AVASCULAR NECROSIS OF THE FEMORAL HEAD AS A COMPLICATION OF TREATMENT FOR CONGENITAL DISLOCATION OF THE HIP IN YOUNG CHILDREN: A CLINICAL AND EXPERIMENTAL INVESTIGATION*


Avascular necrosis of the femoral head following reduction of a congenital dislocation of the hip is a serious complication that militates against a satisfactory long-term result. When the necrosis involves the entire head, the resultant deformity leads inevitably to impaired hip function (Fig. 1). Indeed, this complication is one of the commonest causes of unsatisfactory results of closed treatment for congenital dislocation of the hip in young children (from birth to 18 months of age). The purpose of this paper is to report clinical and experimental investigations concerning this form of avascular necrosis of the femoral head, with particular reference to its pathogenesis, prognosis and prevention.

THE CLINICAL PROBLEM

A review of the relevant literature reveals considerable confusion concerning the incidence, pathogenesis and prognosis of avascular necrosis of the femoral head following closed treatment of congenital dislocation of the hip in young children. Since true avascular necrosis of a congenitally dislocated head does not occur except after treatment, this condition must be considered an iatrogenic complication of treatment.

Incidence

The reported incidence of this complication varies from 0 to 73%: Cregan and

Fig. 1.—Radiograph of the hip joints of a 7-year-old girl who had been treated for bilateral congenital dislocation of the hips at the age of 6 months. Treatment had consisted of closed reduction without previous traction and without adductor tenotomies; the child's hips had been immobilized in the "frog" position for a total of one year in a series of plaster of paris hip spica casts. Note the severe deformities of the femoral heads and necks secondary to total avascular necrosis. Note also that the acetabula have not developed well. This serious complication of treatment will lead to severe degenerative arthritis of both hip joints in early adult life.

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Schwartzman\textsuperscript{1} 0; Scott\textsuperscript{2} (frame reduction) 8%; Severin\textsuperscript{3} 9%; Colonna\textsuperscript{4} 10%; Ponseti and Frigerio\textsuperscript{5} 16%; Wilkinson and Carter\textsuperscript{6} 22%; MacKenzie, Seddon and Trevor\textsuperscript{7} 25%; Hilgenreiner\textsuperscript{8} 33%; Esteve\textsuperscript{9} (frame reduction) 38%; Massie\textsuperscript{10} 45%; Ponseti\textsuperscript{11} 46%; Bost et al.\textsuperscript{12} 52%; Esteve\textsuperscript{13} (manipulative reduction) 67%; Zanoli\textsuperscript{14} 73%. It is apparent from these figures that various forms of treatment have been used and that all clinicians have not used the same diagnostic criteria for true avascular necrosis.

The situation has been confused further by the many terms that have been used to describe this complication. Some of these are: aseptic necrosis, osteochondritis, osteochondrosis, traumatic epiphysitis, Perthes-like changes, cystic degeneration, fragmentation and “stress effects.”

The congenitally dislocated femoral head is most vulnerable to the complication of avascular necrosis during the first 18 months of life—a period during which the head is composed largely of preosseous cartilage; indeed, during the first six months of life when the femoral head is entirely cartilaginous, it is extremely vulnerable. Ironically, this is a period during which the results of closed treatment can and should be virtually perfect.

The complication of true avascular necrosis of the femoral head develops in approximately 30% of children under the age of 18 months, particularly those in whom continuous traction is not employed before reduction. Even more disturbing is the report of Allen that, of 20 newborn infants who had an adduction contracture of the hip and a presumptive diagnosis of dysplasia without dislocation, and who were treated in plaster casts, 14 developed avascular necrosis of the femoral head.\textsuperscript{15} This report reinforces Caffey’s warning concerning the potential danger of overtreating, and thereby damaging, the hip joints of infants who have an adduction contracture but no clinical or radiographic evidence of dislocation.\textsuperscript{16} During the treatment of a unilateral congenital dislocation, this iatrogenic complication may even develop in the contralateral normal hip, if it is immobilized with the abnormal hip.

**Prognosis**

There has been no general agreement concerning the significance, or prognosis, of this complication in relation to long-term results. Some authors emphasize the gravity of this type of avascular necrosis while others believe that it has relatively little bearing on the subsequent development of the hip. This divergence of opinion can be explained, in part, by the observation that, in some children, the avascular necrosis involves the entire femoral head; in some, only part of the head; and in others there are only temporary radiographic changes which in the past have been interpreted as avascular necrosis but which may represent a different process. In the last group, there is no residual deformity of the femoral head and neck; consequently, the prognosis for subsequent development of the hip joint is good. In the first two groups, however, there is always some residual deformity of the femoral head and neck, and the prognosis for the hip, which is directly related to the severity of the deformity, is relatively poor.

**Theories of Pathogenesis**

The various theories concerning the pathogenesis of this complication have been based, for the most part, on speculation rather than on scientific investigation. These theories include the following:

1. Trauma to the blood vessels of the femoral head during manipulative reduction of the dislocation (Platt\textsuperscript{17}).

2. Incongruity between the reduced femoral head and the acetabulum (Alviki\textsuperscript{18}).

3. Stretching of the extra-articular vessels leading to the hip due to extreme positions of immobilization (Nicholson, Kopell and Mattei\textsuperscript{19}).

4. Wringing out of the vessels lying on the femoral neck due to twisting of the fibrous capsule of the hip in the position of extreme internal rotation (Hollow\textsuperscript{20},\textsuperscript{21}).

5. Compression of the posterior superior vessels between the femoral neck and the acetabulum (Tucker\textsuperscript{22}).

6. Congenital abnormality of the blood supply to the femoral head as part of the
"congenital dysplasia" or "aplasia" (Hans-berg\textsuperscript{23} and Gill\textsuperscript{24}).

(7) Susceptibility to vascular damage on the basis of individual variation in the vascular supply to the femoral head (Massie\textsuperscript{10}).

The variety of these theories and the relative lack of precise investigations concerning the pathogenesis of this complication have stimulated us to conduct a clinical investigation in young children, and an experimental investigation in young pigs.

**Clinical Investigation**

A retrospective investigation, conducted at the Hospital for Sick Children in 1965, covered the 10-year period from 1952 to 1962. Children were studied who were under the age of 18 months at the time of initial closed treatment for congenital dislocation or subluxation of the hip. The investigation included 140 children; in 23 of these, the dislocation was bilateral and consequently the results of closed treatment of 163 hips could be assessed.

In each child we studied the radiographs of the hips before, during and after closed treatment. During the second period of five years (1957 to 1962), the method of treatment had been altered considerably; preliminary traction and adductor tenotomy were used much more frequently than during the first five-year period (1952 to 1957). Therefore, the results of treatment and the incidence of avascular necrosis of the femoral head were assessed for each of these two periods.

**Radiographic Changes in the Femoral Head Following Reduction**

In the present investigation, an analysis of the 163 hips showed that, following re-

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**Fig. 2.**—Temporary irregular ossification. (a) Congenital dislocation of the right hip at 3 months of age. (b) One year after closed reduction, irregular ossification of the right femoral head. (c) Fifteen months after reduction, the ossific nucleus is enlarging. (d) Two years after reduction, the ossific nucleus continues to enlarge. (e) Two and one-half years after reduction, ossification is becoming more regular. (f) and (g) Three and one-half years after reduction, ossification is virtually normal. There is no residual deformity of either the head or neck of the femur.
Fig. 3.—Avascular necrosis. (a) Bilateral congenital dislocation of the hips at 2 months of age. (b) Three months after closed reduction. (c) Four months after reduction. (d) Six months after reduction, changes in the femoral necks. (e) One year after reduction, ossific nuclei have failed to appear. (f) One and one-half years after reduction, an ossific nucleus has appeared in the left femoral head but not in the right. (g) Two years after reduction, fragmentation of the left ossific nucleus; the right ossific nucleus is just beginning to appear. (h) Three years after reduction, increased density of both femoral heads and bilateral coxa vara. (i) and (j) Four and one-half years after reduction, the femoral heads are still not completely reossified. Note the deformity of the femoral heads and necks, the coxa magna and the coxa vara.

duction, there were two main types of radiographic change in the femoral head. The first was temporary irregular ossification of the femoral head which was not interpreted as avascular necrosis and proved to be of little clinical significance.
The second was true avascular necrosis of the femoral head, which was either complete or incomplete and invariably led to permanent deformity of the femoral head and neck.

(1) Temporary Irregular Ossification

Before reduction of a congenitally dislocated hip, ossification of the femoral head is relatively delayed; this hypoplasia, or dysplasia, of the ossific nucleus is presumably secondary to a relative lack of functional stimulus for the dislocated femoral head. Following reduction, however, ossification of the femoral head is markedly accelerated and during the ensuing year or more, ossification may proceed in a spotty, irregular manner from more than one centre. Radiographically, this type of irregular ossification may even suggest “fragmentation”, but, since the ossific nucleus enlarges from the time of reduction, the femoral head must be viable. Furthermore, there is little increase in radiographic density, the femoral head does not become...
Fig. 5.—Avascular necrosis. (a) Bilateral congenital dislocation of the hips at 4 months of age. (b) Four months after closed reduction. (c) Six months after reduction, the right ossific nucleus has appeared but the left has not; the left femoral neck, however, is becoming broad and irregular. This indicates that the right femoral head is viable but the left has suffered the complication of avascular necrosis. (d) One year after reduction. (e) and (f) Two years after reduction, ossification is appearing in the left femoral head and the neck is becoming broader. (g) and (h) Three years after reduction, the left femoral head is reossifying; there is coxa magna and coxa vara.

significantly deformed, and at most there is a slight coxa magna several years later (Fig. 2). Thus, the prognosis is excellent.

For all of these reasons the phenomenon of irregular ossification should not be considered to represent avascular necrosis (as it apparently has been in the past) although it may represent some type of temporary vascular disturbance.

(2) Avascular Necrosis

When either part or all of the femoral head loses its blood supply and undergoes true avascular necrosis, a series of readily detectable and characteristic radiographic changes occur. These radiographic changes, which are quite different from those of irregular ossification, are considered to be diagnostic criteria of avascular necrosis.

Criteria for the diagnosis of avascular necrosis.—After an analysis of the series of radiographs of children who developed the complication of avascular necrosis following closed treatment for congenital dislocation
of the hip, the following criteria for its early diagnosis would seem to be valid: (1) Failure of appearance of the ossific nucleus of the femoral head during one year or longer after reduction (Fig. 3). (2) Failure of growth in an existing ossific nucleus during one year or longer after reduction (Fig. 4). (3) Broadening of the femoral neck during one year after reduction (Fig. 5). (4) Increased radiographic density of the femoral head followed by the radiographic appearance of fragmentation (Figs. 3 and 4). (5) Residual deformity of the femoral head and neck when reossification is complete. These deformities include coxa magna, coxa plana, coxa vara and a short broad femoral neck.

Extent of involvement of the femoral head.—In children who show total involvement of the femoral head, the residual deformities of the femoral head and neck are severe (Figs. 3 and 4). In those who have partial involvement of the femoral head, the residual deformities, although significant, are less severe (Fig. 5). The presence of avascular necrosis of the femoral head is also associated with inadequate development of the acetabulum after reduction and residual subluxation of the hip.

Results of the Clinical Investigation

(1) Period From 1952 to 1957

Of the 66 congenitally dislocated hips treated by closed reduction during this five-year period, 20 femoral heads (30%) revealed definite evidence of avascular necrosis. In 9 of these femoral heads, there was total involvement and in the other 11 there was partial involvement. Temporary irregular ossification of the femoral head was seen in an additional 10 femoral heads (15%).

(2) Period From 1957 to 1962

Of the 97 congenitally dislocated hips treated by closed reduction during this second five-year period, 15 femoral heads (15%) revealed definite evidence of avascular necrosis. In seven of these there was total involvement and in the other eight there was partial involvement. Temporary irregular ossification was seen in an additional six femoral heads (6%).

Fig. 6.—Child in a hip spica cast in the first Lorenz (frog) position: the hips are in 90° of flexion and 90° of abduction.

Significant Factors in the Incidence of Avascular Necrosis

The incidence of the complication of avascular necrosis of the femoral head in the present series of young children (from

Fig. 7.—Child in a hip spica cast in the second Lorenz (Lange) position: the hips are in full internal rotation and abduction.
Fig. 8.—Femoral head of a young pig. India ink was injected into the aorta and cleared by the Spalteholtz technique. The epiphyseal vessels enter the cartilaginous portion of the femoral head proximal to the epiphyseal plate. They course through the preossaeus cartilage model to supply the ossific nucleus as well as the surrounding unossified cartilage where they end in delicate sinusoidal expansions.

birth to 18 months of age) was found to be highest under certain circumstances which seem to be significant. These were as follows: (1) Complete dislocation (as opposed to a subluxation). (2) No traction before reduction. (3) No adductor tenotomy before reduction. (4) Rigid immobilization of the reduced hip in an extreme position—(a) first Lorenz (frog) position (Fig. 6), and (b) second Lorenz (Lange) position (Fig. 7).

During the second five-year period, when continuous traction and adductor tenotomy were practised more frequently before reduction, the incidence of this complication decreased from 30% to 15%. Although the Lange position had been abandoned during this second five-year period, the frog position was still in use.

Conclusions From the Clinical Investigation

On the basis of the present clinical investigation, we concluded that the combination of tight muscles about the hip (particularly the adductors) at the time of reduction and rigid immobilization of the hip in an extreme position (particularly abduction) tended to produce avascular necrosis of the femoral head. We further concluded that this combination might produce avascular necrosis because of increased pressure between the reduced femoral head and the acetabulum. Thus, the clinical investigation provided some clues concerning the pathogenesis of this complication, but no definite proof.

It was apparent that further evidence concerning pathogenesis could be obtained only from an experimental investigation in young animals.

Experimental Investigation

The seriousness of avascular necrosis of the femoral head as a complication of closed treatment of congenital dislocation
of the hip in young children, the widely divergent views concerning its pathogenesis, and the clues gained from the present clinical investigation stimulated us to conduct an experimental investigation in young pigs. The experimental investigation included a study of the normal blood supply to the femoral head, experimental production of avascular necrosis, its pathogenesis and its prevention.

Hypothesis and Purpose of the Experimental Investigation

The hypothesis we formulated to explain this complication is as follows: if the adductor muscles are tight when the hip is reduced, extreme abduction of the reduced hip greatly increases the pressure between the femoral head and the acetabulum. The continuous pressure thus exerted—and maintained—by rigid immobilization of the reduced hip in extreme abduction, could compress and thereby occlude the blood vessels as they course through the resilient and compressible cartilaginous portion of the femoral head. Prolonged occlusion of the vessels within the femoral head could lead to avascular necrosis of the preosseous cartilage as well as of any existing ossific nucleus.

The purpose of the experimental investigation was to test the validity of this hypothesis by determining the effects of compression of the immature femoral head on its blood supply.

Normal Blood Supply to the Immature Femoral Head

The normal blood supply to the femoral head in human infants has been well described by Trueta and Crock. In a previous experimental investigation using various intravascular injection techniques, Salter and Schatzker found that the blood supply to the femoral head in infant pigs was comparable to that in human infants, not only in relation to the distribution of vessels leading to the femoral head but also

Fig. 9.—Microscopic appearance of a sinusoidal expansion in the preosseous cartilage of the pig’s femoral head shown in Fig. 8. The central arteriole and surrounding venules are encased in preosseous cartilage which is resilient and compressible.

Fig. 10.—Three-week-old pig with the hips maintained in forced abduction comparable to the frog position by means of a plaster of paris hip spica cast.
in relation to their distribution within the head.

The epiphyseal vessels enter the head from its periphery proximal to the epiphyseal plate and course through the preossous cartilage model to supply the ossific nucleus and the surrounding unossified cartilage (Fig. 8). Within the preossous, or unossified, cartilage of the femoral head, the vessels end in delicate sinusoidal expansions with a central arteriole and surrounding venules (Fig. 9). In the pig, a small ossific nucleus is present in the femoral head at birth whereas, in the human, this nucleus does not normally appear until approximately 6 months of age. Thus, at birth, the pig’s femoral head is developmentally comparable to the human femoral head at approximately 1 year of age. In both the pig and the human, the preossous cartilage containing the epiphyseal vessels is resilient and compressible, rather like India rubber.

The Experimental Design

In an attempt to simulate the clinical situation of young children being treated for congenital dislocation of the hip, the experiment was designed to produce an adduction contracture of the hips in the infant pig after which the hips were immobilized in plaster casts in extreme abduction—a position comparable to the frog position.

Experimental Methods

(1) Production of an Adduction Contracture

The hip joints of infant pigs (within a few days of birth) were maintained in the cross-legged (adducted) position by means
of elastic tape for a period of three weeks. During this time the infant pigs were able to get about by dragging their adducted legs behind them. At the end of three weeks, the adductor muscles were tight and there was a marked adduction contracture of both hips.

(2) Immobilization in Plaster Casts

Plaster of paris hip spica casts were applied with the infant pigs under general anesthesia. The hip joints were forced into a position of abduction comparable to the frog position (Fig. 10).

(3) Injection Technique for Angiography

The angiograms were performed on living pigs under general anesthesia. Micropaque (50% weight by volume) was injected into the aorta at open operation using a Beckman solution metering pump—model 746, which delivered 20 cm. of micropaque per minute at a pressure of 110 mm. Hg (the normal blood pressure of the pig). The perfusion of the lower limbs was continued until micropaque was seen returning through an opening in the inferior vena cava. The animals were sacrificed at the end of the perfusion; the distal one-half of the animal was fixed in 10% formalin for 10 days after which the soft tissues were dissected from the pelvis and femora. The femoral heads were then cut into three relatively equal parts in the coronal plane and radiographed.

In some animals the injection material was India ink; the technique of injection was similar to that described above and the Spalteholz technique was used to clear the specimens.
Fig. 13.—Micropaque angiogram of the femoral head of a 5-week-old pig whose hips had been maintained in adduction for three weeks after which the resultant adduction contracture was overcome by a bilateral adductor myotomy. The hips had then been maintained in abduction for two weeks but without force. Note the normal vascular pattern in the femoral head.

Series of Experiments and Results

(1) Series I. (Adduction Alone)

In 20 newborn pigs, an adduction contracture was produced over a period of three weeks. At the end of this time, angiography was performed after which the animals were sacrificed.

Results.—There was no disturbance of the vascular pattern in any of the femoral heads of these 20 pigs (Fig. 11).

(2) Series II. (Abduction Alone)

In 20 pigs, 3 weeks of age, with no previous immobilization and hence no adduction contracture, the hips were placed in 80° of abduction under general anesthesia and immobilized in a hip spica cast in the frog position. Immobilization was continued for two weeks after which angiography was performed and the animals were sacrificed.

Results.—There was some degree of disturbance of the vascular pattern in 30 of the 40 femoral heads; the disturbance, however, was slight.

(3) Series III. (Adduction Followed by Abduction)

In 20 newborn pigs, an adduction contracture was produced over a three-week period as in Series I. At the end of this time, the hips of these pigs exhibited marked limitation of abduction. The hips of each of these pigs were then placed in abduction under general anesthesia. A force of approximately 10 lb. was required to produce 70° of abduction. The hips were immobilized in the frog position in 70° of abduction for two weeks after which angiography was performed and the animals sacrificed.

Results.—There was a severe disturbance of the vascular pattern in all 40 of the femoral heads (Fig. 12). The vascular block was in the vessels as they course through the preosseous cartilage within the head.
Fig. 14.—Radiograph of a section of the femoral head of a 15-week-old pig whose hips had been maintained in adduction for three weeks, maintained in forced abduction for two weeks and then allowed free for 10 weeks. Note the appearance of a "head-within-a-head". The size of the smaller head is exactly the same as that of the ossified portion of the femoral head at the time the pig's hips had been put in forced abduction.

(4) Series IV. (Adduction Followed by Adductor Myotomy Before Abduction)

In 20 newborn pigs, an adduction contracture was produced over a period of three weeks as in Series I and III. At the end of this time, the hips of these pigs exhibited marked limitation of abduction. We then did a bilateral open adductor myotomy under general anesthesia to release the adduction contracture. The hips of each of these pigs were then placed in abduction. Almost no force (other than gravity) was required to produce 70° of abduction. The hips were immobilized in the frog position in 70° of abduction for two weeks after which angiography was performed and the animals sacrificed.

Results.—There was no disturbance of the vascular pattern in any of the femoral heads of these 20 pigs (Fig. 13).

(5) Series V. (Survival Experiment—Adduction Followed by Abduction)

This series was identical to Series III except that at the end of the two periods of immobilization the animals were allowed to survive and run free. These animals were subsequently sacrificed at periods varying from 3 to 10 weeks later. The femoral heads were radiographed without previous angiography.

Results.—Radiographs of the femoral heads of these pigs revealed the phenomenon of a "head-within-a-head" (Fig. 14). The size of the smaller head was exactly the same size as that of the ossified portion of the femoral head at the time the hips had been put in extreme abduction. Thus, the size of the smaller head serves as a time-marker for the initial vascular occlusion. During the first three weeks, the osseous nucleus did not increase in size but the cartilage model did; subsequently the preosseus cartilage ossified. These changes are best shown schematically (Fig. 15).

Conclusions From the Experimental Investigation
From Series IV, we concluded that the complication of vascular occlusion within the femoral head could be prevented by completely releasing the tight adductor muscles (adduction contracture) before maintaining the hips in abduction. From Series V, we concluded that the first indication of avascular necrosis of the femoral head after the hip was immobilized in extreme abduction was the failure of the osseous nucleus to increase in size over a five-week period (two weeks in the cast and three weeks out of the cast). It was also concluded that the size of the smaller head in the “head-within-a-head” phenomenon serves as a reliable time-marker for the initial vascular occlusion.

CORRELATION BETWEEN THE CLINICAL AND EXPERIMENTAL INVESTIGATIONS

The results of the experimental investigation correlate well with those of the clinical investigation. Both investigations emphasize the importance of the combination of tight adductor muscles and immobilization of the immature hip in a position of extreme abduction in the pathogenesis of this type of avascular necrosis of the femoral head. Furthermore, both investigations indicate one key to prevention of this complication, namely, avoidance of continuous compression of the cartilage model between the immature femoral head and the acetabulum.

In both investigations, failure of the osseous nucleus to increase in size was the first indication of avascular necrosis. The different periods of such growth failure—five weeks in the pig and one year in children—reflect the fact that, in their respective growing periods, five weeks in the pig is roughly comparable to one year in the human. The radiographic phenomenon of the “head-within-a-head” seen in the experimental investigation has also been observed in children; we presume that it has the same significance in the human as in the pig (Fig. 16).

PREVENTION OF AVASCULAR NECROSIS OF THE FEMORAL HEAD

The key to prevention of the complication of avascular necrosis of the femoral
head during closed treatment of congenital dislocation of the hip seems to be avoidance of continuous compression between the immature femoral head and the acetabulum. Therefore, treatment of young children with congenital dislocation of the hip should include the following measures: (1) Continuous traction before closed reduction (Fig. 17). (2) Subcutaneous adductor tenotomy at the time of reduction. (3) Immobilization of the reduced hip in a position that does not compress the femoral head against the acetabulum (Figs. 18 and 19).

We concluded that the frog position—90° of abduction, which is comfortable for the frog and which should probably be reserved for the frog—is potentially harmful.
ing application of the hip spica cast should be that of gravity.

The validity of these conclusions is emphasized by the clinical observation that the more frequent use of continuous traction and subcutaneous adductor tenotomy reduced the incidence of avascular necrosis from 30% in the first five-year period to 15% in the second five-year period. Furthermore, in an additional clinical investigation covering the period since the "human" position was adopted (1962 to 1967), the incidence of avascular necrosis declined further—to approximately 5%—and the vascular changes in these hips have been relatively mild.

**Summary and Conclusions**

Avascular necrosis of the femoral head, either partial or complete, is a serious iatrogenic complication of closed treatment of congenital dislocation of the hip in young children. This complication leads to permanent deformities of the head and neck of the femur, and to inadequate development of the acetabulum, and consequently has a bad prognosis. Criteria for the diagnosis of this type of avascular necrosis have been outlined. The condition of temporary irregular ossification of the femoral head, often confused with avascular necrosis, does not lead to permanent deformity of the head and neck of the femur and consequently has a good prognosis.

From the present clinical investigation of 163 hips in young children (from birth to 18 months), the significant factors in the incidence of avascular necrosis were failure to use traction before reduction, failure to perform adductor tenotomy at the time of reduction and finally, immobilization of the reduced hip in an extreme position—particularly extreme abduction.

From the present experimental investigation in 200 hips of 100 young pigs, we have concluded that the most significant cause of this type of avascular necrosis of the femoral head is continuous compression of the cartilage model between the femoral head and the acetabulum. This compression, which results from the combination of tight adductor muscles and immobilization of the immature hip in extreme abduction, occludes the vessels with-
in the preosseous cartilage of the immature femoral head and leads to avascular necrosis.

The iatrogenic complication of avascular necrosis of the femoral head following closed treatment of congenital dislocation of the hip in young children can, to a large extent, be prevented by continuous traction before reduction, subcutaneous adductor tenotomy at the time of reduction and immobilization of the reduced hip in the "human" position of marked flexion and only slight abduction—a position that does not compress the femoral head against the acetabulum.

References


Résument

La nécrose avasculaire de la tête fémorale est une complication majeure de la réduction féminée de la luxation congénitale de la hanche chez le jeune enfant. L'auteur rappelle les critères sur lesquels il faut baser le diagnostic de cette né-
HOMOTRANSPLANTATION OF CANINE LIVER: THE "REVERSE" WELCH TECHNIQUE

During recent years, it has been established that auxiliary liver homografts which are placed in ectopic locations can undergo involution by other means than rejection. The prototype of this operation was described by Welch, and Goodrich et al. They placed the extra liver in the right paravertebral gutter. The hepatic arterial supply was derived from the aorta or iliac artery. Venous inflow was reconstituted by anastomosing the distal iliac vein or inferior vena cava to the homograft portal vein. It has been shown that the total blood flow to such Welch homografts is equal to that in the dog's own undisturbed liver. Although the transplanted organs are protected from rejection by host immunosuppression, they atrophy in a way not seen in orthotopic homografts that are normally vascularized in the liver fossa.

In the present study, a modification of auxiliary transplantation was designed to test further the interrelationship between host and graft livers in immunosuppressed dogs. The procedure used for these experiments might be considered a "reverse" Welch operation. In Welch's preparation, the autologous liver had a normal blood supply while the homograft was vascularized in the same way as with a portacaval transposition. In our animals the host liver was subjected to the transposition and portal flow was directed through the homograft.

With this change, extreme homograft atrophy was avoided. In three of the seven animals in which all the splanchnic flow seemed by angiography to pass to the homograft, there was little or no weight loss of the transplant; in the other four, the degree of atrophy was moderate, compared to that seen in Welch-type auxiliary livers. The improvement was evidently not due to an imbalance in the volume of blood flow to the dual organs. It has been shown that the blood flow to the two livers after Welch's procedure is equivalent; the same finding pertained in the present study. Instead, the partial protection of the homografts was probably the result of perfusing them with splanchic venous blood according to the concept developed by Marchioro et al.

The fact that some homograft atrophy commonly occurred despite its perfusion with splanchic venous blood adds a further precautionary note about the wisdom of auxiliary hepatic transplantation to patients whose own livers possess significant residual function. In a competitive environment, primary exposure to splanchic blood flow is a physiologic advantage, but one that may not be sufficient if the homograft is subjected to the injury of severe but potentially reversible rejection. Under the latter circumstance, the only hope for long-term transplant function may be if the new organ is unopposed, as is the case with orthotopic homotransplantation.

Auxiliary liver homografts were revascularized in the lower abdomen of dogs with an arterial blood supply from the aorta and with a portal venous supply that was derived from the host splanchnic bed. The animals' own livers were deprived of splanchic flow but this was replaced with systemic venous blood. Total hepatic blood flow in the co-existing livers was approximately equal. Under these conditions the homograft atrophy often seen after auxiliary transplantation was reduced but not prevented. The findings have been discussed as they relate to the mechanism of homograft atrophy and to the applicability of such techniques for the treatment of human disease.—Daloze, P. M. et al.: Auxiliary homotransplantation of the canine liver with the use of a "reverse" Welch technique, Surgery, 64: 934, 1968.