, but there is development of the oblique position of the acetabulum to the sagittal plane. An increase in growth of the posterior portion of the pelvis in proportion to the extent of growth of the pubic portion is a well-recognized anatomical fact. Thus the position of the acetabulum is developed with a forward and a downward inclination. Steindler gives a statistical report of 40 degrees of forward inclination and 60 degrees of downward inclination. In a recent study from our clinic by Donovan and Campbell on the adult pelvis, the angle of forward inclination was 30 degrees and the angle of downward inclination was 60 degrees.

Dega, in a review of 100 foetal skeletons, showed the angle of forward inclination of the acetabulum to be 29.5 degrees. The downward inclination in relation to the transverse plane was 62.8 degrees. Dega showed that the downward inclination increased gradually, paralleling the increase in anteversion of the neck of the femur. The decrease in the forward inclination of the pelvis, however, did not begin until the eighth or ninth foetal month.

Le Damany pointed out that the human foetus has undergone three deformities at the time of birth which are not found in animals: First, he referred to the pelvis deformed by tilting of the iliac bone on the sacrum with enlargement of the pelvisacral angle; second, to obliquity of the acetabulum; and, third, to anteversion of the head and neck of the femur, associated with torsion of the femur. He argued that, if the sum of the obliquity of the acetabulum and the anteversion of the neck is greater than 60 degrees, dislocation occurs.

The author [1,2] pointed out that the characteristic posture of the limbs in arthrogryposis multiplex congenita closely approximates the early foetal position of the limb buds. We attribute this to failure of rotation of the limb buds, due to the absence of muscle function in the extremities, which is characteristic of the syndrome. This emphasizes the importance of the foetal muscles in the production of torsion and anteversion, as, with extensive muscle involvement, neither torsion nor anteversion is evident if the limbs are in the characteristic posture. In the instances of this syndrome associated with dislocation of the hip, the response to abnormal position of the constituent parts of the hip joint is beautifully illustrated by the case of R.L.M. [2]. Operative inspection demonstrated that the head, having laid above the acetabulum anteriorly, had produced a marked notch in the ilium, which was undoubtedly the result of diminished growth from pressure. The ilium above the displaced head of the femur grew forward normally. The head was then pointing directly into the pelvis and into the iliacus.

Lack of rotation of the limb bud, the retention of the foetal position of the limb, and the position of abduction in these cases may have levered the head forward and left it displaced in a subspinous position. The malposition of the head produced a failure of development of the cartilaginous anlage for that portion of the anterior margin of the ilium pressed upon by the head of the femur. This demonstrates environmental restraint of growth. McCarroll and Crego have reported a similar defect in the anterior margin of the ilium in their cases of anterior dislocation of the hip.

This is an adaptive environmental change, not a primary developmental fault. It is, however, analogous to the maldevelopment of the posterior superior surface of the acetabulum, regarded by many as the resultant of a primary developmental fault.

Dega, in his careful study of the skeletal anatomy of 200 foetal hips, noted marked similarity in the measurements of the head and acetabulum. This, he felt, demonstrated the reciprocal relationship between the two structures. He pointed out, however, that perfect adaptation of the component parts was present only in one position,—the intrauterine position of flexion. He was of the opinion that dislocation of the hip was definitely a consequence of a had

adaptation of the human species to an upright posture. Regardless of this opinion, the anatomical fact remains that the shape of the socket and the head were congruous in their development when normal, denoting that, in the absence of an environmental fault, such a development is the mosaic pattern for the hip joint. Perfect formation of the joint for this reciprocal development requires perfect adaptation and timing for the altered positions of the components of the hip joint during rotation of the limb bud.

Embryological Sections

Through the courtesy of the Department of Anatomy, we have selected a few transverse sections of the developing hip joints of embryos, varying from 8 to 58 millimeters. These sections illustrate the findings which were so well presented by Luther Strayer. The author has utilized Strayer's work freely in interpreting these sections.

In a transverse section through the lower limb buds in an 8.5-millimeter embryo (Fig. 2), the neural tube was at the top. The limb bud was in the finlike position, protruding laterally, and consisted of a mass of undifferentiated mesenchyme. From this period on, the limb bud develops as an entity, only the nerves and vessels growing in from the trunk. There is no definite evidence of the trunk sending other constituents to the limb bud.

As the embryo elongates to 14.8 millimeters, the limb bud develops further, descending ventrally and less laterally (Fig. 3-A). The constriction and bend for the knee become apparent. The mesenchyme shapes itself into the outlines of the blastemal innominate, and the dumbbell-shaped femur is outlined (Fig. 3-B). A hint as to the future site of the hip joint may be observed at the dense accumulation of mesenchyme. The abducted position of the thigh and the external rotation of the limb bud may be noted at this stage. Nerve tissue is present in the popliteal space.

At 25 millimeters, there is further differentiation of the hip joints. The innominate structures are clearly outlined; there is a clearly differentiated acetabular development and a rounded femoral head. Undifferentiated mesenchymal tissue is still present between the outlines of the head and acetabulum, connecting the two structures, which are not yet separated. The capsular structures are forming and there is early evidence of the development of the glenoidal labrum. Further rotation of the limb bud is obvious.

A section at 28 millimeters shows more clearly the cartilaginous nature of the innominate bone and the femur, and the retention of numerous cells about the head of the femur (Fig. 4). Mesenchymal tissue, still undifferentiated, fills the space between the acetabulum and the head of the femur. An early appearance of the glenoidal labrum is suggested in the increased density of the mesenchymal

Fig. 2 Transverse section through lower limb buds in an 8.5-millimeter embryo shows neural tube at the top.

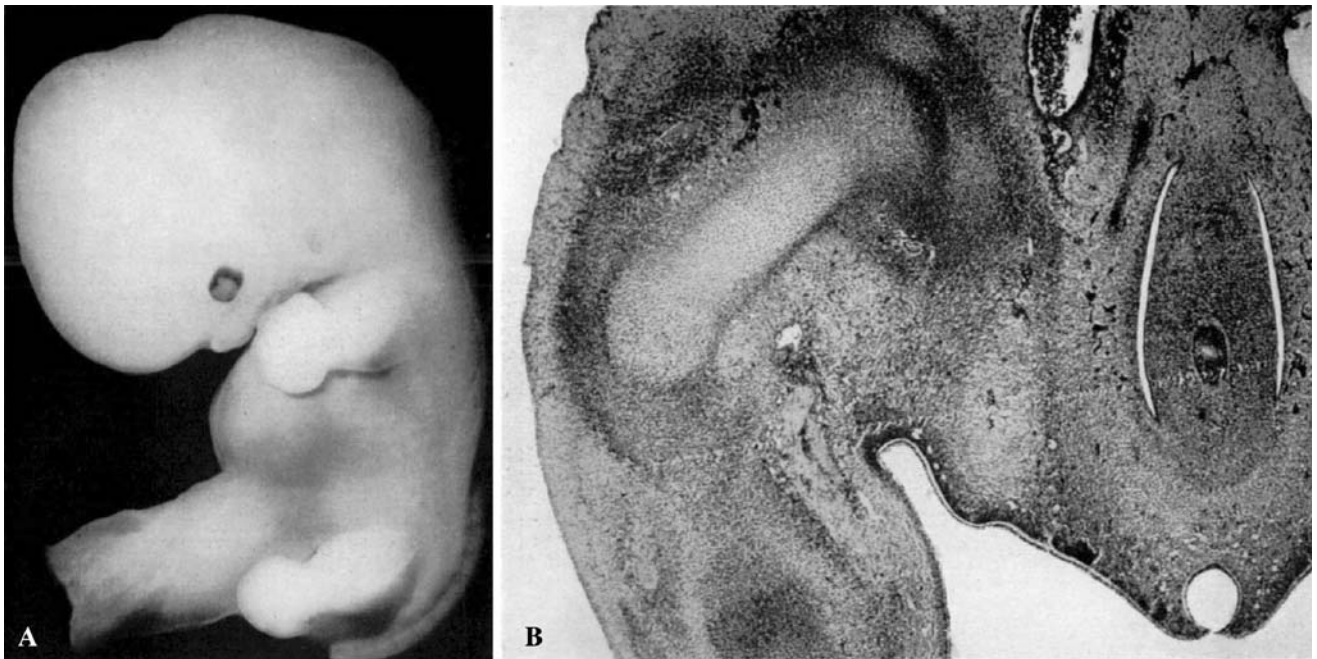
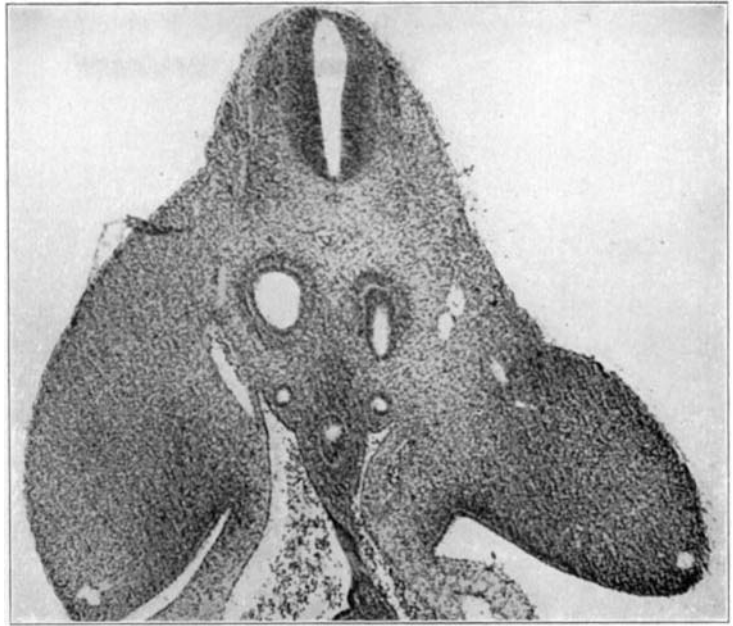


Fig. 3A–B (A) 14.8-millimeter embryo, demonstrating the alteration of position of the limb buds with the development of the knee bend and more central turning of the limb. The angle of inclination of the

neck is probably formed by this bending, prior to separation of the head of the femur from the acetabulum. (B) 14.8-millimeter embryo, after section. Outline of femur can be seen.

tissue above the head of the femur. The two components of the sciatic nerve are present, just medial to the tip of the trochanter. The external rotator muscles are well defined. The amount of abduction of the hip has diminished to about 60 degrees. Inasmuch as there is no separation of the head of the femur from the acetabulum, this must be the method for developing the angle of inclination of the neck of the femur.

As seen in the 33-millimeter embryo, the formation of the joint space is completely differentiated throughout, but clearly separate in the upper third. The ligamentum teres makes its appearance at this time, and the glenoidal labrum has extensive prolongation over the head of the femur. The capsule has separated from the glenoidal labrum and is attached above it; the articular surface of the acetabulum is continuous with the articular margin of the glenoidal

Fig. 4 Section of 28-millimeter embryo shows cartilaginous nature of innominate bone and femur.



labrum. The differentiation of muscle structures and nerves is clearly seen. Photographs of the embryo show the continued rotation of the limb buds (Figs. 5-A and 5-B).

In a section of a 53-millimeter female, taken at the level of the greater trochanter (Fig. 6), the ligamentum teres, the transverse acetabular ligament, and the glenoidal labrum are shown. At this stage the ligamentum teres is attached to the head; the capsule is attached above the glenoidal labrum.

In a 58-millimeter male, there was marked lessening of the abduction, and the ligamentum teres and its vascularity could be seen. The importance of the glenoidal labrum as a “sucker” ligament was obvious in the deepening of the socket. Beginning ossification of the ilium was taking place.

Comment

These sections reveal the development of the limb bud, and particularly of all the elements of the hip joint from the undifferentiated mesenchymal tissue to a structure that closely resembles the developed hip joint with all of its recognizable adult characteristics, in the short period of growth from an 8.5-millimeter embryo to a 53-millimeter female foetus. The hip-joint space was first noted in the 33-millimeter embryo in this series. Strayer pointed out that, in six embryos between 3b and 45 millimeters which he examined, development of the joint space was well under

way. He said it had been suggested that factors of muscle innervation and function probably have some influence on the time of opening of the joint space. Early maturation of the neuromuscular mechanism might cause a joint to be opened early, while a slowly developing neuromuscular apparatus might allow the embryo to reach a greater length before this occurred. This constitutes a possible determining time factor which may alter the rigid mosaic of the intrinsic design.

One must be impressed by the orderly development of this undifferentiated mesenchymal mass into the predetermined structural design of the mature characteristics of the hip joint. The intrinsic mosaic pattern for development in a normal environment is illustrated (Fig. 7). Can there be an inherited failure of development of the posterior superior border of the acetabulum in such a developmental cycle, indicating that all of the elements of the hip joint are differentiated *in situ* from one mass of mesoderm? Such a concept seems incredible.

Environmental Theory

Hereditary factors and environmental factors, such as geography and nutrition, may well alter the normal rate of growth and interfere with chronological development by delay or stimulation of growth. Delay in innervation of the muscles or in rotation of the limb buds at a period of rapid

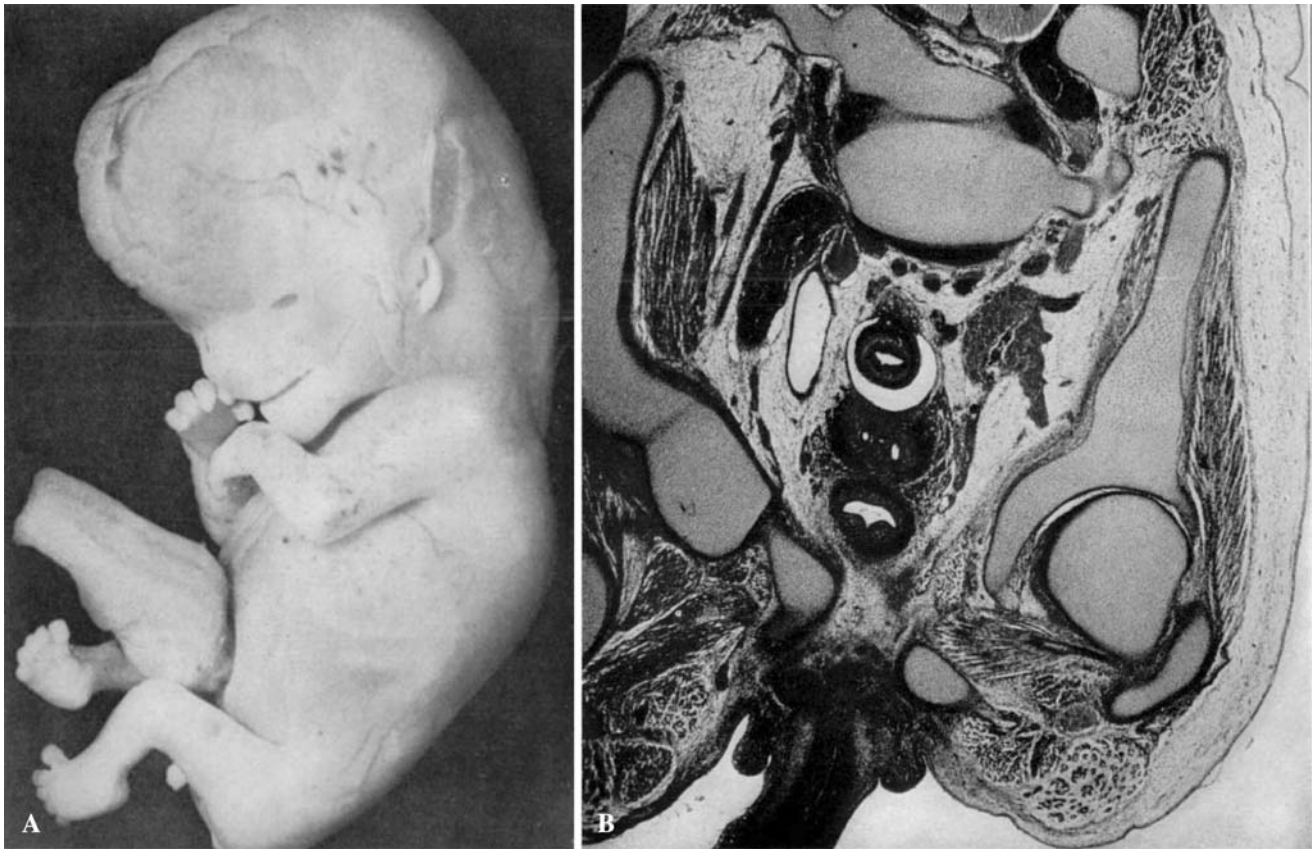


Fig. 5A–B (A) Photograph shows the continued rotation of the limb buds in a 33-millimeter embryo, with the patella still facing somewhat laterally. (B) Section of 33-millimeter embryo, showing the continued rotation of the limb buds. The capsule is separated from the glenoidal

labrum; the articular surface of the acetabulum is continuous with the articular margin of the glenoidal labrum. The differentiation of muscle structures and nerves is shown.

growth may produce an alteration in the deepening of the socket and changes in the head and neck of the femur. The adaptive variations to the stimulus of pressure and abnormal positions of the constituent parts of the hip joint recognized by clinicians in the postnatal changes, can certainly occur with even more marked alterations in a shorter period of time in the early prenatal phase. Why do we accept the postnatal changes in congenital dislocation of the hip as adaptive to malposition, and yet fail to recognize the potentialities of environmental or adaptive changes *in utero*?

Can it be said in one breath, as Murk Jansen and numerous of his followers have reported, that the flat socket is primarily the developmental fault, and in the next breath that the changes in the femur are secondary adaptive changes? Is it not more reasonable to assume in a structure of component parts, such as the hip joint, conjugated from a single mass into its integral parts, that the fault lies not in a hereditary failure of one part, but in an interference in the orderly time development of reciprocal parts after the formation of the joint cavity? It seems more logical to anticipate changes from extrinsic factors in both the

acetabular structures and the femur, on the basis of a secondary adaptive fault from an alteration in the normal timing of development. A delay in position, even of short duration, might well alter the normal dynamic rearrangement of the component parts, so that normal development is interfered with to the extent of reciprocal changes in the hip structures. These may well be manifested by a shallow socket, enlargement of the head to accommodate it, increased anteversion, and even subluxation. This would account logically for all of the recognized deformities noted in congenital dislocation of the hip.

The Glenoidal Labrum

It is, of course, an anatomical fact, that the acetabulum and its margins are entirely cartilaginous at the time of birth. The depth of the true acetabulum is greatly increased during prenatal life by the fibrocartilaginous structure called variously the limbus, the cotyloid ligament, and the glenoidal labrum. This ligament aids in enveloping the head of the femur. Severin developed an excellent illustration from a roentgenogram, demonstrating the size and

Fig. 6 Section of a 53-millimeter female embryo, taken at the level of the greater trochanter.



importance of the glenoidal labrum in the newborn, by painting with an opaque substance the dissected edge of the limbus. No one has mentioned any primary deficiency of the limbus, and yet it is an early embryonic development of obvious importance in increasing the depth of the socket and thereby maintaining stability of the femoral head within the socket.

Severin [27] also described how Faber demonstrated in normal hip joints, by opaquemedia arthrograms taken after birth, that the cartilaginous acetabulum and limbus covered at least half of the femoral head. The normal head is spherical in shape.

In subluxation, a term which we believe to be synonymous with dysplasia of the hip, the limbus is displaced upward, covering less of the head but still remaining above the head, which is between the limbus and the socket. The head is displaced, laterally, with apparent coxa valga, in Severin's illustrations. This malposition is the result of anteversion and not of coxa valga, as can readily be proved by roentgenograms taken with the lower extremity in internal rotation. In true dislocation, the limbus lies between the head and the acetabulum. Severin stated that the head glides past the glenoidal labrum which, because of its elasticity, returns to its position below the displaced head of the femur.

An important report was made by di Prampero, based on a roentgenographic study of 200 patients with unilateral dislocation, to determine the principal characteristics of the

so-called healthy side. Subluxation or dysplasia of the hip was found in 108 cases, subnormal hips in 41, and normal hips in only 51 cases. Eighty per cent of the so-called normal hips showed pathological changes. Di Prampero believed this parallelism of deformity demonstrated that the articular imperfection is not located in the hip, but in the pelvis as a whole and in the individual himself. With this observation the author is in full accord.

Various observers have demonstrated roentgenographic of determining abnormalities in the very young infant, leading to or suggesting the possibility of the development of so-called acetabular dysplasia or congenital dislocation of the hip. These measurements, namely, Wiber's lines, the acetabular index of Kleinberg, the Y line, and Shenton's line, all tend to demonstrate Putti's triad for so-called preluxation of the hip. This consists in:

1. An increased distance between the upper femoral epiphysis and the acetabular floor,—that is, lateral displacement;
2. Hypoplasia of the bony nucleus for the epiphysis of the head;
3. Increased angulation of the acetabular roof.

These changes are not evidence of a primary flat socket or of primary hip dysplasia. All can be produced by adaptive changes. The anteversion of the head and neck of the femur can be demonstrated by correction of the relationship through internal rotation and abduction only. How

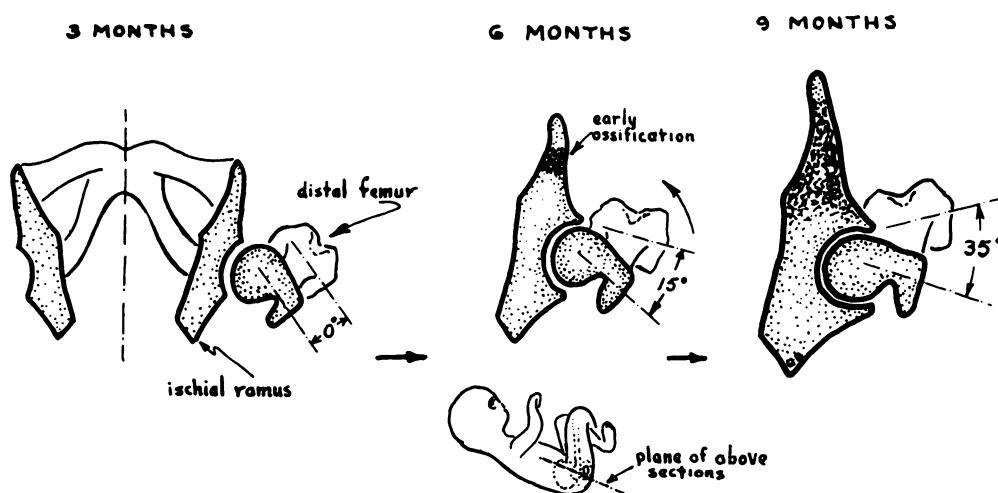


Fig. 7 Development of the normal hip joint. Graphic attempt to illustrate the reciprocal development of the head of the femur and the acetabulum, as the limb buds alter their lateral position to one of parallelism. The torsion of the femur, the change of position of the ilium, and the increasing anterior obliquity of the acetabulum produce

an increasing anteversion of the diaphyseal junction from 0 degrees at three months to 35 degrees at birth. The acetabulum steadily deepens and the head, properly fitting the socket at all stages of rotation, is symmetrically developed for the socket.

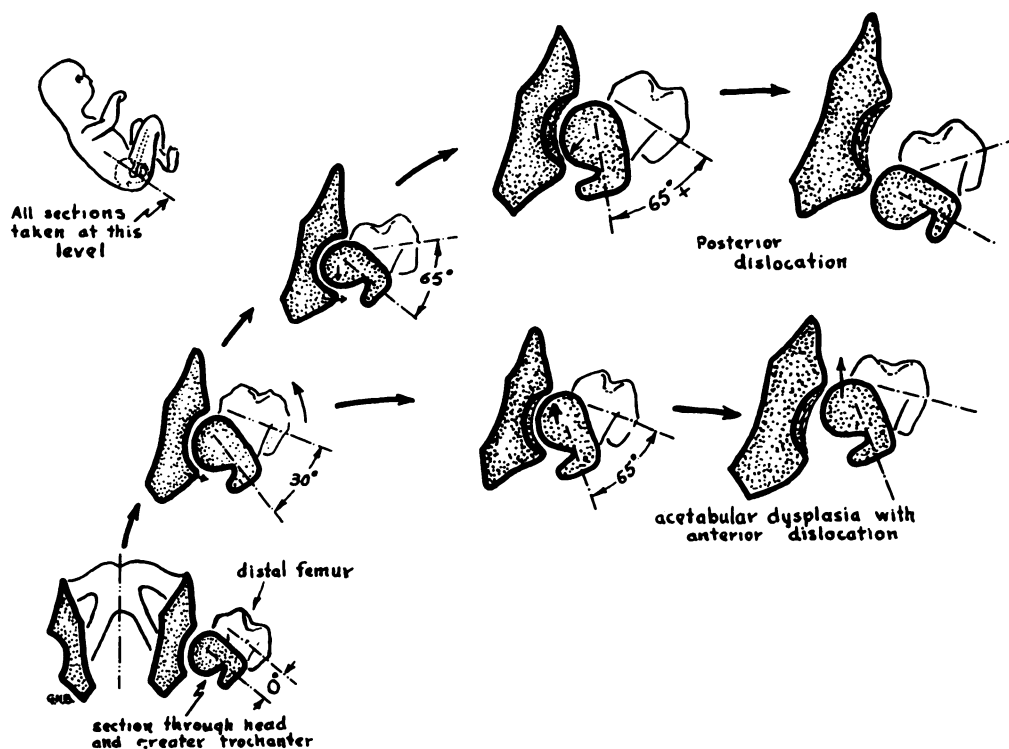


Fig. 8 Development of congenital dislocation of the hip and acetabular dysplasia. Schematic concept to illustrate abnormal alteration in various foetal ages from the 33-millimeter foetus to the matured infant. Thirty-millimeter foetus shows no rotation or torsion of femur and anteversion. Older foetus shows anteversion of 30 degrees, which increases in time to 65 degrees. The frontal obliquity of the acetabulum, plus the 65 degrees of anteversion, turns the head anteriorly away from the socket. The stimulus of contact and pressure, to produce normal deepening of the socket and the reciprocal shapes of the socket and the head of the femur, is lacking.

The head and neck, pointing forward, may rest against the posterior cartilaginous border of the socket and produce hypoplasia. The head may then become spontaneously displaced posteriorly. If, however, the delay in the proper timing of the rotation of the head into the socket is minimal or less prolonged as growth continues, anteversion with a malshaped head and a flat socket, may develop without the demonstration of a true dislocation, but rather with changes from mild to severe acetabular dysplasia. These are adaptive changes to a loss of the normal chronological fit of the head and socket, during the process of adaptation to parallelism of the limbs.

can one ignore the reciprocal changes in the head and neck in these young infants, or call them secondary changes, when they are as marked a part of the deformity as is the acetabular change? Convention and habit may be the chief reason. The author believes that both deformities are reciprocal faults, secondary to a developmental error, possibly both hereditary and environmental in character. They are not hereditary in the sense of a gene which fails to develop a part of the acetabulum, but rather a hereditary quality of interference of the timely development of the intrinsic mosaic pattern, producing environmental extrinsic faulty development which leads to the abnormality. This is entirely consistent with the known facts of embryological joint development. In what other joint formation has there been a failure of development from a primary developmental fault of part of a structure? One cannot compare the failure of approximation of separate components, such as is seen in rachischisis of the spine or cleft palate, with a structure originating as a single mesenchymal mass, differentiating originally as a continuous structure which separates into its conjugate parts after a growth of the fetus to 30 millimeters.

Anteversion

Some observers, including Fairbank, deny the frequency of anteversion in hip dysplasia and dislocations, possibly because of a faulty concept of where anteversion occurs. Anteversion occurs primarily in the diaphysis below the upper epiphyseal line. The head and neck may be in normal relation with the trochanters and yet anteverted in relation to the shaft; or, probably more accurately, torsion of the shaft is increased. Anteversion can occur with retroversion of the head. In fact, retroversion of the head may be a compensatory mechanism for anteversion. Most clinicians feel, however, that anteversion is a common accompaniment of hip dysplasias.

Murk Jansen stated: "In congenital dislocation of the hip, anteversion of the femoral neck... is a constant phenomenon". He attributed this to a primary flattening or widening of the socket. Tubby, Hibbs, Krida, and others have emphasized that anteversion of the neck is a frequent complication of congenital dislocation, usually a secondary phenomenon. Watkins stated that Lange had contended the head of the femur "originally left the acetabulum by the anterior route". He stated that the forward twist of the head and neck on the shaft was apparent in all cases of congenital dislocation in which the patient had walked.

The author has previously stressed that anteversion is not a secondary adaptive change, but is rather an integral reciprocal deformity, resulting and developing concurrently with the changes in the socket, the one dependent

upon the other for the extrinsic or environmental factors necessary for the dynamic development of the hip joint. The roentgenographic evidence of an apparent coxa valga is proved faulty when roentgenograms are taken with the lower extremities in internal rotation. The angle of inclination will be found to approximate the normal, demonstrating that coxa valga itself rarely occurs; but anteversion rotates the head and neck outward, producing this false or apparent coxa valga.

Similarly, we have mentioned that Putti's triad is evidence primarily of anteversion and not of true displacement. Internal rotation associated with abduction will show a normal angle of inclination, and the epiphysis is properly replaced in its relation to the acetabulum.

Conclusions

From the facts presented, it would seem logical to conclude that the etiology of congenital dislocation of the hip is a developmental fault of the hip system produced by extrinsic factors—growth circumstances—with a combination of hereditary and environmental faults which alter the normal growth potential or the intrinsic mosaic pattern. Congenital dislocation and congenital dysplasia are developmental deformities produced by secondary adaptive changes. Inherited characteristics and environmental factors may alter the intrinsic mosaic pattern by a faulty timing in development. Patten states that "local overgrowth may be responsible for certain anomalies, just as local arrests may be responsible for others...".

Our concept of congenital dysplasia of the hip and congenital dislocation is that, through a developmental fault, the acetabulum has failed to deepen and the head and neck of the femur have become anteverted. The anteversion tends to turn the head forward, displacing the cartilaginous sphere laterally, so that the glenoidal labrum and acetabulum cover less of the head than usual. The poor adaptation of the head and acetabulum continues dynamically to require altered growth changes, altering the intrinsic mosaic pattern. These growth changes are manifested in subluxation or acetabular dysplasia by pushing upward of the glenoidal labrum, widening of the socket, and enlargement of the head (Fig. 8). If the head escapes completely past the edge of the glenoidal labrum, a true dislocation results. The glenoidal labrum, unobstructed, returns by its elasticity to its proper position, lying between the head and the acetabulum. The head of the dislocated hip no longer has the stimulus for overgrowth seen in the subluxation, so that it remains small and round, although becoming flattened on the side if in contact with the ilium.

This mechanism is in complete accord with the pathological findings in early postnatal hip dysplasias,

demonstrated by arthrograms and by operative findings. It is to be remembered that anteversion occurs below the trochanters and actually is associated with torsion of the femoral shaft.

It would seem a logical hypothesis that the rotation of the limb buds may be an important factor in the abnormal development. Presumably there must be an adaptive alteration in the change of position, from the origin of the hip joint in the first few weeks of embryonic life to the 90 degrees of rotation and adduction of the hip in the second four weeks of life. Interference with the orderly timing of this rotation—the embryo being held in a position overlong for even a short time—could produce a failure, mild or severe, in the intrinsic mosaic design. The altered environment could produce the adaptive features which are seen in all the structures of the hip joint, and not a primary change in the acetabulum alone.

These studies lead to the following hypothesis: Congenital dislocation and congenital dysplasia of the hip may be regarded as the result of faulty development, due to environmental factors extrinsic to the hip joint. An inherited fault in the timing of development may produce these extrinsic changes. The loss of the normal dynamic reciprocal relationship of the component parts of the hip joint during the stage of rotational adjustment of the limb buds may produce the secondary adaptive changes which lead to acetabular dysplasia or congenital dislocation. Heredity can play an important part in altering the growth and time factors. The known embryological development of the hip joint is certainly opposed to the theory of a primary inherited failure of development of a portion of the acetabulum.

References

1. BADGLEY, C. E.: Rehabilitation in Cases of Arthrogryposis Multiplex Congenita. *Arch. Phys. Therapy*, **24**: 733–736, 1943.
2. BADGLEY, C. E.: Correlation of Clinical and Anatomical Facts Leading to a Conception of the Etiology of Congenital Hip Dysplasias. *J. Bone and Joint Surg.*, **25**: 503–523, July 1943.
3. BAILEY, F. R., and MILLER, A. M.: *Text-Book of Embryology*, Ed. 5. New York, William Wood and Co., 1929.
4. BARDEEN, C. R., and LEWIS, W. H.: Development of the Limbs, Body-Wall and Back in Man. *Am. J. Anat.*, **1**: 1–35, 1901.
5. BRUCE, JOHN: A Reconsideration of Congenital Dislocation of the Hip in Childhood. *Practitioner*, **125**: 642–648, 1930.
6. DEGA, W.: Ricerche anatomiche e meccaniche sull'anca fetale rivolte a chiarire l'etiologia e la patogenesi della lussazione congenita. *Chir. d. Org. di Movimento*, **18**: 425–505, 1933.
7. DONOVAN, J. M., and CAMPBELL, L. S.: To be published.
8. FABER, ALEXANDER: Erbbiologische Untersuchungen über die Anlage zur "angeborenen" Hüftverrenkung. *Ztschr. f. Orthop.*, **66**: 140–166, 1937.
9. FAIRBANK, H. A. T.: Congenital Dislocation of the Hip: With Special Reference to the Anatomy. *British J. Surg.*, **17**: 380–416, 1930.
10. HAMBURGER, VIKTOR, and WAUGH, MOLLY: The Primary Development of the Skeleton in Nerveless and Poorly Innervated Limb Transplants of Chick Embryos. *Physiol. Zool.*, **13**: 367–380, 1940.
11. HARRISON, R. G.: Cited by Hamburger and Waugh.
12. HART, V. L.: Primary Genetic Dysplasia of the Hip with and without Classical Dislocation. *J. Bone and Joint Surg.*, **24**: 753–771, Oct. 1942.
13. HEY GROVES, E. W.: The Treatment of Congenital Dislocation of the Hip-Joint. In *The Robert Jones Birthday Volume*, pp. 73–96. London, Oxford University Press, 1928.
14. HIBBS, R. A.: Anteversion of the Neck of the Femur. In Connection with Congenital Dislocation of the Hip. In *Trans. Sect. Orthop. Surg., Am. Med. Assn.*, pp. 48–51. Chicago, Am. Med. Assn. Press, 1915.
15. JANSEN, MURK: The Large Brain, the Wide Pelvic Girdle and the Outstanding Number of Hip Anomalies in Man (Coxa Vara, Coxa Fracta, Coxa Plana, Coxa Valga, Slipping Epiphysis, Malum Coxac) *J. Bone and Joint Surg.*, **11**: 461–531, July 1929.
16. KRIDA, ARTHUR: Congenital Dislocation of the Hip; The Effect of Anterior Distortion: A Procedure for Its Correction. *J. Bone and Joint Surg.*, **10**: 594–604, July 1928.
17. KRIDA, ARTHUR: A New Departure in the Treatment of Congenital Dislocation of the Hip. *J. Bone and Joint Surg.*, **13**: 811–815, Oct. 1931.
18. LE, DAMANY: Die angehorene Hüftgelenksverrenkung. Ihre Ursachen.—Ihr Mechanismus.—Ihre anthropologische Bedeutung. *Ztschr. f. Orthop. Chir.*, **21**: 129–169, 1908.
19. MCCARROLL, H. R., and CREGO, C. H., JR.: Primary Anterior Congenital Dislocation of the Hip. *J. Bone and Joint Surg.*, **21**: 648–664, July 1939.
20. MORRISON, L. B.: A Study of the Hip Joint from the Standpoint of the Roentgenologist. *Am. J. Roentgenol.*, **28**: 484–518, 1932.
21. MURRAY, P. D. F.: *Bones. A Study of the Development and Structure of the Vertebrate Skeleton*, p. 19. London, Cambridge University Press, 1936.
22. PATTEN, B. M.: *Human Embryology*. Philadelphia, Blakiston Co., 1946.
23. DI PRAMPERO, ANTONINO: Morfologia radiografica dell'anca cosi detta sana nella lussazione congenita unilaterale. *Chir. d. Org. di Movimento*, **25**: 1–16, 1939.
24. PUTTI, VITTORIO: Early Treatment of Congenital Dislocation of the Hip. *J. Bone and Joint Surg.*, **15**: 16–21, Jan. 1933.
25. PUTTI, V., e ZANOLI, R.: Tecnica dell'artrotomia per la riduzione della lussazione congenita dell'anca. *Chir. d. Org. di Movimento*, **16**: 1–28, 1931.
26. SEVERIN, E.: Early Treatment of Congenital Dislocation of the Hip. *Nord. Med. (Hygiea)*, **5**: 59–62, 1940.
27. SEVERIN, ERIK: Contribution to the Knowledge of Congenital Dislocation of the Hip. Joint. Late Results of Closed Reduction and Arthrographic Studies of Recent Cases. *Acta Chir. Scandinavica*, **84**: Supplementum 63, 1941.
28. STEINDLER, ARTHUR: *Mechanics of Normal and Pathological Locomotion in Man*. Springfield, Illinois, Charles C. Thomas, 1935.
29. STOCKARD, C. R.: Cited by Patten.
30. STRAYER, L. M., JR.: The Embryology of the Human Hip Joint. *Yale J. Biol. and Med.*, **16**: 13–26, 1943.
31. TUBBY, A. H.: Deformities Including Diseases of the Bones and Joints. A Text-Book of Orthopaedic Surgery, Ed. 2, Vol. 1. London, Macmillan and Co., Ltd., 1912.
32. WARKANY, JOSEF, and NELSON, R. C.: Skeletal Abnormalities in the Offspring of Rats Raised on Deficient Diets. *Anat. Rec.*, **79**: 83–100, 1941.

33. WARKANY, JOSEF, and NELSON, R. C.: Congenital Malformations Induced in Rats by Maternal Nutritional Deficiency. *J. Nutrition*, **23**: 321–333, 1942.
34. WARKANY, JOSEF, and SCHRAFFENBERGER, ELIZABETH: Congenital Malformations Induced in Rats by Maternal Nutritional Deficiency. VI. The Preventive Factor. *J. Nutrition*, **27**: 477–484, 1944.
35. WATKINS, JAMES: Congenital Dislocation of the Hip Reduced by Manipulation Followed by Arthrotomy. *In* *Trans. Sect. Orthop. Surg., Am. Med. Assn.*, pp. 52–57. Chicago, Am. Med. Assn. Press, 1915.
36. WEISS, PAUL: *Principles of Development. A Text in Experimental Embryology*. New York, Henry Holt and Co., 1939.