Sonography and intracapsular pressure in Perthes' disease

39 children examined 2–36 months after onset

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39 children with Legg-Calvé-Perthes Disease (LCPD) and with a radiographically unsatisfactory development were examined with sonography regarding synovitis and with intracapsular pressure recording and aspiration performed 15 (2–36) months after the onset of symptoms. The mean anterior sonographic capsular distension was 3.0 (1.0–7.0) mm greater than that of the contralateral, asymptomatic hip. The mean intracapsular pressure was 4.5 (0–11.5) kPa with the hips in extension and neutral rotation, 9.7 (1.3–27.3) kPa with the hips in extension and inward rotation and 0.9 (–0.8–4.7) kPa in 45° of flexion. We conclude that these children have synovitis as diagnosed sonographically. This synovitis is probably symptomatically and prognostically important in LCPD due to increased intracapsular pressure, with pain, a decreased range of motion and, potential joint contracture.

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In childhood, the blood supply to the proximal femoral epiphysis (PFE) is mainly routed intracapsularly with little or no contribution via vessels in the ligamentum teres (Trueta 1957, Theron 1980). Such a blood supply to the PFE is exposed to an increase in intracapsular pressure. This mechanism has been thought to cause epiphyseal ischemia due to synovitis with ensuing increase in intracapsular pressure (Kallio and Ryöppy 1985, Wingstrand et al. 1985b, Vegter 1987).

However, little attention has been paid in the literature to the clinical effects of synovitis and increased intracapsular pressure in LCPD. Synovitis is clinically common in the early phase of the disease (Terjesen 1993), with pain and a decreased range of hip motion and with a subsequent risk of hip joint contracture.

Our purpose was to diagnose synovitis sonographically and to correlate it to intracapsular pressure recordings and to the duration of symptoms in LCPD.

Patients and methods

39 consecutive children with LCPD were included in the study during the period 1984–1992 (Table 1). 35 were boys. 5 had bilateral disease with already healed, or healing, contralateral hips. The mean age at the time of the investigation was 7 (5–12) years. The mean duration from onset of symptoms was 15 (2–36) months. 30 hips were classified as Catterall group 4, 9 hips were classified as Catterall group 3 (Catterall 1971). 9 hips were in the condensation stage of the disease and 30 were in the fragmentation stage.

All the children had been examined on one or several occasions with conventional radiography and, in 29 cases, with MRI, demonstrating an unsatisfactory development with metaphyseal widening and anterolateral flattening. Thus, they were scheduled for further analysis with arthrography (31 hips) and/or surgery. 28 hips subsequently underwent surgery with femoral varus derotation osteotomy, 22 of which were in Catterall group 4 and 6 in Catterall group 3.

Sonography with real-time equipment (Diasonics DRF 12, 7.5 or 10 MHz sector or linear transducers), was performed according to Egund et al. (1986) from the ventral aspect of the hip in a plane along the axis of the neck of the femur with the patient supine and with the hips in a position of extension and neutral rotation (Figure 1). The anterior capsular distance, as defined by Egund et al. (1986), perpendic-

Table 1. Sonography and intracapsular pressure in 39 children with Legg-Calvé-Perthes' disease

A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
1	M	R (Bil)	6.8	4	f	12	0.3	0	1.7	2.7	9.7	9	6	•
2	M	R	4.9	4	f	8	0.3	3.9	1.1	3.7	10	9	6	3
3	M	R	9.8	4	f	15	0.3	7.1	0.8	1.1	13.6	10	7	3
4	M	L (Bil)	7	4	c	11	0.3	4	0	3.3	9.3	7	4	•
5	F	R (Bil)	6.2	4	f	12	0.1	1.6	0.9	1.1	1.9	6	6	•
6	M	R	9.5	4	f	21	0.1	11.1	4.7	5.3	17.1	7	6	1
7	M	L (Bil)	5.4	4	f	9	0.3	6	2.1	4.5	7.7	5	4	•
8	M	R	6.8	4	c	11	0.3	6.7	2.3	1.7	26.7	9	6	3
9	M	L	5.3	4	f	16	0.8	2.7	0.4	1.7	4.1	11	5	6
10	M	L	6.9	4	f	31	0.8	1.6	0	0.1	6.4	8	5	3
11	M	L	7.2	4	c	2	2.8	9.5	0	3.6	12	9	6	3
12	M	R	6.1	4	f	18	1.8	5.5	1.7	4.1	12.5	•	•	•
13	M	L	7.3	3	c	4	0.3	3.7	1.1	3.1	6.4	•	•	•
14	F	R	6.1	3	f	5	0.3	8.3	1.3	0.7	16.3	9.5	6	3.5
15	M	L	9.6	3	f	18	0.8	5.1	0	3.9	10.9	8	8	0
16	M	R	9.5	4	f	18	0.8	2	0.3	0.4	2.4	6	5	1
17	M	R	5.7	4	f	20	0.1	2.9	0	0	10.8	•	•	•
18	M	L	8.4	3	f	13	0	7.3	0.9	2.1	14.3	9	6	3
19	M	R	6.6	3	f	18	0	2.3	1.2	2.3	4.1	8	5	3
20	M	L	8.8	4	f	21	0	3.1	1.6	2.9	10.4	7.5	6	1.5
21	M	L	7.1	4	f	20	0	•	•	•	•	9	6	3
22	M	L	5	4	f	17	0	•	•	•	•	6	6	0
23	M	R (Bil)	6.6	4	f	36	0	0.3	0	0	1.3	5	5	•
24	M	R	6.6	4	f	27	0	1.9	0	0.7	4.8	7	5	2
25	M	L	7.4	3	c	8	0	1.6	1.5	2.3	5.7	8	5	3
26	M	L	10.8	4	c	11	0	4.8	3.3	6	7.2	10	5	5
27	M	R	9.1	4	f	10	0	3.7	0	0.9	8.1	12	6	6
28	M	L	6.9	4	f	30	0	3.1	0	1.3	5.1	7	5	2
29	F	L	10.5	3	c	8	0.3	5.3	1.3	3.9	9.3	9	7	2
30	M	R	7.2	4	f	18	0.8	6.1	0	0	11.1	10	7	3
31	M	R	5.8	4	f	21	0	4.7	1.1	5.3	8	6	5	1
32	F	L	8.7	3	f	16	0	7.3	1.2	4.9	14.7	12	5	7
33	M	R	11.8	4	c	10	1.3	5.5	0	3.3	27.3	9	5	4
34	M	L	7.7	4	c	7	0	1.7	0.5	0.9	2.7	•	•	•
35	M	R	6.1	4	f	15	0.5	6.7	–0.8	3.5	7.3	7	6	1
36	M	R	11.3	4	f	20	0	5.7	0.9	3.5	6.4	10.5	7.5	3
37	M	L	5.3	4	f	13	0.5	11.5	2.7	7.5	25.3	9	5.5	3.5
38	M	R	6.8	3	f	24	0	3.3	1.2	1.9	4	5	4	1
39	M	R	7.3	4	f	13	0.6	0.4	0	0	4.4	9	5	4

A Patient number
B Sex
C Side
D Age (yrs) at the time of investigation
E Catterall group
F Stage
  c condensation
  f fragmentation
G Duration of symptoms (months) at the time of investigation
H Aspirated volume (mL)
I–L Intracapsular pressure (kPa) with the hip in extension and neutral position, with the hip in flexion 45 °, with the hip in extension/outward rotation and with the hip in extension/inward rotation
M Anterior capsular distance in the symptomatic hip (mm)
N Anterior capsular distance (mm) in the asymptomatic hip
O Capsular distension (mm)

ular to the neck of the femur, was measured in the symptomatic as well as in the asymptomatic hip (Figure 2). A side difference ≥ 1 mm (Wingstrand 1986), was considered pathologic. This difference was defined as the capsular distension. The sonographic investigation and analysis were performed by a radiologist, without knowledge of any radiological or pressure data.

Intracapsular pressure recordings were performed in all 39 hips in conjunction with arthrography or surgery. All patients had general anesthesia and were placed supine with the hips freely movable. A 1.2 mm epidural cannula on a 2 mL syringe was connected via a 3-way stop-cock to a piezoelectric pressure transducer via pressure-resistant tubing. This non-volume consuming system was filled with saline and calibrated before the procedure. Using an image intensifier, the cannula was introduced anterolaterally, in order to penetrate the joint capsule over the ventral, mid-part of the neck of the femur, where the capsular distension in cases of synovitis has its maximum. The cannula was introduced through the joint capsule and synovia only once to avoid leakage. The intracapsular position of the cannula was verified by

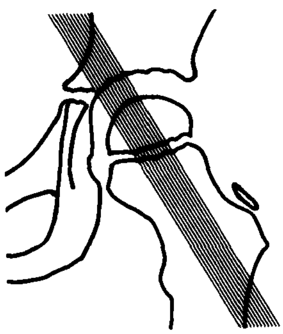

Figure 1. The direction of the sonographic section from the ventral aspect along the neck of the femur.

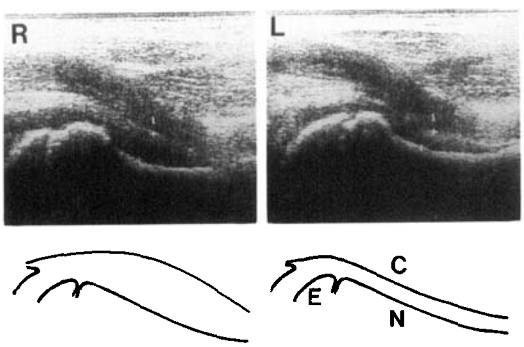

Figure 2. Anterior capsular distance in the right (R) symptomatic hip as compared to the contralateral (L) asymptomatic hip. Epiphysis (E), neck of femur (N) and joint capsule (C)

observing the sudden rise in pressure on the oscilloscope when penetrating the capsule and/or the fluctuations when rotating the joint and when aspirating at the end of the procedure. Readings were obtained digitally on an oscilloscope.

The pressure was recorded with the hip in the following positions: 1) extension and neutral rotation, 2) extension and maximum inward rotation, 3) extension and maximum outward rotation, and 4) with the hip in 45° of flexion. Aspiration was performed with the hip in extension and neutral position. Pressure values were presented in kPa (7.5 mmHg). Linear regression was used in the statistical analysis.

Results (Table 1)

In the sonographic study, 9 cases were not included in the analysis since 5 children had bilateral disease and 4 children were not examined. A capsular distension ≥ 1mm was recorded in 28 out of 30 cases. The mean capsular distension was 3.0 (1.0–7.0, SD 1.6) mm.

On aspiration, the presence of intracapsular effusion was verified in 23 out of 39 hips, a mean volume of 0.6 (0.1–2.8, SD 0.6) mL was aspirated. In 16 hips we found no fluid on aspiration. Sonography diagnosed a capsular distension in 13 of these hips, while in 1 hip no distension was found, in 1 hip no sonography was performed and in one case there was bilateral disease. In one case (Case 15) 0.8 mL of fluid was aspirated, but sonography had not been able to demonstrate any distension.

Intracapsular pressure recordings were obtained in 37 of the hips. 2 cases were excluded due to technical failure during pressure recordings. The intracapsular pressure with the hips in various positions, as described above, are given in Table 2. We recorded a mean value of 4.5 kPa with the hip in extension and neutral rotation, 9.7 kPa in extension and inward rotation, and 0.9 kPa in 45° of hip flexion.

There was a tendency towards a decrease in sonographic capsular distension of the joint capsule with time since the onset of symptoms (*P* 0.056) and a decrease in intracapsular pressure in extension and neutral rotation with the passage of time (*P* 0.058) (Figure 3).

There was no correlation between intracapsular pressure and the aspirated volume of fluid, nor between intracapsular pressure and sonographic distension of the capsule, nor between the aspirated volume of fluid and sonographic distension of the capsule.

Discussion

Little attention has been paid in the literature to the effects of synovitis and ensuing increased intracapsular pressure in LCPD. Experimentally induced synovitis (Gerschuni et al. 1981, Gershuni and Kuei 1984) was shown to cause cartilage edema, degradation of the mechanical properties, cartilage hypermetabolism and cartilage hypertrophy. Cartilage edema was also observed in transient synovitis (Egund et al. 1987). These observations would very well explain the unfavorable cartilage hypertrophy with metaphyseal widening and subsequent lateral sublux-

Table 2. Intracapsular pressure in 37 patients with Legg-Calvé-Perthes' disease

Position of hip joint	Intracapsular pressure (kPa)		
	Mean	SD	Range
Extension, neutral rotation	4.5	2.8	0–11.5
Extension, inward rotation	9.7	6.4	1.3–27.3
Extension, outward rotation	2.5	1.9	0–7.5
Flexion 45 °	0.9	1.1	–0.8–4.7

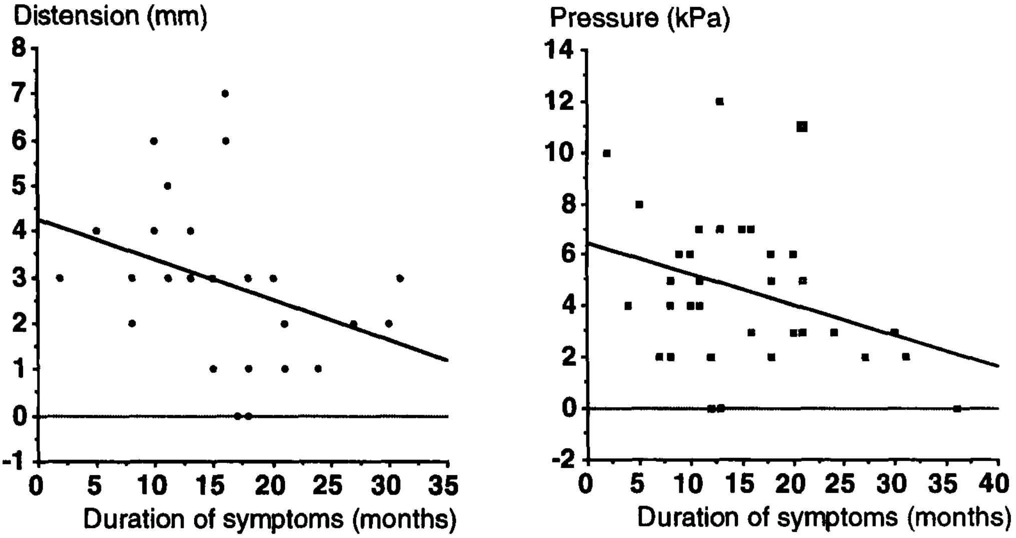

Figure 3. Sonographic distension of the joint capsule with the duration of time since onset of symptoms (left) and intracapsular pressure in extension and neutral rotation with the duration of time since onset of symptoms (right).

ation with anterolateral deformation due to poor containment and incongruency in the head-acetabulum fit in LCPD.

Synovitis is clinically common in LCPD, in particular in the earlier phases of the disease, with pain and a decreased range of hip motion. We found a tendency towards a decrease in sonographic distension of the joint capsule with time since the onset of symptoms and a decrease in intracapsular pressure occurred over a period of time. We found an anterior capsular distension, indicating synovitis, in 28 out of 30 unilateral cases. Our findings in this respect are in accordance with the sonographic findings of Terjesen (1993), who reported an increased capsular distension ≥ 2mm in 12 out of 19 hips with LCPD.

In the normal joint without intracapsular fluid, there is no increase in intracapsular pressure nor any tension in the capsule within the normal range of rotation around the axis of the neck of the femur (Wingstrand et al. 1990). The prerequisite for this is its hyperboloid shape with the smallest diameter in the midpart of the neck of the femur. This design permits the rotation of the hip around the neck of the femur without causing any stretching and thus tension in the capsule. This hyperboloid shape of the capsule is distorted in a hip with synovial edema and/or intracapsular effusion. Under such circumstances, rotation causes a significant increase in intracapsular pressure, thus limiting the normal painless range of rotation around the axis of the neck of the femur; pain-transmitting nerves have been identified in synovial membranes (Kellgren and Samuel 1950, Grönblad et al. 1985).

Recent attention in the literature to intracapsular pressure in LCPD has been focused mainly on the unfavorable circulatory effects. That an intracapsular effusion or hematoma with ensuing increase in intracapsular pressure compromises the blood flow to the PFE has been shown experimentally (Woodhouse 1964, Kemp 1981, Launder et al. 1981, Lucht et al. 1983, Vegter and Klopper 1991), and it has been suggested clinically in hip joint tamponade caused by transient synovitis (Kloiber et al. 1983, Kallio and Ryöppy 1985, Vegter 1987, Wingstrand et al. 1985a), in septic arthritis (Wingstrand et al. 1987), intracapsular bleeding due to fractures (Wingstrand et al. 1986) and intracapsular bleeding due to hemophilia (Pettersson et al. 1990). Animal studies have shown decreased blood circulation to the PFE or even necrosis if the intracapsular pressure approaches 50 mm Hg (6.7 kPa) and is maintained for several hours (Woodhouse 1964).

Our pressure recording results are well in accordance with the findings of Kallio and Ryöppy (1985) and Vegter (1987), showing mean pressures of 3.4 kPa and 7.7 kPa, respectively, in LCPD in extension and neutral rotation and mean pressures of 3.3 and 36.5 kPa, respectively, in extension/inward rotation, the latter in 4 cases. In flexion, they reported 2.6 and 2.0 kPa, respectively.

The magnitude of these pressures in many cases much exceeds the pressure in the vessels draining the femoral epiphysis and would thus reduce the pressure gradient across the capillary bed, possibly to a level where the metabolic demands of the epiphyseal cells are no longer met (Green and Griffin 1982, Liu and Ho 1991). This concept of ischemic episodes in the PFE due to intracapsular tamponade would be in accordance with the histological findings by Inoue et al. (1976).

We could not establish any correlation between intracapsular pressure and the aspirated volume of fluid. Such a correlation has been demonstrated in other cases of synovitis, mainly transient synovitis (Kallio and Ryöppy 1985, Wingstrand et al. 1985b, Erken and Katz 1990). Such a correlation has not yet been demonstrated in LCPD due to a wide range of individual variations in capsular compliance in this age group (Wingstrand 1985b) and since the volumes of aspirated fluid are significantly lower and the number of cases smaller. For the same reasons we were unable to establish any correlation between intracapsular pressure and sonographic distension of the capsule, nor between aspirated volume of fluid and sonographic distension of the capsule.

In 16 hips we found no fluid on aspiration. However, in 14 of these the intracapsular pressure was increased. This may be explained by synovitis with synovial edema, but without free or accessible fluid. In 2 hips no values were obtained due to technical failure.

We conclude that synovitis can be diagnosed sonographically in LCPD. This synovitis is probably a symptomatically and prognostically important factor in LCPD due to increased intracapsular pressure with ensuing pain and a decreased range of motion and, if it is persistent, a joint contracture. Synovitis in LCPD causes an increase in intracapsular pressure, the magnitude of which may well, in some cases, intermittently compromise the blood supply to the PFE.

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