THE CANADIAN MEDICAL ASSOCIATION JOURNAL

LE JOURNAL DE L'ASSOCIATION MÉDICALE CANADIENNE

Volume 98 • Number 20 • May 18, 1968

Etiology, Pathogenesis and Possible Prevention of Congenital Dislocation of the Hip

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AS a seventh-generation Canadian, I feel particularly honoured in Canada's Centennial Year to have been invited to deliver the Eighth Alexander Gibson Memorial Lecture at the University of Manitoba. Having read the many glowing tributes to Dr. Alexander Gibson—and having been inspired by them—I only regret that, for chronological reasons, it was not my privilege to have had the opportunity to meet and know this outstanding man whom we all honour this evening.

One of the most inspiring tributes to Dr. Gibson came from the pens of the late Dr. R. I. Harris of Toronto and Dr. W. B. MacKinnon of Winnipeg; it reads as follows: "His qualities of integrity and sincerity, his capacity for clear thinking and convincing speech, his great fund of knowledge accumulated by study and experience, and his strong sense of duty made him a great citizen as well as a great surgeon."

As President of the Canadian Orthopaedic Association, Dr. Alexander Gibson designed the official crest of the Association. Of this crest, Dr. Gibson himself wrote as follows: "The significance of the design is that the maple leaf stands for English-speaking Canada, combined with the fleur-de-lys representing French-speaking Canada, the stalk of both emblems being in common."

Of the 77 scientific publications of this truly great scholar, including as they do many original ideas, it has been written that they "presented uncommon clarity of mind and lucidity of language which enabled him to make the complicated simple and the chaotic orderly". It has also been recorded that before permitting publication of any material, Dr. Gibson applied the following rigid formula: "No man has any right to publish unless he has something to say and has done his best to say it aright." Such high standards of academic excellence represent a challenging goal for all of us.

In the First Alexander Gibson Lecture, Sir Walter Mercer[1] referred to Dr. Gibson's lifelong interest in congenital dislocation of the hip among Indian children in northern Canada. In the Fifth Alexander Gibson Lecture, Professor William Boyd,[2] a classmate and close personal friend of Dr. Gibson, discussed what he referred to as "a subject of general interest to every scientist and to every doctor, namely, the relation of *cause* to *effect*". It would seem appropriate, therefore, in the present Alexander Gibson Lecture to include some aspects of both of these subjects in a discussion of the etiology, pathogenesis and possible prevention of congenital dislocation of the hip.

Congenital dislocation of the hip represents one of the most important and most challenging congenital abnormalities of the musculoskeletal system. Since its detection at birth requires a specific method of examination, it is still not being recognized sufficiently early throughout the world, and may even escape detection until after the child has started to walk. Under these circumstances, treatment becomes progressively more difficult and the results become progressively less satisfactory. Furthermore, congenital dislocation of the hip, unless treated early and skilfully, leads inevitably to degenerative arthritis of the hip in adult life (Fig. 1).

In most congenital abnormalities of the musculoskeletal system, such as clubfoot and spina bifida, the anatomical deformity is maximal at birth and it is obvious that the abnormality has been present from an early stage of intrauterine development. In congenital dislocation of the hip, by contrast, the anatomical deformity

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The Eighth Alexander Gibson Memorial Lecture, presented at the University of Manitoba on October 27, 1967.
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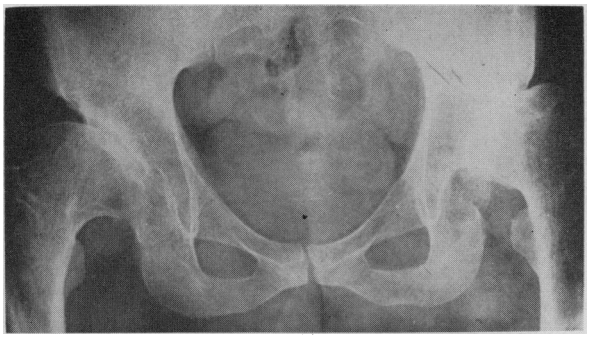

Fig. 1.—Radiograph of the hip joints of a 45-year-old woman who is disabled by painful bilateral degenerative arthritis of the hips as a late result of unsuccessful treatment of congenital dislocation of the hips in early childhood.

is minimal at the time of birth and, if untreated, becomes progressively more marked during postnatal growth. This observation raises the possibility of prevention of at least the postnatal acquired deformities of the hip which are secondary to the original dislocation. It also raises the possibility even of preventing the initial dislocation. Thus, a consideration of the etiological factors and the sequence of events in the pathogenesis of congenital dislocation of the hip is of more than academic interest.

Unfortunately, the controversial question of the etiology of congenital dislocation of the hip has given rise to a plethora of theoretical speculations and a paucity of scientific investigations. Each of the many physical phenomena associated with congenital dislocation, such as acetabular dysplasia, femoral anteversion and elongation of the capsule, has at some time been considered to be the primary cause of the dislocation.

In the organization of this lecture, I have chosen firstly to discuss many of the proven facts about congenital dislocation of the hip as well as my interpretation of these facts; secondly, to develop a hypothesis concerning the etiological factors and the sequence of events in the pathogenesis of this abnormality, and thirdly, on the basis of this hypothesis, to suggest what *might* be done to prevent the initial dislocation. It should be pointed out that the present discussion concerns only the "typical" form of congenital dislocation of the hip to the exclusion of the "atypical" teratologic form associated with arthrogryposis and spina bifida.

What are the known facts about congenital dislocation of the hip? Facts, as opposed to opinions, are of the utmost importance to us; they are the building blocks with which we must build the structure of hypothesis, and the more facts we can gather, the more complete will be our hypothesis. Furthermore, we must make the hypothesis fit the known facts rather than try to make the facts fit a preconceived hypothesis.

FACTS ABOUT CONGENITAL DISLOCATION OF THE HIP

1. *Facts Concerning Embryology*

It has been established by Strayer[3] that the hip joint develops from a single mass of mesodermal tissue in the blastema, or primary limb bud. By about the tenth week, a joint space appears in this mass of mesodermal tissue and joint movement becomes possible. Therefore, it would seem reasonable to assume that the hip joint is not dislocated from the beginning of its formation, but that something must happen to it during its late prenatal or early postnatal development that produces a dislocation.

2. *Facts Concerning Anatomy*

The normal hip joint is one of the most stable of all synovial joints in the body. Three structural factors contribute to this stability: the shape of the opposing bony and cartilaginous joint surfaces of this ball and socket joint, the action of the muscles controlling hip movement, and the integrity of the capsule and ligamentum teres. From a clinical and radiographic study, including arthrography, of congenitally dislocated hips in newborn infants, it is apparent that the shape of the joint surfaces is virtually normal at birth; furthermore, the shape of the joint surfaces becomes progressively abnormal only if the hip remains dislocated during subsequent growth. Therefore, the shape of the joint surface at the time of birth cannot be an etiological factor in the initial dislocation. Clinical studies of paralytic dislocations of the hip secondary to poliomyelitis and spina bifida reveal that while muscle imbalance alone can indeed result in dislocation of the hip, it does so only gradually and over a relatively long period of time. As far as can be determined, in congenital dislocation of the hip the surrounding muscles are perfectly normal at the time of birth. The importance of the fibrous capsule in hip joint stability is emphasized by some studies that we have conducted in the postmortem room on the hip joint of stillborn infants. These anatomical studies reveal that the normal hip can be made to dislocate only after the capsule and ligamentum teres have been divided. I will enlarge upon these studies subsequently.

These clinical, radiographic and anatomical observations suggest that of the three possible structural factors that could account for the instability of the congenitally abnormal hip, the most significant is an abnormality of the joint

capsule and ligamentum teres. Furthermore, the most likely abnormality is an undue laxity of these structures as demonstrated by Andren,[4] as well as by Carter and Wilkinson.[5]

3. *Facts Concerning the Incidence of Congenital Dislocation of the Hip*

(a) *General incidence.*—While the general incidence of clinically detected congenital dislocation of the hip is approximately 1 to 1.5 per 1000 live births, the incidence of this abnormality—unlike the incidence of other equally common congenital musculoskeletal abnormalities, such as clubfoot—varies widely in different parts of the world. This suggests the possibility that postnatal environmental factors may be superimposed upon the original congenital abnormality and have an effect, either for better or for worse, on the natural course of the condition. It also suggests that there may be a racial variation in the incidence of any underlying genetic factor.

(b) *Hereditary and familial incidence.*—In approximately 20% of children with congenital dislocation of the hip, a family history of the abnormality can be obtained. This means that in 80% of the children the abnormality has appeared for the first time in the family. Furthermore, in a clinical study conducted at The Hospital for Sick Children, Toronto, in collaboration with Mrs. D. Wilson Cox[6] of the Department of Genetics, we found that of 226 children—one parent of whom had congenital dislocation—only four had the abnormality. This means that the other 222 children, each of whom had one afflicted parent, were perfectly normal (Table I). Therefore, the genetic factor alone cannot explain the incidence of congenital dislocation of the hip.

TABLE I.—INCIDENCE OF CONGENITAL DISLOCATION OF THE HIP IN OFFSPRING WHEN ONE PARENT IS AFFLICTED

Based on a genetic study of 226 offspring, The Hospital for Sick Children—1963.

Sons	1 in 112.....Incidence of 0.9%
Daughters	3 in 114 (1 in 38).....Incidence of 2.6%

Furthermore, studies of identical twins by Idelberger[7] reveal that of 29 pair of identical twins, both twins were afflicted in only 10 of the pairs. Thus, in two out of three pair of identical twins, one twin was afflicted and the other was normal despite an identical genetic composition. All of these observations on the hereditary and familial incidence indicate that while there is definitely a genetic factor in the etiology of congenital dislocation of the hip, there must be other factors as well.

(c) *Sex incidence.*—The repeated observation that 80 to 90% of the children with congenital dislocation of the hip are girls is very significant in that such a predominantly female incidence is not seen in other congenital abnormalities of the musculoskeletal system. Furthermore, there is the impression, which cannot be documented, that the girls with congenital dislocation of the hip tend to be more feminine than the average. They have a very feminine type of body build with wide hips. In addition, they are rather coquettish and know how to use their eyes even as young girls. Furthermore, the boys who do have congenital dislocation of the hip tend to be less masculine (or more feminine) than the average. These observations suggest the possibility of an endocrine or hormonal factor. Recent investigations by Andren and Borglin[8] suggest that an abnormality of estrogen metabolism in afflicted infants may be responsible for the laxity of the hip joint capsule associated with congenital dislocation of the hip.

(d) *Age incidence.*—This may seem to be an unusual point to consider in any condition that is deemed congenital. However, there is growing evidence that the initial dislocation does not always occur at the same chronological age. While the atypical or teratologic type of dislocation almost certainly occurs long before birth, the typical type usually occurs at or shortly after birth and in most infants probably within the first two weeks. Rarely, however, we see a child in whom the initial dislocation does not appear until the child begins to stand. In these children there is no dysplasia of the acetabulum, which is another reason why we believe that dysplasia is secondary to the dislocation; that is, that the dysplasia is the result rather than the cause.

(e) *Geographical and racial incidence.*—The wide variation in the incidence of congenital dislocation of the hip encountered in different geographical areas and among different races is

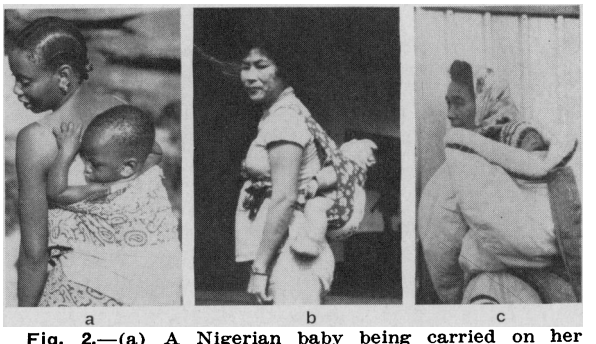

Fig. 2.—(a) A Nigerian baby being carried on her mother's back with the hips in flexion and abduction. (b) A Chinese baby in Hong Kong being carried in a sling on his mother's back with the hips in flexion and abduction. (c) A northern Canadian Eskimo baby being carried in her mother's parka; the baby is in a sitting position with the hips in flexion and abduction.

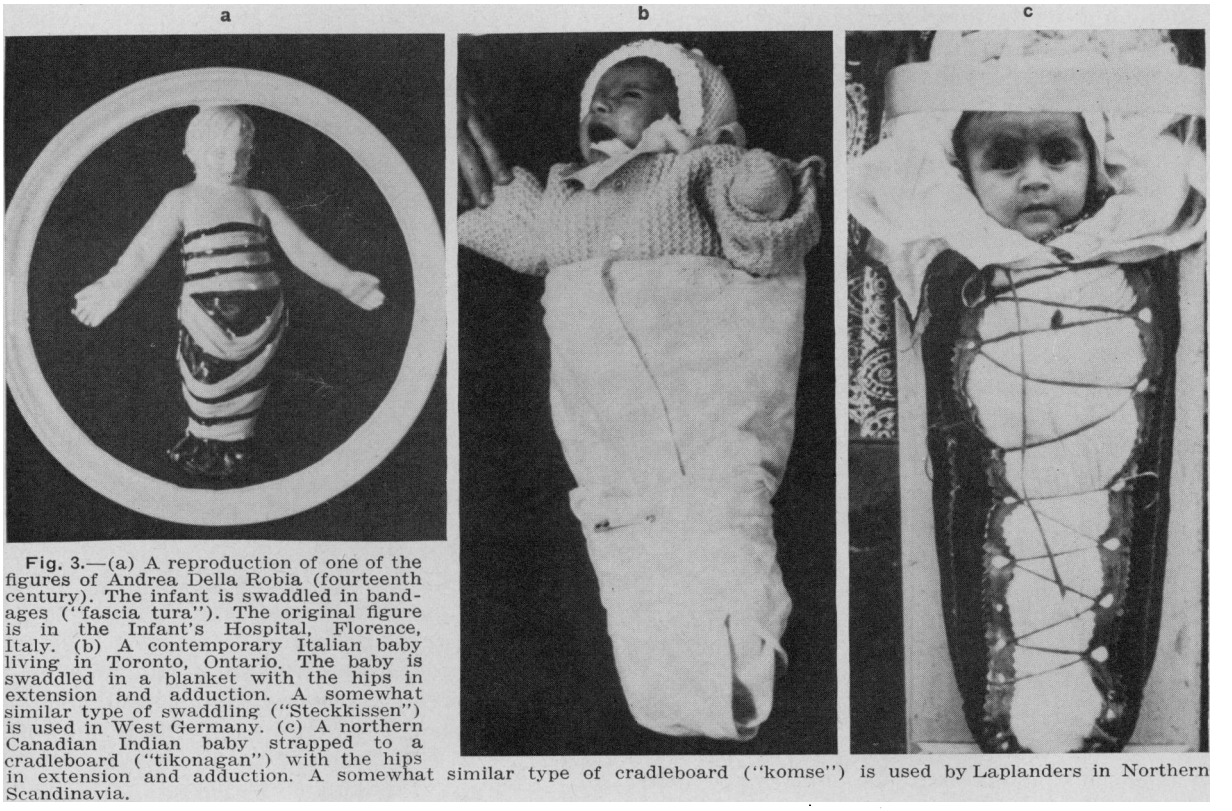

Fig. 3.—(a) A reproduction of one of the figures of Andrea Della Robia (fourteenth century). The infant is swaddled in bandages ("fascia tura"). The original figure is in the Infant's Hospital, Florence, Italy. (b) A contemporary Italian baby living in Toronto, Ontario. The baby is swaddled in a blanket with the hips in extension and adduction. A somewhat similar type of swaddling ("Steckkissen") is used in West Germany. (c) A northern Canadian Indian baby strapped to a cradleboard ("tikonagan") with the hips in extension and adduction. A somewhat similar type of cradleboard ("komse") is used by Laplanders in Northern Scandinavia.

not seen in relation to other congenital abnormalities of the musculoskeletal system. This wide variation is undoubtedly due to a combination of genetic factors and environmental influences. A study of the geographical and racial incidence suggests that one of the environmental influences that may be significant in the etiology of the initial dislocation is the position in which the hips of newborn infants are maintained during the early months of postnatal growth and development. Among those races in which the hips of newborn infants are commonly held in flexion and abduction, the incidence of congenital dislocation of the hip is remarkably low. Low-incidence groups are the Negroes of Central and South Africa,[9, 10] the Chinese of Hong Kong[11] and the Eskimos of Northern Canada (Figs. 2a, 2b and 2c). The position of flexion and abduction is the very position maintained by various types of splints during the treatment of infants with congenitally unstable hips.

By contrast, among those races in whom the hips of newborn infants are commonly held in extension and adduction by various methods of swaddling, the incidence of congenital dislocation of the hip is remarkably high. Examples of high incidence groups are Northern Italians,[12] North American Indians,[13-16] West Germans[17] and Laplanders of Northern Scandinavia[18] (Figs. 3a, 3b and 3c).

These observations suggest that a congenitally unstable and dislocatable hip, which has been maintained in the position of flexion and abduction during intrauterine life, is "protected" by the postnatal position of flexion and abduction and made worse by the postnatal position of extension and adduction. It would seem that the newborn hip joint of the human is not developmentally prepared for a sudden, complete and maintained change from the intrauterine position of flexion to the erect position of extension in the early months of postnatal life, particularly if the hip joint is already congenitally unstable from some cause such as capsular laxity.

While it is difficult to separate the genetic factors from the environmental influences in these observations, a study of Indian children in Northern Canada has proved helpful. We found that whereas the incidence of congenital dislocation of the hip throughout the Indian population

TABLE II.—CORRELATION OF THE CRADLEBOARD (TIKONAGAN) TO THE INCIDENCE OF CONGENITAL DISLOCATION OF THE HIP

Based on a Survey of Canadian Indian Tribes, 1963	
Cradleboard not used	17 cases C.D.H. in 1347 live births (1.2% incidence)
Cradleboard used	250 cases C.D.H. in 2032 live births (12.3% incidence)

Incidence of C.D.H. 10 times greater with use of cradleboard.

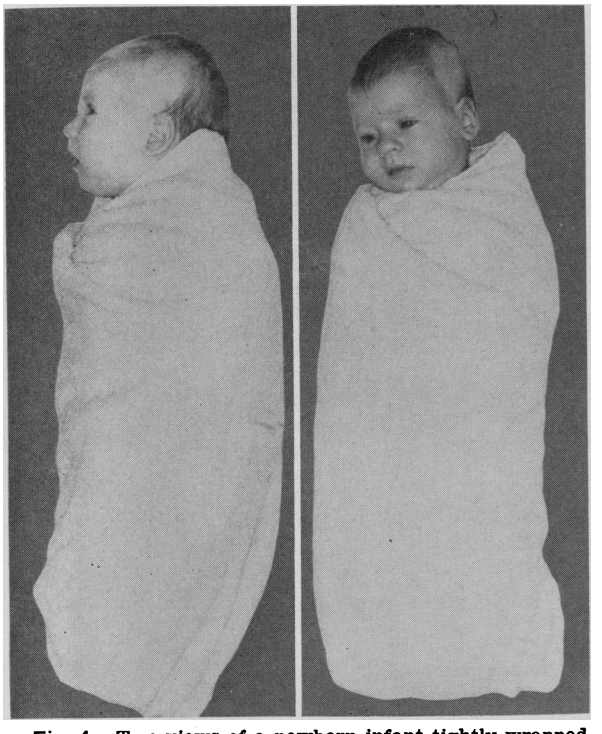

Fig. 4.—Two views of a newborn infant tightly wrapped in a blanket. The hip joints are maintained in extension and adduction.

is higher than in the white population of Canada (possibly due to consanguinity), the incidence in those tribes in which the cradle board (tikonagan) was used for their children was 10 times higher than in those tribes in which it was not used (Table II). Studies by Rabin *et al.*[15] as well as by Houston and Buhr[16] have not, however, shown such a striking relationship between the use of the cradleboard and the incidence of congenital dislocation of the hip. An extremely low incidence, on the other hand, may not be so clearly related to postnatal position of the hips. Edelstein,[9] in an examination of 9000 South African Negro newborn infants, found no congenital instability of the hips—an observation that may be due to the absence of predisposing genetic factors in this particular racial group. Huckstep[10] has made similar observations in Central Africa.

(f) *Seasonal incidence.*—Record and Edwards[19] in England and others in some of the European countries have reported a significantly higher incidence of congenital dislocation of the hip in children who are born during winter months. This has not been noticed in more temperate climates or where central heating is generally used. This interesting seasonal incidence suggests the possibility that, when the weather is cold, the newborn babies and young infants may be more tightly wrapped in blankets which passively extend the hips and limit active movement (Fig. 4). The common habit of wrapping infants tightly in blankets with their hips in extension and adduction should be abandoned.

(g) *Incidence in relation to birth presentation.*—In a combined clinical and genetic study of congenital dislocation of the hip at The Hospital for Sick Children,[20] we found that 23% of all children with congenital dislocation of the hip had been delivered as a breech presentation, whereas in the normal population the incidence of breech presentation is below 5%. Furthermore, the incidence of congenital dislocation of the hip is 10 times higher in children born by breech presentation than in those born by vertex presentation. Another fascinating aspect of this study is that in those children with congenital dislocation of the hip who have been born by breech presentation, the usually predominant female sex incidence is much less striking—only two to one, instead of nine to one. These facts suggest that the initial dislocation in a congenitally unstable hip might be caused (at least in this group of infants) by an iatrogenic factor such as forceful passive extension of the hips associated with traction on the lower limbs during delivery.

4. *Facts About Physical Findings in Children*

The physical findings associated with congenital dislocation of the hip vary with the child's age, and this suggests a progression of changes in the hip after birth. Within the first few days of postnatal life, the most significant

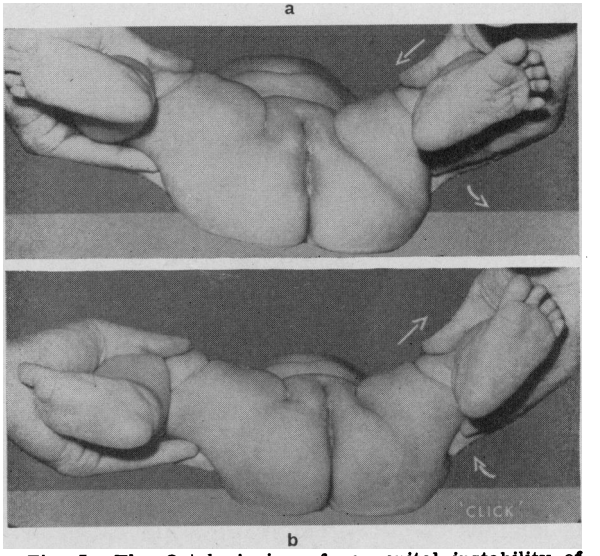

Fig. 5.—The Ortolani sign of congenital instability of the hip joint. (a) With slight adduction and pressure along the long axis of the thigh, the hip can be felt to dislocate or subluxate posteriorly. (b) With slight abduction and traction on the thigh the hip can be felt to relocate.

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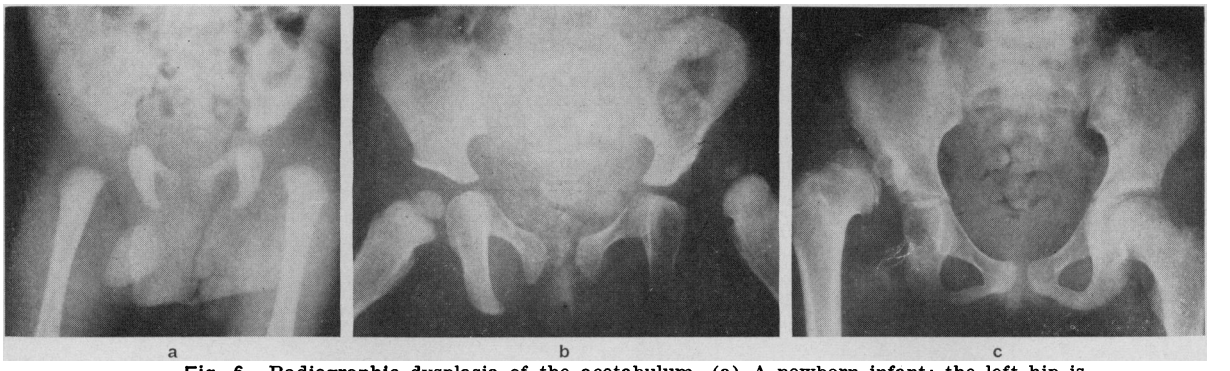

Fig. 6.—Radiographic dysplasia of the acetabulum. (a) A newborn infant; the left hip is completely dislocated but there is no evidence of dysplasia of the acetabulum. (b) A 15-month-old child with an untreated dislocation of the left hip; the dysplasia of the left acetabulum and femoral head is quite marked. (c) A 10-year-old girl with an untreated congenital dislocation of the right hip; the dysplasia of the acetabulum is extreme.

and indeed often the only physical sign is instability of the involved joint as depicted by the Ortolani sign[21] (Fig. 5a and 5b). At this stage the hip joint is lax and is dislocatable but not permanently dislocated and there is usually no adduction contracture. Barlow,[22] in an examination of over 9000 newborn infants in England, found that on the first day of life 1 infant in 60 had instability of one or both hips. Of these, 68% became stable within one week, and 88% had become stable by the end of the second month, leaving only 12% of the original group still demonstrating instability—an incidence of 1.5 per 1000 live births (which is the general incidence of congenital dislocation of the hip). This suggests that the primary problem in the newborn period is congenital laxity of the hip joint capsule with resultant instability of the joint. It also suggests that there is a tendency for the capsular laxity to improve spontaneously, provided the hip does not remain dislocated. After the first few months of life, if the hip remains dislocated, the well-known physical signs of adduction contracture, apparent and real shortening, telescoping and a Trendelenburg sign become progressively more apparent and indicate anatomical changes that would seem to be the result of the dislocation rather than the cause.

5. *Facts Concerning Dysplasia of the Acetabulum*

In congenital dislocation of the hip, radiographic dysplasia of the acetabulum is minimal, and indeed often undetectable, on the first day of life, but it becomes progressively more severe as long as an abnormal relationship exists between the head of the femur and the acetabulum (Figs. 6a, 6b and 6c). Furthermore, during at least the first year of life, the acetabular dysplasia is to a large extent reversible, provided a normal relationship between the femoral head and the acetabulum has been restored (Figs. 7a and 7b). These observations suggest that dysplasia is not a primary genetic defect of the

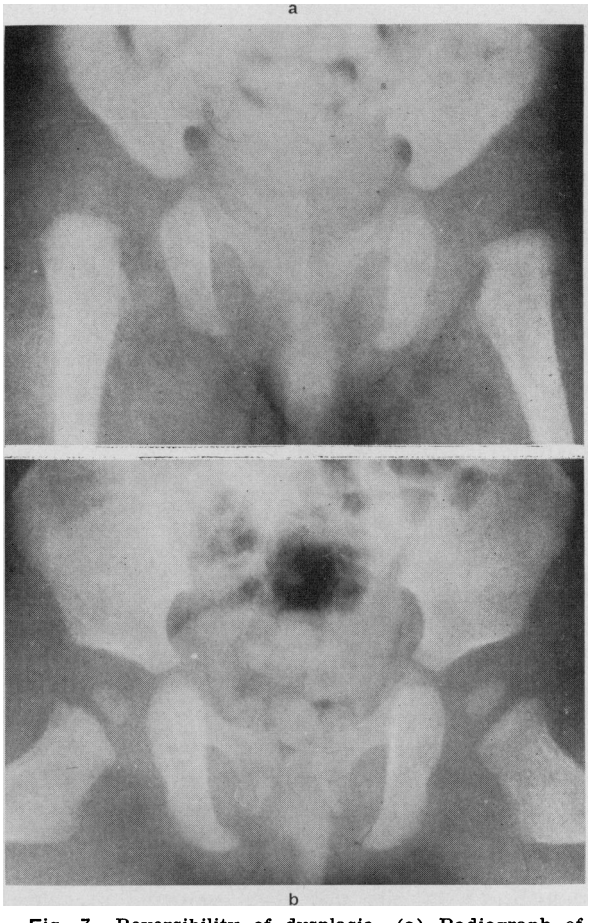

Fig. 7.—Reversibility of dysplasia. (a) Radiograph of the hips of a 4-month-old child with bilateral congenital dislocation of the hip. There is a moderate degree of dysplasia of the acetabulum on each side. (b) Radiograph of the same child one year after bilateral closed reduction; both hip joints have developed well, indicating that the acetabular dysplasia is reversible in the very young child provided a normal relationship between the femoral head and acetabulum has been restored and maintained.

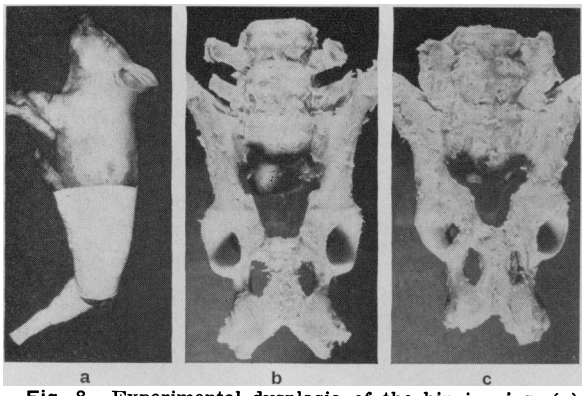

Fig. 8.—Experimental dysplasia of the hip in pigs. (a) Newborn pig with the left hip maintained in flexion and the right hip in extension. (b) Normal pelvis of a 6-week-old control pig. Note the direction in which each acetabulum faces. (c) Pelvis of a 6-week-old experimental pig. The left hip had been maintained in flexion and the acetabulum on this side has developed normally. The right hip was maintained in extension during the six-week period. The right acetabulum is dysplastic: it is not only smaller and more shallow but it also faces in a completely abnormal direction.

acetabulum, but rather that it is secondary to displacement.

Experimental investigations by Smith *et al.*[23] using dogs, by Harrison[24] using rats and by Langenskiöld, Sarpio and Michelsson[25] using rabbits revealed that dysplasia of the acetabulum always followed experimentally produced dislocation.

In an experimental investigation using newborn pigs[26] we have shown that maintained extension of the hip joint for six weeks consistently resulted in dysplasia of the acetabulum, whereas maintained flexion of the hip joint led to normal acetabular development (Figs. 8a, 8b and 8c). Furthermore, the most impressive feature of the dysplasia was the abnormal direction in which the entire acetabulum faced. In addition, we have shown in another series of pigs that the experimentally produced dysplasia was reversible when the hip was released and the animals were allowed to run free for a period of 10 weeks (Figs. 9a and 9b).

The results of these experimental investigations suggest that maintained extension of the newborn hip joint has a deleterious effect on its subsequent development, whereas maintained flexion has a beneficial effect. These experimental results in animals, as well as the clinical investigations in children, suggest that in congenital dislocation of the hip, the dysplasia, rather than acting as a cause, is a result of the dislocation.

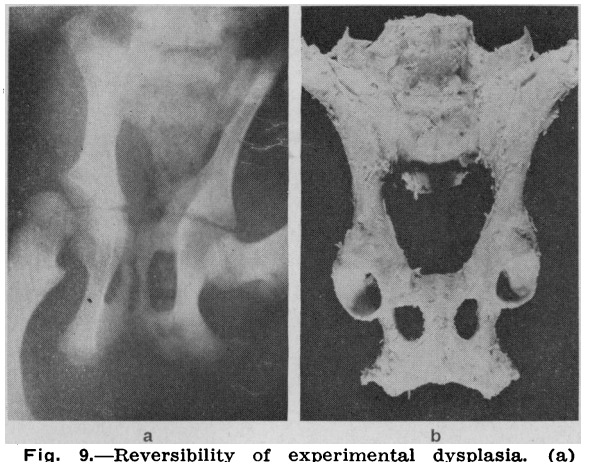

Fig. 9.—Reversibility of experimental dysplasia. (a) Radiograph of the hip of a 6-week-old experimental pig. The left hip had been maintained in flexion and the acetabulum on this side has developed normally. The right hip was maintained in extension during the six-week period. The right acetabulum is dysplastic. The "sloping acetabular roof" is an indication of the abnormal direction in which the acetabulum faces. (b) Pelvis of a 16-week-old experimental pig. The left hip had been maintained in extension for six weeks and was then released so that the animal could run free. Radiographic examination at that time revealed dysplasia of the left acetabulum. During the ensuing ten weeks the dysplasia of the left hip was almost completely reversed.

6. *Facts Concerning Acetabular Direction*

The direction in which the acetabulum faces is difficult to assess by radiographic examination limited to two dimensions, and can be determined correctly only by seeing the entire acetabulum in three dimensions. At birth, the normal acetabulum faces more forward and more laterally than it does in adult life, and it is presumed that the change in direction takes place gradually as the position of the hip changes from the intrauterine position of flexion and abduction to the erect position of extension and adduction. However, if the hip dislocates at or shortly after birth, there is no longer any corrective force being applied to the acetabulum through the capsule and, as a result, the acetabulum continues to face relatively forward and laterally. This residual abnormal direction of the entire acetabulum is best appreciated in children over the age of 18 months at the time of open reduction for a congenital dislocation, particularly

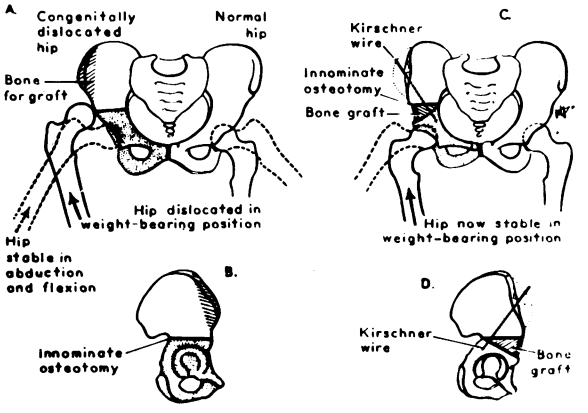

Fig. 10.—The principle of innominate osteotomy—redirection of the entire acetabulum as a unit.

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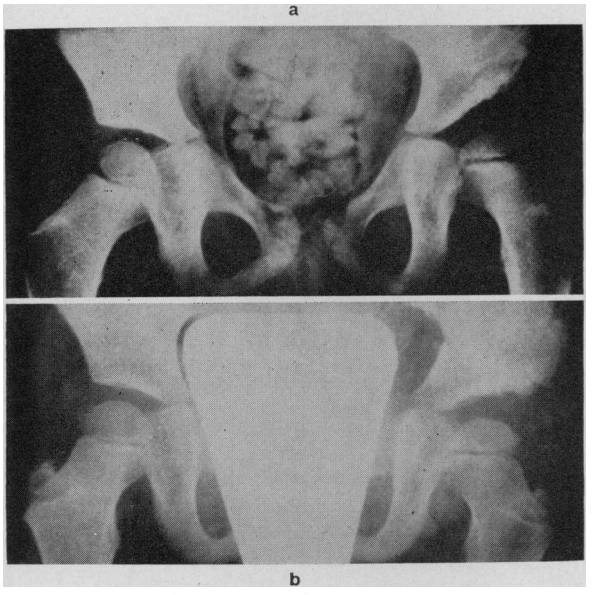

Fig. 11.—Preoperative and postoperative radiographs of the hips of a 4-year-old girl. (a) Residual acetabular dysplasia and subluxation despite one year of closed treatment; there would seem to be "sloping of the acetabular roof". This child's left hip was then treated by innominate osteotomy. (b) Six weeks after innominate osteotomy the left acetabulum appears comparable to the right. The only change during the six-week interval had been redirection of the entire acetabulum by the operation. Therefore, the correct interpretation of (a) is an abnormal direction of the entire acetabulum (acetabular maldirection) rather than a "sloping of the acetabular roof".

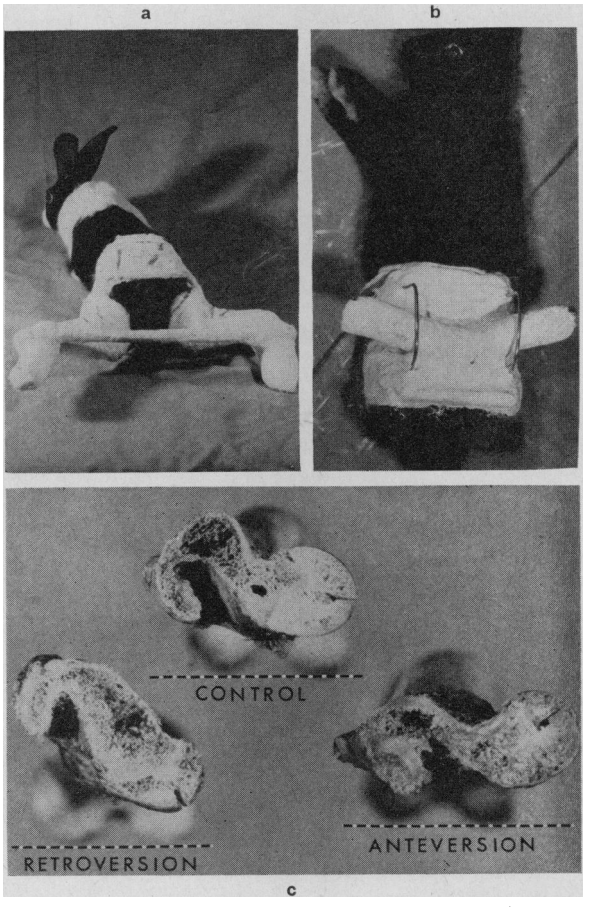

Fig. 12.—Experimental investigation of femoral anteversion in growing rabbits with the hips maintained in a position of (a) full internal (medial) rotation and (b) full external (lateral) rotation. (c) Axial views of femoral necks from above; (top) normal femur of control rabbit—there is no anteversion; (left) retroversion of the femoral neck from a rabbit whose hips had been maintained in external (lateral) rotation for six weeks; (right) femoral anteversion from a rabbit whose hips had been maintained in internal (medial) rotation for a period of six weeks.

when compared with the normal acetabulum of a child of comparable age in the postmortem room. Therefore, we have come to interpret the usually described "sloping of the acetabular roof" as seen in the radiograph not as a defect in the roof, but rather as an abnormal direction of the entire acetabulum, i.e. acetabular maldirection. Indeed, this concept is pivotal in the principle of innominate osteotomy (Fig. 10), which is re-direction of the entire acetabulum as a unit.[27] Further evidence of this interpretation is provided by a comparison of the preoperative radiographs of a child with residual acetabular dysplasia and the radiographs following innominate osteotomy (Figs. 11a and 11b).

7. *Facts Concerning Femoral Anteversion*

At birth the average normal femoral anteversion is approximately 30°, but under normal circumstances it gradually decreases with subsequent growth to 10° or less. However, if the hip dislocates at or shortly after birth, there is no longer any corrective force being applied to the upper end of the femur and, as a result, the femoral anteversion not only persists but may even increase. During the year or so following complete restoration of the stability of the hip in young children, the femoral anteversion corrects spontaneously with further growth. These observations suggest that the femoral anteversion, rather than being a cause of the dislocation, is a result of the dislocation. Furthermore, we have shown by experimental investigation in rabbits that a maintained position of forced internal rotation of a growing hip increases femoral anteversion during subsequent growth, whereas a maintained position of forced external rotation results in femoral retroversion (Figs. 12a, 12b and 12c).

8. *Facts Concerning the Hip Joint Capsule*

Howorth[28] has rightly pointed out that the most significant anatomical finding in congenital dislocation of the hip at the time of birth is laxity and elongation of the hip joint capsule and the ligamentum teres. Indeed, since the dislocation is intracapsular, the capsule and ligamentum teres must be elongated, as can be seen

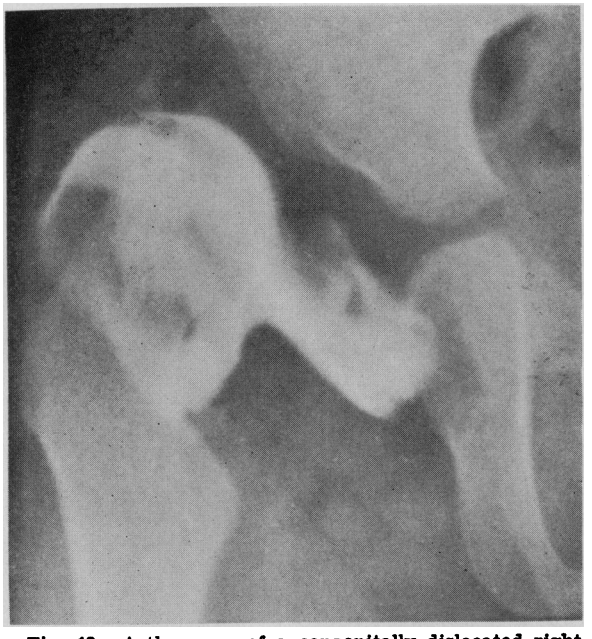

Fig. 13.—Arthrogram of a congenitally dislocated right hip joint in a 2-year-old child. The dislocation is intracapsular and the capsule has become markedly elongated.

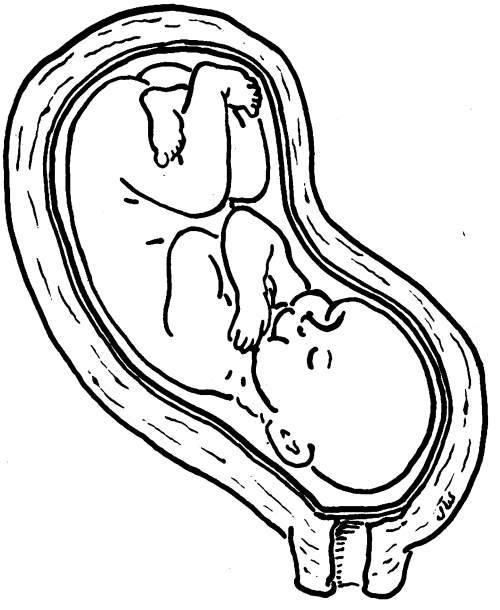

Fig. 14.—Fetal position *in utero*. Regardless of the position of the fetus, the hips are always maintained in a position of flexion.

in an arthrogram (Fig. 13) However, the cause of the underlying capsular laxity is as yet unknown. It is known that children with congenital dislocation of the hip tend to show a greater degree of generalized congenital laxity of ligaments than those without a dislocation; this has been well documented by Carter and Wilkinson[5] and by Wilkinson.[29] However, there may well be an additional factor acting in the newborn period that unduly increases the capsular laxity. Andren[4] and Andren and Borglin[8] have demonstrated excessive laxity of the hip joints as well as of the pelvic joints in newborn infants with congenital dislocation of the hip and ascribe this phenomenon to the effect of maternal hormones on the fetus. This type of investigation merits further attention, since it would seem that undue laxity of the hip joint capsule and ligamentum teres at the time of birth is the most important structural abnormality that permits the initial dislocation when the hip is first extended passively. Moreover, it may well be that the genetic factor in the etiology of congenital dislocation of the hip is related to the degree of capsular laxity at birth, rather than to any of the other abnormalities associated with congenital dislocation.

9. *Effects of Passive Extension of the Newborn Hip Joint*

During the entire intrauterine period of development of the human hip joint, the joint is maintained in some degree of flexion in every single fetus (Fig. 14). As a result, at the time of birth every infant exhibits a marked hip flexion deformity which is associated with a tight iliopsoas muscle (Fig. 15), a deformity that, as Somerville[30, 31] has suggested, renders the hip vulnerable to passive extension.

Anatomical studies were carried out in the postmortem room of The Hospital for Sick Children on the left hip joint of a 1-day-old female infant who had died as a result of multiple congenital cardiac abnormalities. With the capsule of the joint still intact, passive extension of the hip produced only a mild anterolateral sub-

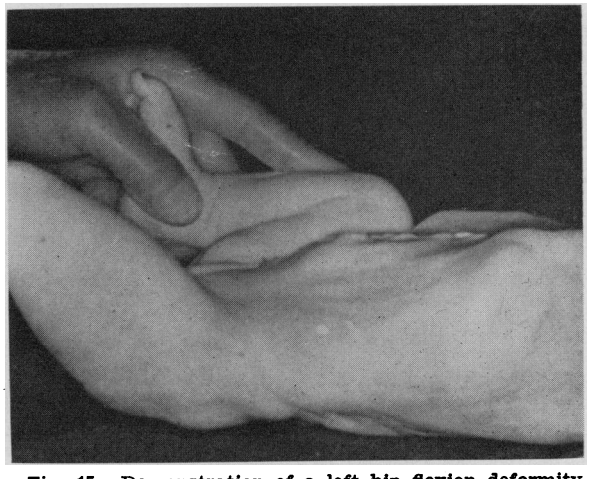

Fig. 15.—Demonstration of a left hip flexion deformity in a 1-day-old infant cadaver, who died as a result of multiple congenital cardiac abnormalities. At birth, the iliopsoas muscle is always tight.

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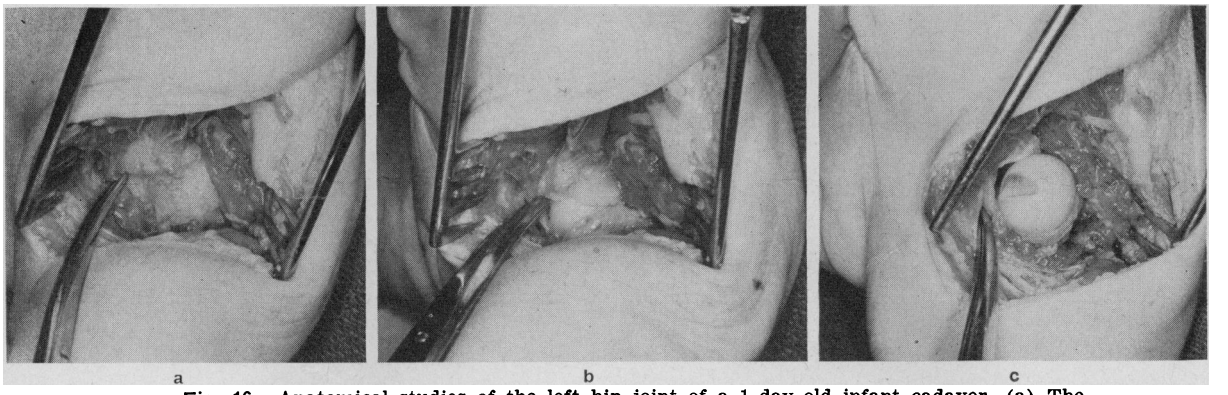

Fig. 16.—Anatomical studies of the left hip joint of a 1-day-old infant cadaver. (a) The anterior aspect of the intact capsule can be seen to bulge owing to a mild anterolateral subluxation when the hip is passively extended. (b) The anterior aspect of the capsule has been incised transversely; with the hip in flexion and abduction the femoral head remains well contained by the acetabulum. (c) Passive extension and adduction of the dissected hip has produced a complete anterolateral dislocation.

luxation (Fig. 16a). The anterior part of the capsule and the ligamentum teres were then incised to remove their normal restraint and thereby to stimulate capsular and ligamentous laxity. With the hip in flexion and abduction, the femoral head remained well contained by the acetabulum (Fig. 16b). However, passive extension and adduction of the hip produced a complete anterolateral dislocation (Fig. 16c).

Suspension of the same infant cadaver by the ankles extended the hips and produced a complete anterolateral dislocation of the previously dissected left hip (Fig. 17).

The same infant cadaver was then swaddled in a genuine Indian cradleboard (tikonagan). Radiographs of the hips taken through the cradleboard revealed complete anterolateral dislocation of the previously dissected left hip (Fig. 18).

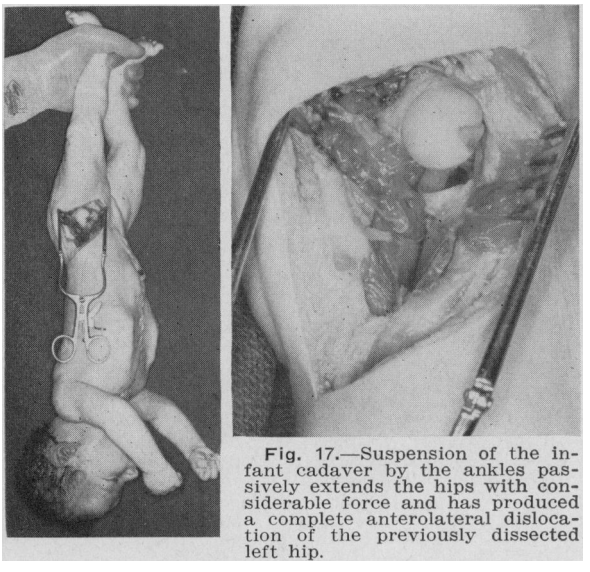

Fig. 17.—Suspension of the infant cadaver by the ankles passively extends the hips with considerable force and has produced a complete anterolateral dislocation of the previously dissected left hip.

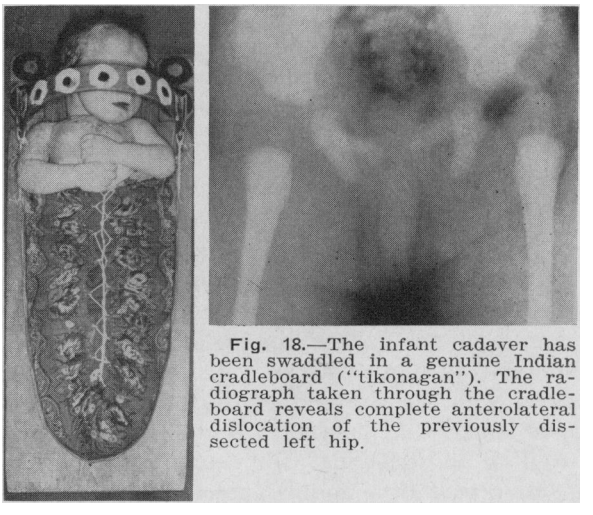

Fig. 18.—The infant cadaver has been swaddled in a genuine Indian cradleboard ("tikonagan"). The radiograph taken through the cradleboard reveals complete anterolateral dislocation of the previously dissected left hip.

As a result of these anatomical investigations it would seem reasonable to assume that in the presence of a congenitally lax hip joint capsule at birth, sudden, passive extension of the previously flexed hip joint by suspending the newborn infant by the feet may well lead to the initial dislocation (Fig. 19). Furthermore, the habit of wrapping a newborn infant tightly with the hips in extension and adduction, in the presence of a lax capsule (and therefore a dislocatable hip), would seem to produce a permanent dislocation of a previously only dislocatable hip (Fig. 20). Both of these practices are probably harmful and should be abandoned.

HYPOTHESIS OF THE SEQUENCE OF EVENTS

On the basis of a consideration of the known facts concerning the various aspects of congenital dislocation of the hip, as well as of an interpretation of these facts, it would seem reasonable to propose a hypothesis of the sequence of events

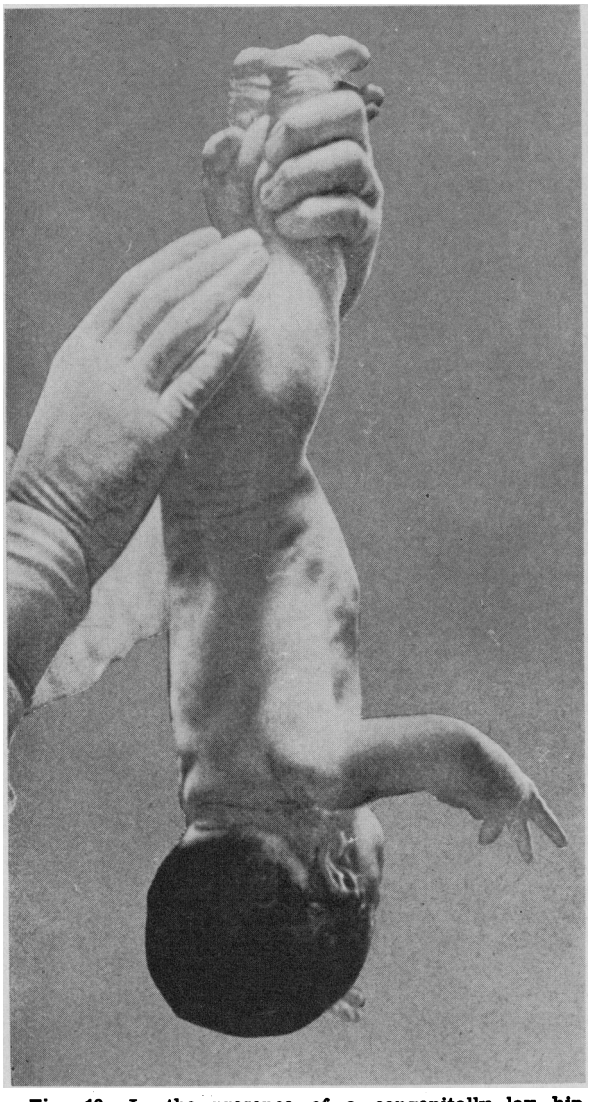

Fig. 19.—In the presence of a congenitally lax hip joint capsule at birth, sudden passive extension of the previously flexed hip joint by suspending the newborn infant by the feet may well lead to the initial dislocation.

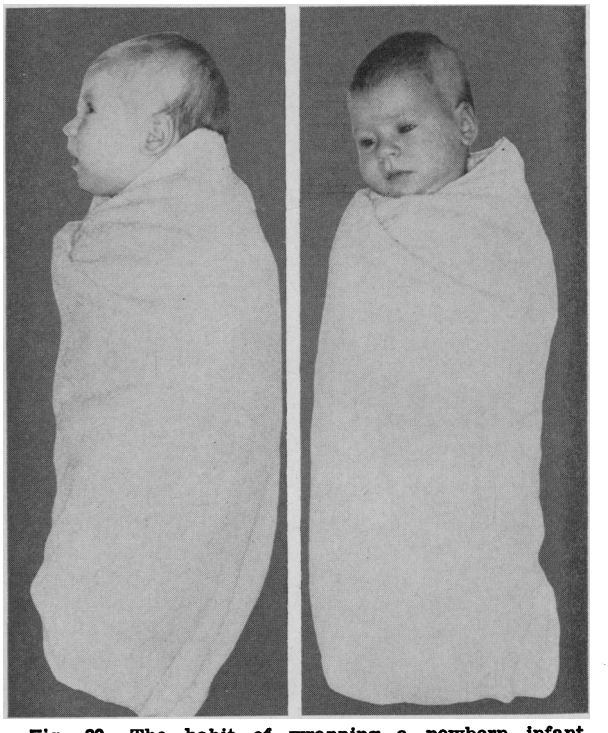

Fig. 20.—The habit of wrapping a newborn infant tightly with the hips in extension and adduction, in the presence of a congenitally lax capsule (and therefore a dislocatable hip) would seem to produce a permanent dislocation of a hip that was previously only unstable.

in the pathogenesis of this important clinical entity. While some of the factors in the proposed sequence of events are obviously beyond medical or surgical control, others can be not only controlled but also prevented (Fig. 21).

PREVENTIVE ASPECTS

Early diagnosis and early expert treatment will continue to be extremely important in the clinical condition of congenital dislocation of the hip. However, the possible prevention of the initial dislocation, or at least of a persistent dislocation, may become of even greater importance. It is quite possible that if infants' hip joints were never suddenly passively extended—either at birth or shortly after birth—and if infants' hips were never maintained in extension and adduction for long periods during the early months of postnatal life, the initial dislocation in congenitally unstable or dislocatable hips might never occur. Furthermore, if the hips of all infants were protected by maintaining them in mild flexion and mild abduction during this period, it is possible that even if the congenitally dislocatable hip did in fact dislocate shortly after birth, it would not remain in the dislocated position and therefore would not become a persistent dislocation.

Early diagnosis and early treatment of congenital dislocation of the hip are now possible. The next great challenge is the prevention of this serious and disabling condition.

Summary The etiology of congenital dislocation of the hip has been studied by an assessment and interpretation of the known facts in relation to embryology, anatomy, incidence, physical findings and pathological anatomy. Clinical investigations in young animals have led the author to the following conclusions: (1) the basic abnormality at birth is congenital laxity of the hip joint capsule and at this stage the hip is unstable, or dislocatable, rather than permanently dislocated; (2) dysplasia of the acetabulum, femoral anteversion, elongation of the capsule and contracture of the hip muscles are all the result, rather than the cause, of the initial dislocation; (3) passive extension of the hip joint

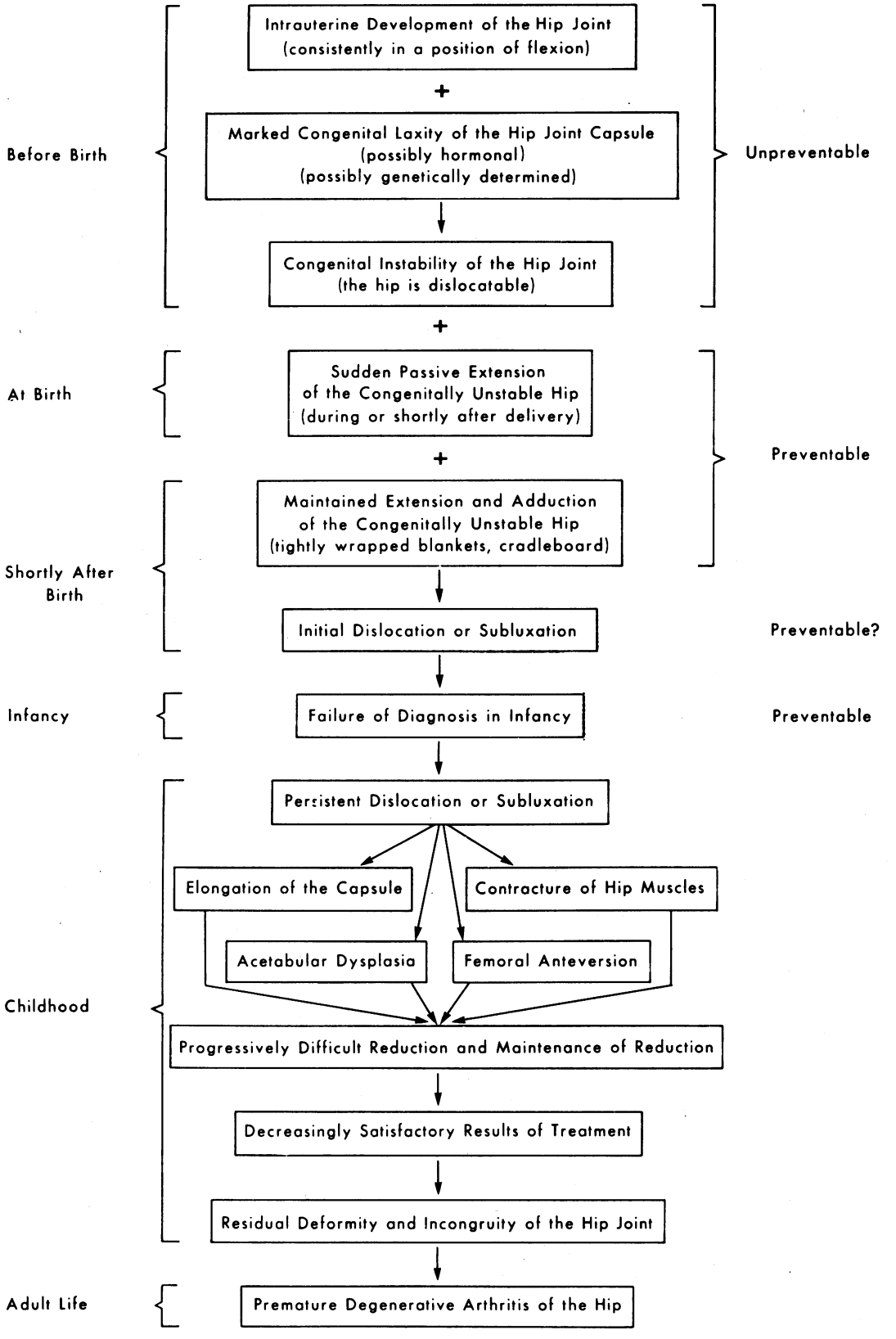

Fig. 21.—Hypothesis of the sequence of events in the etiology and pathogenesis of congenital dislocation of the hip.

in the infant cadaver after division of the capsule causes the hip to dislocate anterolaterally; (4) maintained extension of the human infant's hip by various means of swaddling has a deleterious effect on a congenitally lax hip joint.

A hypothesis has been developed concerning the sequence of events in the pathogenesis of congenital dislocation of the hip. Two important events in the sequence are preventable, namely passive extension of the hip at birth and maintained extension of the hip by any type of swaddling during infancy. It is believed that if these two practices could be avoided it might be possible to prevent persistent dislocation of congenitally unstable hips.

Résumé L'étiologie de la luxation congénitale de la hanche a été étudiée en évaluant et en interprétant les faits connus aux points de vue embryologie, anatomie, fréquence, constatations somatiques et anatomie pathologique. Les études cliniques chez l'enfant et les expériences sur l'animal ont amené l'auteur à formuler les conclusions suivantes: (1) à la naissance, l'anomalie principale est une laxité congénitale de la capsule articulaire de la hanche et, à ce stade, la hanche est instable, sujette à se luxer plutôt que luxée en permanence; (2) une dysplasie de la cavité cotyloïde, une antéversion fémorale, une élongation de la capsule et la contracture des muscles de la hanche sont tout autant d'éléments qui sont le résultat, et non pas la cause, de la luxation originelle; (3) une extension passive de l'articulation de la hanche sur un cadavre d'enfant après division de la capsule, a provoqué la luxation antérolatérale de la hanche; (4) l'emploi de divers bandages pour maintenir en extension la hanche de l'enfant exerce un effet défavorable sur l'articulation de la hanche qui est entachée d'une laxité congénitale.

L'auteur a formulé une hypothèse concernant la suite des événements qui surviennent pour expliquer la pathogénie de la luxation congénitale de la hanche. Il croit possible de prévenir deux de ces principaux événements: il incrimine l'extension passive de la hanche à la naissance et son maintien dans cette position par un type quelconque de bandage pendant la première enfance. Il estime qu'en évitant de recourir à ces deux pratiques, il peut être possible de prévenir la luxation persistante d'une hanche dont l'instabilité a une origine congénitale.

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