

# Intra-articular pressure in rheumatoid arthritis of the knee

## II. Effect of intra-articular pressure on blood circulation to the synovium

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Synovial effusions when under high pressure could theoretically interfere with the blood circulation to the joint lining. During the course of a study of intra-articular pressure in the resting knee joint with increasing volumes of simulated effusion, observations were made to illustrate this possibility.

### Observations (Figure)

In the course of measurement of knee joint elastance, pressures in simulated effusions were continuously recorded. With the joint empty, or containing only small volumes at relatively low pressures, straight-line tracings were obtained, indicating that the intra-articular pressure was constant. However, in some subjects, at increasing volumes of simulated effusion, a fine pulsation appeared. As further fluid was added, the pulsation increased at first, then diminished, and was finally obliterated. The pulsations were always synchronous with the arterial pulse.

### Subjects studied

This finding was sought in the fourteen rheumatoid knees and twelve normal control knees of subjects who volunteered for joint pressure studies (Jayson and Dixon, 1970a).

### Results

Pulsations synchronous with the arterial pulse were found in all fourteen rheumatoid joints but in none of the control knees.

The magnitudes of pulsation together with the volume and pressure ranges over which they were noted, the patients' blood pressures, and the intra-articular pressures at initial joint puncture are shown in the Table. The pulsation began at pressures below the diastolic pressure and was obliterated sometimes below and sometimes above the systolic pressure, reaching a maximum between the two. In three subjects pulsation was observed at only one

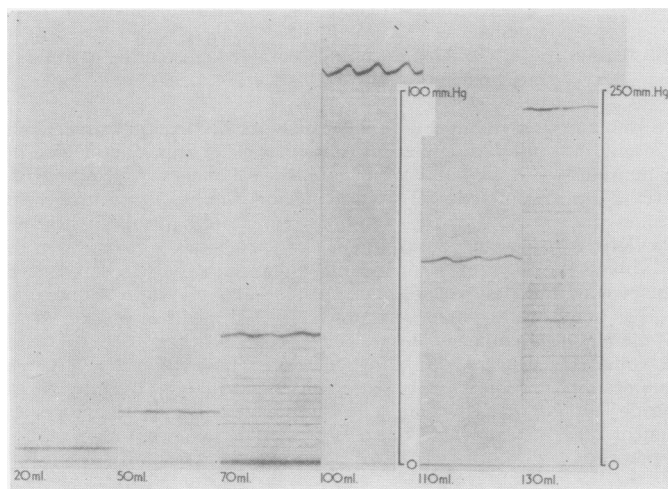


FIGURE Sections from resting pressure traces with increasing effusion volumes in a typical rheumatoid knee.

**Table** Pulsation in resting pressure traces from fourteen rheumatoid knees

Experiment no.	Pressure (mm. Hg)	Maximum pulsation (mm. Hg)	Volume range (ml.)	Pressure range (mm.Hg)	Blood pressure (mm.Hg)
1	9	± 1.5	70-115	33-152	150/70
2	10	± 1.5	20-50	22-140	180/110
3	27	± 1.0	40-90*	29-160*	160/90
4	23	± 1.5	50-90	25-110	140/80
5	7	± 1.0	20-45	39-145	140/70
6	9	± 1.0	20-60	60-165	170/90
7	11	± 0.5	40-80*	60-140*	130/80
8	16	± 1.5	30-75	22-130	150/80
9	36	± 1.5	50-90*	26-247*	180/100
10	4	± 0.5	60	83	170/105
11	34	± 0.25	10	165	200/120
12	22	± 0.25	10	45	190/100
13	30	± 1.0	15-40	28-156	180/105
14	26	± 1.0	15-45	31-113	120/80

Pressure = Resting intra-articular pressure at initial joint puncture. Volume and pressure ranges = Ranges over which pulsation was recorded.

\* = Pulsation not obliterated at maximum distension.

level of intra-articular pressure. In three rheumatoid knees the pulsation was reduced but not entirely absent at the maximum distension studied.

The magnitude of the pulsation observed varied from  $\pm 0.25$  to  $\pm 1.5$  mm. Hg. In general, the magnitude of this pulsation corresponded with the clinical signs of activity of the arthritis and of synovial hypertrophy.

### Discussion

This pulsation was observed only in rheumatoid joints. The synovial lining of normal joints has a relatively small blood supply but, in the inflammatory process that affects rheumatoid synovium, there is a marked increase in circulation. It seems likely that these pulsations are caused by the intra-articular pressure becoming similar to the pressure of the vessels permeating the synovium. At low intra-articular pressures the blood supply to the synovium is unimpeded and at very high pressures the vessels are compressed and all vascular flow stops. At each of these extremes no pulsation would be transmitted into the effusion. However, at intermediate pressures, blood flow during diastole would be arrested before that during systole. Pulsatile changes would appear in the synovium and would be communicated to the intra-articular fluid.

Compression of the innermost and smallest vessels probably starts at relatively low pressure, as these vessels may represent more terminal branches of arterioles and already have suffered a significant fall in blood pressure. Fluctuation in the intra-articular pressure therefore begins at pressures well below the diastolic pressure. Similarly, some of the outermost and largest vessels may be inefficiently

compressed and only partially obliterated by high pressure within the joint. In three knees the pulsation was not completely obliterated although reduced at the highest pressures used experimentally.

In subjects with chronic 'burnt-out' rheumatoid arthritis in whom there was only a small increase in joint circulation, only small fluctuations in intra-articular pressure ( $\pm 0.25$  mm. Hg) were observed. The larger pressure swings of  $\pm 1.5$  mm. Hg occurred in patients with relatively active joints who presumably had the greatest increase in blood supply to inflamed tissue.

In the control knees in which there was no pathological circulation this pulsation was not found. However, McCarty, Phelps, and Pyenson (1966) observed minute fluctuations in the intra-articular pressure of the normal dog knee when distended with fluid. From a trace they published, this fluctuation appeared to be of the order of  $\pm 0.05$  mm. Hg. Changes as small as this would not have been demonstrable by the techniques used in the present studies.

With increase in the pressure of the simulated effusion, there was progressive obstruction to the blood flow to the synovium, which became complete when the pulse wave was obliterated. This could cause ischaemic damage to the joint lining. In only two knees, however, were the pressures within the resting joint when first punctured within the pulsation range, and in none were the initial pressures above the levels at which the pulsations were obliterated. It would appear that effusions in rheumatoid arthritis do not reach a pressure high enough to cause a significant obstruction to the synovial circulation. Ropes and Bauer (1953), in an extensive study of synovial fluid from a wide variety of conditions, did not encounter levels of intra-articular pressure likely to be above the systolic blood pressure and therefore likely to produce synovial ischaemia. However, Ranke (1875) found that, in the presence of acute haemarthrosis, the intra-articular pressure could increase up to 200 mm. Hg. This could prevent blood flow into the synovium but might also act as a haemostatic mechanism.

Manoeuvres in which the intra-articular pressure is raised further, such as knee flexion and quadriceps contraction in the presence of disease and of effusions (Jayson and Dixon, 1970b), could affect the blood circulation to the synovium. This would be of significance only if the manoeuvre was continued for long enough to produce ischaemic damage.

### Summary

During distension of joints with simulated effusions, fluctuations in intra-articular pressure synchronous with the arterial pulse were observed in rheumatoid

knees but not in controls. These suggested that increased intra-articular pressure could interfere with the circulation to the synovium. The resting pressures were insufficient in any of these joints to obstruct the circulation completely, but this may well occur during temporary pressure increases due to joint use.

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### RÉSUMÉ

**La tension intra-articulaire du genou dans l'arthrite rhumatoïde.**

**II. L'effet de la tension intra-articulaire sur la circulation du sang au synovium.**

Pendant la distension des articulations par des épanchements simulés, les fluctuations de la tension intra-articulaire synchrones au pouls artériel ont été observées dans les genoux rhumatoïdes mais pas chez les témoins. Elles ont suggéré que la tension intra-articulaire accrue pouvait intervenir dans la circulation au synovium. Les tensions de l'articulation au repos n'étaient pas suffisantes dans aucune de ces articulations pour obstruer la circulation complètement, mais cela pouvait bien avoir lieu pendant des augmentations temporaires de tension dues à l'usage de l'articulation.

### SUMARIO

**Presión intraarticular en la artritis reumatoide de la rodilla**

**II. Efecto de la presión intraarticular en la circulación de la sangre hacia la sinovia**

Durante distensión de articulaciones con derrames simuladas se observaron fluctuaciones en la presión sincrónica intraarticular con el pulso arterial en rodillas reumatoides, pero no en testigos. Esto sugiere que la incrementada presión intraarticular podría afectar la circulación hacia la sinovia. Las presiones de reposo eran insuficientes, en cualquiera de estas articulaciones, para obstruir por completo la circulación, pero esto muy bien pudiera ocurrir durante aumentos temporarios de presión debidos al uso de las articulaciones.