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The femoral head – dead or alive!

Guest Editorial

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Ischemia of the femoral head from hip joint hyperpressure tamponade has only recently progressed from speculation and experiments to become a solid clinical reality diagnosed by scintimetry. In this issue of *Acta Orthopaedica Scandinavica,* three papers deal with problems concerning vascular disturbance in the child's hip. In Lund, Wingstrand et al. (1985) have examined consecutive cases of transient synovitis. Four out of 25 patients had markedly defective isotope uptake in the proximal femoral epiphysis. At repeat scintimetry 6 weeks later, normalization of isotope uptake occurred in three patients, whereas in the fourth patient defective isotope uptake persisted in the proximal femoral epiphysis. This patient subsequently developed radiographic Legg-Calvé-Perthes' disease. In a second series 14 patients were examined with sonography, scintimetry and intracapsular pressure measurements. All patients had intracapsular effusion, as diagnosed sonographically, and highly elevated intracapsular pressure. In two patients there was reduced blood flow to the proximal femoral epiphysis in the acute phase. Following aspiration of joint fluid with subsequent reduction of intracapsular pressure, the isotope uptake in the epiphysis returned to normal.

Kallio & Ryöppi (1985) of Helsinki have measured the intracapsular pressure in 94 hips with transient synovitis, other forms of synovitis and Legg-Calvé-Perthes' disease. The pressure was only slightly elevated in the latter but in various types of synovitis it was well above systolic values.

Both groups of authors have demonstrated that intracapsular pressure in transient synovitis is dependent on the position of the hip. Thus, pressure was markedly decreased with the hip joint in flexion, whereas maximum intracapsular pressure was noted in the position of extension and internal rotation.

Even before the pioneer microangiographic studies by Trueta (1957), orthopaedic surgeons had suspected that intracapsular effusion or hemarthrosis may result in obliteration of the retinacular vessels and eventually cause infarction of the epiphysis, i.e. Legg-Calvé-Perthes' disease (Ferguson & Howorth 1934). The vascular anatomy of the proximal femoral epiphysis in childhood has been thoroughly clarified by Trueta (1957). Lauritzen (1974) and Chung (1976) have contributed valuable complementary studies to this field. Experimentally induced hip joint tamponade in puppies has been shown to produce diminished circulation or even infarction of the proximal femoral epiphysis (Kemp 1981, Launder et al. 1981). In adult animals, because of the collateral intraosseous blood supply, it is not possible to alter the state of circulation by tamponade.

The vascular supply to the proximal femoral epiphysis in the child is separated from that of the metaphysis by the growth plate, which constitutes an obstacle to collateral nutrition. The growth plate is situated intracapsularly, and therefore the epiphyseal or retinacular vessels have an extended and most likely vulnerable course on the surface of the femoral neck before entering the epiphysis. This is particularly true for the lateral retinacular vessels, the most important supply to the epiphysis, emanating from the medial circumflex artery. Additional vascular supply via vessels in the ligamentum capitis femoris is insignificant.

Following closure of the growth plate in adolescence, the former proximal femoral epiphysis gains access to the collateral circulation from the femoral neck. In adults this collateral supply can be cut off, for instance in cervical fracture. In this situation the femoral

head circulation relies solely on the intracapsular epiphyseal vessels, the capacity of which determines the viability of the femoral head.

The three papers presented in this issue constitute important steps forward in this field and give a substantial elucidation of transient synovitis of the hip, which obviously results in markedly increased intracapsular pressure, most likely compromising the vascular supply to the epiphysis. In spite of this, the risk of epiphyseal infarction seems relatively small. There are several possible reasons for this:

1. Individual anatomic variation in the arrangement of the vessels may make some individuals more prone to infarction of the epiphysis.
2. Flexion of the hip may intermittently reduce intracapsular pressure; this position is also the least painful. Unfortunately, however, transient synovitis has often been treated with traction in extended position of the hip.
3. The etiology of transient synovitis is poorly understood. Inflammatory cells in the synovial membrane may release metabolical factors that stimulate angiogenesis or fibroplasia and may contribute to circulatory effects.

Harrison & Burwell (1981) argued that the Trueta concept of an etiological connection between the vulnerability of the vascular supply to the proximal femoral epiphysis and the development of Legg-Calvé-Perthes' disease did not harmonize with a number of clinical facts:

1. Legg-Calvé-Perthes' disease also occurs before and after the age of maximum vascular vulnerability (3–8 years).
2. The initial stages of Legg-Calvé-Perthes' disease are often insidious, whereas intracapsular tamponade in transient synovitis of the hip might be expected to be very painful.
3. Hip synovitis of other origins, for instance rheumatoid arthritis, does not result in osteonecrosis.
4. The male dominance of the disease.

However, these arguments are not indisputable.

Ad 1. Considering individual anatomic variations, the numbers of hips in each group investigated by Trueta, Chung and Lauritzen were too few to permit definition of exact age limits.

Ad 2. The history and clinical picture in transient synovitis and Legg-Calvé-Perthes' disease are often identical. Thus Case 3 in the current article by Wingstrand et al. (1985) had a clinical history of transient synovitis with normal radiography, and 6 weeks later radiographic Legg-Calvé-Perthes' disease had developed.

Ad 3. Synovitis due to rheumatoid arthritis is always, even in early stages, characterized by substantial formation of new vessels. Even so, Legg-Calvé-Perthes'-like radiographic changes in these patients are not uncommon.

Vascular occlusion due to tamponade may also explain the defective isotope-uptake and/or radiographic necrosis observed in the epiphysis in cervical fracture in the child, in septic arthritis, and in hemarthrosis due to hemophilia (Ahlberg 1965, Murray 1982, Gelfand et al. 1983, Minikel et al. 1983).

In congenital dislocation of the hip, necrosis of the epiphysis is a known complication occurring also in the *non-affected* hip (Westin et al. 1975). This may be explained as iatrogenic, following treatment with immobilization in flexion-abduction, a position that experimentally causes occlusion and eventually necrosis, most likely due to capsular torque (Calvert et al. 1984).

After cervical fracture in the adult, the vascular supply to the femoral head may be dependent only on retinacular vessels as in childhood. Strömqvist et al. (1985) presented two patients with hip joint tamponade due to hemarthrosis following trauma. These patients had very high intracapsular pressures and scintimetrical evidence of avascularity which was reversed following aspiration, i.e. reduction of intracapsular pressure. In these patients there was no obvious fracture on conventional radiographs, whereas there was scintimetrical evidence of fracture on follow-up 2 weeks later. It is likely that the reversible decrease in blood flow to the femoral head was a result of occlusion of retinacular vessels due

to hemarthrosis, while intraosseous collateral circulation to the head was blocked by cervical infraction. The authors' suggestion of the connection between repeated hip traumata with hemarthrosis and the relatively common occurrence of osteonecrosis of the femoral head in alcoholics is intriguing.

We should, thus, consider further examination of patients with normal radiographs after acute trauma to the hips. The case reported by Bauer (1985) in this issue suggests that we have been ignorant or negligent in the past. CT scans or ultrasonography may visualize intracapsular bleeding, and radionuclide scintimetry may identify threatening death of the femoral head. Aspiration should then be performed promptly.

It is difficult to disregard the connection between the apparently vulnerable intracapsular arrangement of the retinacular vessels nourishing the proximal femoral epiphysis and the occurrence of epiphyseal infarction or necrosis following hip trauma. I am convinced that, before long, points which are still unclear will be elucidated with further scintimetric studies and possibly, in transient synovitis, with the aid of arthroscopy and synovial biopsies.

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