Intracapsular pressure and loosening of hip prostheses

Preoperative measurements in 18 hips

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We measured the intracapsular pressure preoperatively in 18 hips (17 patients) before revision of a total hip arthroplasty because of aseptic loosening. Distension of the joint capsule was measured with sonography in 13 cases.

In extension, the mean intracapsular pressure was 26 (0–60) mmHg, in extension and inward rotation it was 159 (24–280) mmHg, in extension and outward rotation it was 30 (3–67) mmHg and in 45° of flexion it was 12 (0–28) mmHg. A mean of 6 (0.5–20) mL of joint fluid was aspirated after the pressure measurements.

Sonography showed increased joint fluid/synovial edema and/or increased capsular thickness, as compared to 34 unrevised, radiographically not loose prosthetic hips, and that the capsular distension correlated to intracapsular pressure during extension and inward rotation.

We conclude that the intracapsular pressure usually is elevated in a hip joint with loose prosthetic components, that the intracapsular pressure varies with the position of the hip and that capsular distension reflects increased intracapsular pressure. The increased and often very high pressure, varying during gait, may pump debris away from the joint along the interfaces and even by itself cause osteolysis and loosening.

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How polyethylene wear debris is transported away from a prosthetic joint is poorly understood. Particles have been found as far distally as in the distal metaphysis of the femur (Pazzaglia and Byers 1984, Schmalzried et al. 1992), and osteolysis sometimes appears locally, with or without polyethylene particles, in the granulomatous tissue (Willert et al. 1990), sometimes without other signs of loosening (Anthony et al. 1990, Maloney et al. 1990, Tanzer et al. 1992).

Effusion causing distension of the capsule and/or increased intracapsular pressure has been observed in several hip disorders, such as arthrosis (Timofeeva 1975, Goddard and Gosling 1988, Robertsson et al. 1995), transient synovitis (Kloiber et al. 1983, Wingstrand et al. 1985a,b), septic arthritis (Wingstrand et al. 1987) and juvenile chronic arthritis (Rydholm et al. 1986).

We measured the intracapsular pressure in patients with aseptic loosening of hip prostheses and correlated the pressure to observations during sonography.

Patients and methods

We examined 17 patients (18 hips) scheduled for revision of a cemented hip arthroplasty due to aseptic loosening, as defined by Carlsson et al. (1993), after 4–20 years. This was the first revision in all cases except in 1 patient, who had previously been reoperated on during the first postoperative year, with exchange of the femoral head to gain length of the neck. The mean age at revision was 77 (66–90) years. The reason for the primary hip replacement was arthrosis in 17 hips, psoriatic arthritis in 1 hip.

Preoperative radiography showed loosening of both components in 12 cases, of the femoral component only in 4 cases and of the acetabular cup only in 2 cases.

Preoperative sonography was done in 13 cases with a 7.5 MHz transducer from the ventral aspect of the thigh along the axis of the neck of the femoral component. The anterior capsular distension was defined as the distance between the anterior contour of the neck of the prosthesis and the outer contour of the capsule. The results were compared to the corresponding find-

Preoperative observations in 18 hip arthroplasties revised for aseptic loosening

A	B	C	D	E	F	G	H	I
1	11	21	5	120	46	1	Both	90
2	18	42	20	142	61	8	Both	82
3	20	45	20	280	20	1	Both	79
4	•	19	5	150	4	0.5	Cup	80
5	20	26	0	200	12	3.5	Fem	82
6	•	12	1	210	45	11	Fem	66
7	•	12	4	202	17	15	Both	75
8	20	30	25	200	30	20	Both	70
9	10	29	3	120	16	4	Both	86
10	23	0	4	110	3	1.5	Both	83
11	12	12	13	24	42	3	Both	74
12	•	45	28	145	38	0.5	Fem	78
13	16	30	23	128	55	1	Cup	73
14	18	12	4	98	9	10	Both	77
15	22	38	18	150	67	15	Both	66
16	23	60	11	220	5	10	Fem	86
17	16	12	13	140	9	2.5	Both	74
18	•	29	22	230	53	0.5	Both	66

A Case no.
B Capsular distension, mm
C Intracapsular pressure in extension, mmHg
D Intracapsular pressure in 45° flexion, mmHg
E Intracapsular pressure in extension and inward rotation, mmHg
F Intracapsular pressure in extension and outward rotation, mmHg
G Volume of aspirated fluid, mL
H Components with radiological signs of loosening
I Age of the patient at revision

ings in 34 prosthetic hips without loosening after 10 years. The reference group consisted of all primary hip arthroplasties asked to return to a 10-year follow-up at our department from January 1995 to February 1996 without clinical or radiographic signs of loosening.

The pressure recordings were performed during general anesthesia just before revision surgery. The pressure was recorded with a 0.8 mm cannula connected via a saline-filled pressure-resistant tube to a piezo-electric pressure transducer. Thus, the recording system was not volume-consuming. With the patient supine and using fluoroscopy, the cannula was entered anterolaterally into the joint, until we felt the cannula against the metallic prosthetic neck of the femoral component. The pressure was then recorded with the hip in extension and neutral rotation, in 45° of flexion and neutral rotation, in extension and outward rotation and finally in extension and inward rotation. Following the pressure registration, joint fluid was aspirated and the volume was measured.

The Student's t-test and regression analysis were used for statistical analysis.

Figure 1. Mean Intracapsular pressure (±SD) in 18 hips scheduled for revision of a cemented hip arthroplasty due to aseptic loosening. Pressure was recorded with the hips in extension and neutral rotation, in 45° of flexion and neutral rotation, in extension and inward rotation and finally in extension and outward rotation.

Results (Table)

The mean preoperative sonographic capsular distension in the 13 loose prosthetic hips was 17 (range 10–23, SD 4.4) mm, and in the 34 hips without radiographic loosening it was 13 (range 3–22, SD 4.6), ($p = 0.006$).

The mean intracapsular pressure in the 18 loose prosthetic hips was 26 (0–60) mmHg in extension, 159 (24–280) mmHg in extension and inward rotation, 30 (3–67) mmHg in extension and outward rotation and 12 (0–28) mmHg in 45° of flexion (Figure 1). All patients had a pressure of 42 mmHg or higher in some position of the hip.

There was a positive correlation between the intracapsular pressure in extension and inward rotation and sonographic distension of the capsule ($p = 0.05$, $r = 0.55$) (Figure 2).

6 (0.5–20) mL of joint fluid was aspirated after the pressure registration.

Discussion

Hendrix et al. (1983) noted that physiologic movements in a prosthetic hip joint cause intracapsular pressure alterations; they recorded pressure variations during walking after pressure arthrography. In spite of an attempt to aspirate the contrast medium until the pressure was zero, the maximum pressure during gait increased to a mean of 207 (155–517) mmHg. Anthony et al. (1990) measured the pressure in a lytic

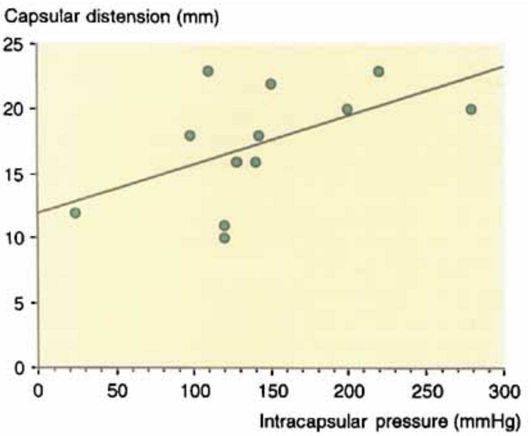

Figure 2. Correlation between capsular distension (mm) and intracapsular pressure (mmHg) with the hip in extension and inward rotation in 18 hips scheduled for revision of a cemented hip arthroplasty due to aseptic loosening.

lesion in the femur in a patient with a loose prosthesis. They found that passive flexion of 15° could raise the pressure to 198 mmHg, and they speculated that the elevated pressure could cause osteolysis, but did not determine whether elevated pressure was a common phenomenon.

Polyethylene wear is the major source of particulate wear debris (Boynton et al. 1991, Hirakawa et al. 1996). A relationship between wear of the socket and loosening of the prosthetic components was shown by Hamilton and Gorczyca (1995), and it is believed that wear particles induce osteolysis. Polyethylene particles have been found far from the joint in the distal metaphysis of the femur (Pazzaglia and Byers 1984, Schmalzried et al. 1992). It is very likely that capsular tension and cyclic loading during gait function as a pump that distributes particle-containing joint fluid through the port of least resistance—i.e., along the interfaces between the bone and the cement or between the components and the cement. Cracks in the cement would also provide a quick route for the fluid towards more distal sites.

An interesting question is whether the very high pressure in itself may cause resorption of bone by interfering with the perfusion and oxygenation of bone (Anthony et al. 1990). Bone cysts and intraosseous ganglia have been thought to be produced by joint fluid, which is forced by the pressure through defects in the cartilage (Landells 1953, Crane and Scarano 1967). Pressure from aortic aneurysms also causes bone resorption (Carruthers et al. 1986). One indication of this mechanism is that wear particles from the joint are not always found in areas of osteolysis (Willert et al. 1990).

In our study, the amount of aspirated joint fluid was often small, even in cases with high intracapsular pressure. This might be explained by variations in joint volume and compliance of the joint capsule and, more likely, the fact that the main content of the cavity is not fluid, but a mass, which represents thickened, edematous and hypertrophic synovium (personal observation). In children with Legg-Calvé Perthes' disease it has been shown that synovial edema, without substantial increase in the amount of joint fluid, can increase intraarticular pressure (Eckerwall et al. 1994).

In primary arthrosis and several other hip disorders, there is a correlation between increased intracapsular pressure and pain (Goddard and Gosling 1988, Robertsson et al. 1995). A similar correlation may exist in cases with a loose hip prosthesis. Since capsular distension can be measured by sonography, it may be of diagnostic value in a patient with painful hip replacement.

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