

Guest editorial

Legg-Calvé-Perthes' disease

With the advent of clinical radiography, it became possible for Legg, Calvé and Perthes to describe in 1910 the intriguing condition that was later called Legg-Calvé-Perthes' disease (LCPD). The historical fact that the disease was discovered, defined and described radiographically may be the primary reason why most research interest, until recently, has concentrated on phenomena disclosed by conventional radiography. The natural history of the disease and its radiographic course are very well described. Prognostically important features in the conventional radiograph have been defined but, in the individual child, are difficult to assess. In consequence, the treatment has been guided by radiography and aimed towards a spherical head of the femur and joint congruency. The assumption that the head of the femur should be protected from axial load during the course of healing has led to treatment regimens such as sling and crutches, wheel-chair and even long-term bedrest. None of these regimens has proven beneficial.

With the advent of scintimetry, it became possible to visualize epiphyseal avascularity and the process of revascularization and healing prior to the occurrence of the corresponding radiographic changes in LCPD. It was also possible with scintimetry to visualize transient epiphyseal ischemia in various pediatric hip conditions associated with increased intracapsular pressure, such as transient synovitis, septic arthritis, hemarthrosis due to hemophilia or trauma. Scintimetry, however, has not substantially contributed to the prognostic evaluation in LCPD.

The most important feature of the disease is not radiographic or scintimetric but clinical—i.e., pain and, because of the pain, limited joint motion. Little attention has so far been paid in the literature to the possible cause of this pain and to its clinical consequences. Three articles in this issue of *Acta Orthopaedica Scandinavica* deal with this new and obviously important aspect of clinical symptoms in LCPD.

In the paper by Eckerwall et al. (pp 575–80), 39 children with LCPD were examined with sonogra-

phy and intracapsular pressure recordings and aspiration demonstrated synovitis with significantly increased intracapsular pressure. The authors conclude that synovitis is common in LCPD, in particular in the early phase of the disease, and that it will probably be symptomatically and prognostically important because of the following chain of events: increase in intracapsular pressure, pain on motion, a decreased range of motion and, potentially, joint contracture.

That synovitis is common and clinically important in LCPD is confirmed arthroscopically in a paper by Suzuki et al. (pp 581–4). In 19 children, arthroscopy revealed proliferation of the synovium in the acetabular fossa and on the inner wall of the capsule. Microscopically, hyperplasia of the synovial lining cells was observed. Joint pain was relieved and motion improved after irrigation during arthroscopy, indicating the clinical relevance of synovitis.

In a third paper in this issue, Vanpaemel et al. (pp 585–8) report severe, destructive contralateral synovitis in 2 cases with LCPD, confirming a previous report of epiphyseal necrosis in juvenile chronic arthritis and suggesting an association with LCPD and a common etiology.

Experimentally-induced synovitis has been shown to result in edema of the cartilage, deterioration of the mechanical properties, cartilage hypermetabolism and, subsequently, cartilage hypertrophy. These observations, combined with the findings reported in the 3 papers in this issue, could well explain the cartilage hypertrophy with subsequent lateral subluxation, deformation of the head and joint incongruency seen in LCPD. Furthermore, synovitis causes an increase in intracapsular pressure which may, in some cases, intermittently compromise the blood supply to the epiphysis.

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