

Acetabular Dysplasia and Osteoarthritis Developed by an Eversion of the Acetabular Labrum

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There is much confusion in orthopedic literature regarding the nature and significance of the so-called acetabular labrum in congenital dislocation of the hip.

This experiment describes an animal model in which the relationship between eversion of the acetabular labrum and acetabular dysplasia with degeneration of the articular cartilage was studied.

The acetabulum gradually become shallower and more vertically oriented and the femoral head gradually subluxed, but never dislocated. The radiographic appearance of the hip dysplasia was very similar to that seen in human beings. The animals remained asymptomatic without clinically visible signs of hip abnormality throughout the study.

It was clearly demonstrated that there was substantial amounts of degenerative change in the articular cartilages of the subluxed hip with no evidence of degenerative change radiologically.

It could be concluded that excision of the acetabular labrum should be avoided during open reduction of congenital dislocation of the hip.

Key Words: Acetabular labrum, Acetabular dysplasia, Osteoarthritis.

The human acetabular labrum, a fibrocartilaginous structure very similar to the meniscus within the knee joint, is at the margin of the acetabular cartilage and the joint capsule and is inserted several millimeters above the rim of the acetabulum into the fibrous tissue covering the outer surface of the acetabular cartilage. It has a triangular cross section with a free atypical border just like meniscus and is elastic in consistency. It is incomplete inferiorly, but the two ends are bound together by the

transverse ligament of the acetabular notch (Gruebell Lee, 1983).

The acetabular labrum has two physiologic functions: (1) It enlarges the acetabular socket and contributes to the stability of the femoral head within the acetabular socket, and (2) by being in direct continuity with the hyaline cartilage of the acetabulum, it participates in the growth and development of the acetabular roof (Tachdjian Mihrano, 1982).

If the femoral head does dislocate, the free border of the labrum has a disadvantage.

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Manipulative reduction, whether occurring under anesthesia or gradually by traction, may result in the infolding of the labrum into the joint. This may include a large portion of the lip or merely a segment, usually in the posterosuperior quadrant. It has been believed that the infolded labrum constitutes a powerful obstruction to the reduction of the hip because the head, although within its bony socket, is not contained by the soft tissues that complete that socket.

There is much confusion in orthopedic literature regarding the nature and significance of the so-called acetabular labrum in congenital dislocation of the hip (Dunn, 1969; Fellonder et al., 1970; Leveuf, 1946; Lust et al., 1972; Ortolani, 1948; Putti, 1937; Scapinelli, 1972; Somerville, 1953). Ponseti (1953) has shown that complete interposition of the acetabular labrum is rare and is only seen in very severe hip dysplasia such as teratologic dislocation. In fact, a defect interpreted as the inverted acetabular labrum is actually caused by a bulge or thickening of the acetabular hyaline cartilage. In the usual degree of hip dysplasia, the fibrocartilaginous labrum is atrophic and adherent to the inner aspect of the hip capsule; the labrum is not inverted and does not participate in the formation of the acetabular ridge.

Sommerville (1953) and Scott (1965) insisted on the necessity of excision of the limbus when it was inverted sufficiently to cause an obstruction. After the initial frame reduction they recommended routine arthrography of the hip. If the limbus was small or if it did not prevent easy complete reduction, they left the limbus in place; but in all other cases when the inverted limbus appeared to cause obstruction, they advocated limbusectomy. They pointed out that the inverted limbus was a substantial fibrocartilaginous structure that stood up to the pressure of the femoral head. The pressure of the limbus was on the soft car-

tilaginous acetabular roof and produced a deficient acetabulum. An inverted limbus diminished the size of the acetabulum, and any attempt at forced reduction would cause considerable compression of the femoral head and aseptic necrosis. Mitchele (1970) supported the view of Sommerville (1953). Salter (1974) inspected the acetabular cavity and removed all obstacles except the acetabular labrum that prevented concentric seating of the femoral head. Severin (1941) demonstrated by serial arthrography that the femoral head could be progressively seated into the acetabulum. The soft tissue obstacles flattened and allowed the femoral head to be seated into the acetabulum. Petit (1962) concluded that open reduction of the hip, limbusectomy or simple operative excision of the labrum was unnecessary.

This project describes an animal model of acetabular dysplasia produced by an eversion of the acetabular labrum in which the relationship between the eversion of the acetabular labrum and acetabular dysplasia with degeneration of articular cartilage was studied.

MATERIAL AND METHODS

To standardize experimental conditions, the 20 mongrel puppies used were all 6 weeks of age at the time of the operation on the hip joint. They were anesthetized using intravenous nembutal and inhalation penthrane. The right hip joint was exposed through a small posterior incision (Fig. 1). The hip joint capsule was opened parallel and just distal to the acetabular labrum. The labrum was left intact but reflected superiorly, and attached to the periosteum along the ilium (Fig. 2). The hip joint capsule was then closed. Groups of dogs were housed in isolation units and were fed dry dog food mixed with canned meat and milk once a day and were given water *ad libitum*. The animals were

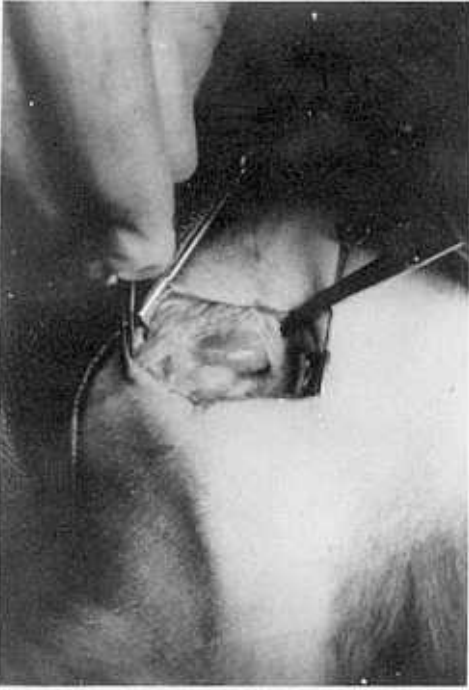


Fig. 1. The right hip joint was exposed through a small posterior incision. The hip joint capsule was opened parallel and just distal to the acetabular labrum. Acetabular margin and labrum are exposed but the femoral head is not visible in this figure.

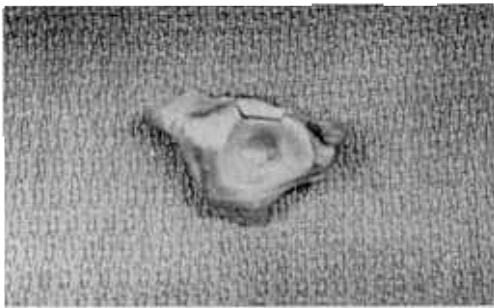


Fig. 2. The segment of the acetabular labrum in weight bearing portion is cut with the base attached to the periosteum along the ilium. This figure does not yet demonstrate the status of the eversion of the labrum.

obtained prior to surgery, and at biweekly intervals following surgery. According to the results of radiographic examination prior to surgery, the animals with natural hip dysplasia were excluded.

Two animals were sacrificed at monthly intervals for nine months following surgery.

The operated and normal hips were examined and compared roentgenographically, grossly and histologically. Counterposing areas on the femoral head and acetabulum were identified with the hip in a weight bearing position and blocks containing portions of the femoral head and acetabulum were removed from the counterposing areas. For the histological studies, the tissue was fixed in buffered 4 per cent paraformaldehyde, decalcified in 3 per cent formic acid and embedded in paraffin. Sections were cut 5um. thick and stained with hematoxylin and eosin and Safranin-O.

RESULTS

A deviation from normal acetabular development was roentgenographically visible on the operated hip 4 weeks following surgery. The acetabulum gradually became shallower and more vertically oriented and the femoral head gradually subluxed, but never dislocated (Fig. 3a and 3b). The radiographic appearance of the hip dysplasia was very similar to that seen in human beings. The animals remained asymptomatic without clinically visible signs of hip abnormality throughout the study.

Gross cartilage abnormalities were observed in all puppies as early as five months following surgery. The articular cartilage of the opposing femoral head and acetabulum became thinner with dulling of the cartilage and then there was flaking at the surface and fibrillation (Fig. 4a and b).

Inflammation was not seen in the dysplas-

allowed full cage activity as rapidly as tolerated.

Antero-posterior and frog lateral radiographic examination of the pelvis and hips were



Fig. 3a. Antero-posterior view of both hips taken 4 weeks following surgery revealed mild acetabular dysplasia with subluxation of the femoral head on the right side.



Fig. 3b. Same roentgenographic view in the same dog taken 9 months following surgery demonstrated a much more severe degree of acetabular dysplasia and subluxation of the femoral head. The joint cartilaginous space is well preserved without evidence of subchondral bone plate sclerosis.



Fig. 4a. Specimen of the acetabulum obtained 9 months following surgery shows thinning, dulling of cartilage and fibrillation.

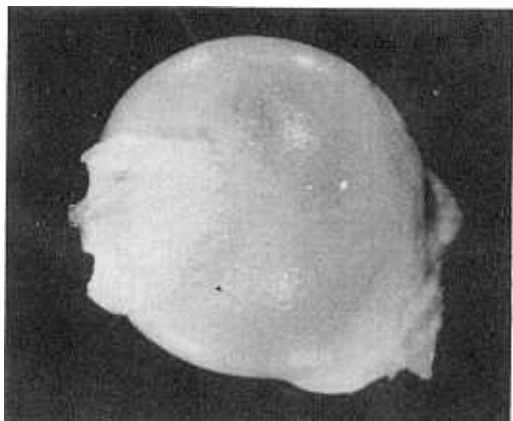


Fig. 4b. Femoral head specimen 9 months following surgery shows the same degree of degenerative change as the acetabular cartilage.

tic hip joints but each joint contained approximately 2 to 4 ml. of synovial fluid. The connecting round ligament of the femoral head was frayed and enlarged in some joints.

Histologically, the articular cartilage of the femoral heads and acetabuli on the operated side began to be distinguishable from that of the unoperated side by the fifth month. There was irregularity or loss of the surface layers of the articular cartilage, but there were no soft

clefts into the tangential zone (Fig. 5). Increased cellularity in the area of the weight-bearing cone occurred and persisted from the fifth month to the ninth month following surgery. Safranin-O staining disclosed a decrease in ground substance, which indicated depletion of proteoglycans, and this began to occur at the sixth and seventh months. A general correlation was found between the gross appearance of the lesion area and the severity of microscopic

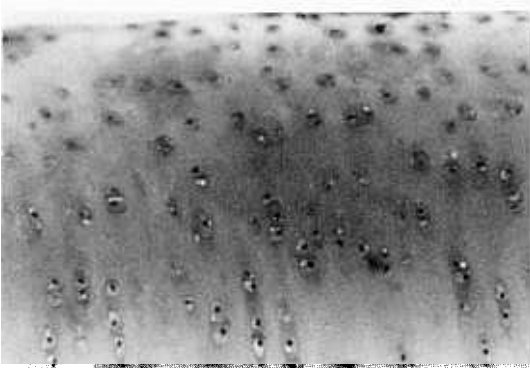


Fig. 5. Photomicrograph of the section of the acetabulum (H-E. staining with 250 x magnification).

Note that there is irregularity and loss of the surface layers of the articular cartilage, but there are no soft clefts into the tangential zone.



Fig. 7. Photomicrograph of the section of the dome of the femoral head (Safranin-O staining with 100 x magnification).

Note that there is loss of the surface layer of the articular cartilage and subchondral bone thickening.

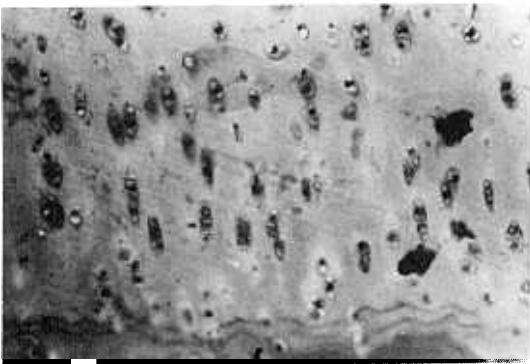


Fig. 6. Photomicrograph of a section of the weight bearing portion of acetabulum (Safranin-O staining with 250 x magnification).

Note that increased cellularity in the weight-bearing cone occurred. Also, it discloses a decrease in ground substance staining. A substantial degree of cloning of chondrocytes is noticed as well.

alterations. There was no evidence of increase in density of subchondral bone radiologically; but, histologically, there was thickening of the subchondral bone under the weight-bearing articular cartilage (Fig. 7).

DISCUSSION

Natural hip dysplasia in dogs is considered to be an hereditary disease common to many breeds (Henricson et al., 1966; Lust, 1970; Riser, 1969). This condition appears with about equal frequency in males and females, and its incidence has been reported to be high for several breeds of large dogs (Henricson et al., 1966; Lust, 1970; Riser, 1969). Joint laxity and degenerative changes of the hip joint connective tissues are commonly associated with the disease (Gustafsson et al., 1971; Henricson et al., 1966; Lust, 1970; Riser, 1969). In this experiment, the puppies with some abnormality of the hip joint were excluded to study the sequence of morphological and histological changes in the hip after eversion of the acetabular labrum.

Despite the tight closure of the joint capsule after eversion of the acetabular labrum, acetabular dysplasia with subluxation of the femoral head gradually developed following surgery. The acetabular cartilage complex was composed mostly of very cellular hyaline car-

tilage which also contained some cartilage canals. The lateral part of this cartilage complex, which is homologous with other epiphyseal cartilages (Harrison, 1958), lined the hip socket and was thicker toward its periphery where it was covered by perichondrium and by an overlying thick layer of fibrous tissue. Much appositional cartilage growth appeared to be taking place under the perichondrium. Interstitial growth within the triradiate part of the cartilage complex caused the hip socket to expand during growth. The concavity of the acetabulum developed in response to the presence of the spherical femoral head. The depth of the acetabulum increased during development as the result of interstitial growth in the acetabular cartilage, of appositional growth at the periphery of this cartilage, and of periosteal new-bone formation at the acetabular margin (Fairbank, 1930; Harrison, 1961; Ponseti, 1978).

Eversion or excision of the acetabular labrum might cause the damage of acetabular peripheral hyaline cartilage (which is epiphyseal cartilage), and impede development of the acetabulum. Subsequently, acetabular dysplasia should develop. Subluxation of the femoral head with stretching of the joint capsule occurred by the combination of the loss of the stabilizing buttress and growth arrest of the acetabular epiphysis in the everted labrum portion of the acetabulum.

Therefore, removal of the acetabular labrum during open reduction of congenital dislocation should be condemned. If the acetabular labrum is inverted to obstruct the reduction of the dislocated femoral head, it should be repositioned at least. The labrum can often be brought over the femoral head by inserting a blunt hook beneath it and everting its free border. If it is too tight, it can be split radially by a single incision, or even two; but it should not be excised since it plays a significant part in the later

development of the acetabulum.

The radiographic appearance of acetabular dysplasia in this study is very similar to that seen in human beings even if the mechanism of producing the deformity is different. In subluxation of the femoral head in the human, the periosteal bone formation at the margin of the acetabular roof is stunted by the medial pressure of the subluxated femoral head. After the subluxated femoral head is reduced, normal development of the acetabulum can occur by the restoration of normal growth once relief from the medial pressure occurs (Ponseti, 1978).

No frank dislocation of the femoral head was noticed in this animal model experiment due probably to an insufficient time period to develop the severe acetabular dysplasia which would dislocate the femoral head. Frank dislocation may develop if the experimental follow-up period is longer than nine months. Also, the animals may gradually become symptomatic because of deteriorating hip anatomy.

Many authors have attempted to estimate the incidence of osteoarthritis related to underlying acetabular dysplasia (Hart, 1952; Hass, 1951; Lloyd-Roberts, 1955; Muller et al., 1953; Wiberg, 1939). Their estimates range from 20% to 48%, but Wiberg's finding of 25% is the most commonly accepted figure (Wiberg, 1939). Wiberg (1939) stated that early or late secondary osteoarthritis develops in congenital hip dysplasia when acetabular coverage of the femoral head is less than optimal. Wedge (1953) emphasized that acetabular dysplasia and subluxation led much more frequently to frank osteoarthritis than to dislocated hips. Pauwels (Pauwels, 1963) has demonstrated the forces and the areas of weight-bearing on the acetabulum in a normal and in a subluxated hip. He has emphasized that when the hip is subluxated there is a marked increase in the forces which are concentrated on a small area of the acetabulum.

bulum. Pugh, Radin, and Rose (Pugh et al., 1974) have demonstrated that the response to such an increase in pressure per square unit of cartilage area in the human is sclerosis in the subchondral bone characterized by osteoid and woven bone formation. The sclerosis, in turn, renders the subchondral bone stiff and may be a factor in the associated degeneration of the overlying articular cartilage.

Degenerative lesions seen in hip joint cartilage of operated hips in this experiment are secondary manifestations caused by abnormal surface articulation resulting from subluxation of the hip joint. The subclinical degenerative change of articular cartilage may gradually progress to severe osteoarthritis.

The source of pain in a subluxated hip joint with no significant loss of articular cartilage as revealed by radiographic narrowing of the cartilaginous space is not clearly explained. It may come from the impingement of synovium in the localized portion between the subluxed femoral head and the acetabulum. Lust and associates (Lust et al., 1972 ab) demonstrated severe loss of glycosaminoglycans in the acetabular cartilage after spontaneous dislocation of the hip in young dogs. Very abnormal articular cartilage has been observed in dissections done some months after reduction. In this animal model, it was clearly demonstrated that substantial amounts of degenerative change in articular cartilage in the subluxated hip developed with no evidence of degenerative change radiologically. This is consistent with Ponseti's view (Ponseti, 1978) that defective articular cartilage in the acetabulum and in the femoral heads could cause pain.

This model of acetabular dysplasia appears to approximate, radiographically and morphologically, the equivalent human hip abnormality. This model is consistently reproducible. The surgical procedure is easy to perform and

atraumatic. Therefore, this model could be used for experimental osteoarthritis of the hip joint. Moreover, the reversibility of the acetabular dysplasia could be studied by simply correcting the surgical defect originally produced.

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