Intra-articular pressure in rheumatoid arthritis of the knee

III. Pressure changes during joint use

M. I. V. JAYSON* and A. St. J. DIXON*

From the Royal National Hospital for Rheumatic Diseases, Bath,* and the Department of Medicine, Bristol Royal Infirmary1

Studies of pressure/volume relationships in control and diseased knees have demonstrated significant alterations in joint elastance (Jayson and Dixon, 1970). However, these studies were performed at rest and did not give information about changes in intra-articular pressure during joint use. One would never consider measurements in the stopped heart as adequate for study of its physiology, and similarly measurements in the resting joint present an incomplete picture. A further study was, therefore, conducted of the changes in intra-articular pressure occurring during both a standard series of exercises and walking.

Subjects studied

Measurements during formal knee exercises were performed on nine knees from eight control subjects and on sixteen knees from sixteen patients with definite or classical rheumatoid arthritis (Ropes, Bennett, Cobb, Jacox, and Jessar, 1959). They were repeated on one control and one rheumatoid knee after intervals of 4 and 3 months. The repeated studies demonstrated that the results were reproducible but were not used for the statistical analysis. In nine rheumatoid knees, these investigations were performed immediately after initial joint aspiration, but in the others and in all the control knees they immediately followed measurement of the pressure/volume relationships described by Jayson and Dixon (1970).

Measurements of pressures during walking were made in six control and four rheumatoid knees. Both knees from one control subject were used and showed similar results. In four of the control knees and in one rheumatoid knee measurements had also been made during formal exercises.

Method

In all studies the techniques for cannulating the joint, recording the intraarticular pressure, and adding and withdrawing effusion, were as described by Jayson and Dixon (1970). Measurement of the pressures produced with each movement were made, when possible, with volumes of simulated effusion of 0, 20, 40, 60, 80, and 100 ml. On some occasions additional readings were taken at narrower intervals. Some subjects were unable to tolerate large volumes of simulated effusion and in others joint rupture occurred so that useful readings were obtained only at lower volumes.

During the formal exercises four manoeuvres were performed in a regular sequence at each volume: elevation of the extended leg, isometric elevation, quadriceps setting, and passive knee flexion. In these and other studies this last movement appeared particularly likely to cause joint rupture and was therefore discontinued after nine experiments.

Elevation of the leg

The subject was encouraged to relax completely with the knee extended. He was then instructed to elevate the extended lower limb if possible so that the hip was flexed to 90°, and to hold this position for 5 seconds before slowly lowering the leg back onto the couch and relaxing. With larger volumes of simulated effusion, some subjects developed increasing difficulty and were unable to maintain the knee in the fully extended position (0°) during elevation. There was a ‘lag’ of a few degrees. A correction was applied to compensate for the hydrostatic pressure generated by raising the level of the knee.

Isometric elevation

The subject was again instructed to attempt to elevate the extended lower limb, but on this occasion movement was prevented by pressure of a hand over the lower shin.

Quadriceps setting

The subject was asked to tighten his quadriceps muscle forcibly and to push the back of the knee downwards onto the couch.

Passive knee flexion

The subject was encouraged to relax completely. The lower leg was firmly grasped by an observer and the knee was lifted and the heel firmly flexed up to the thigh. With progressive distension this movement produced symptoms of extreme joint tightness and tension and the maximum degree of flexion was progressively limited. A correction was again applied because of the change in height of the knee.
For measurements during walking, the transducer was strapped vertically to the subject's thigh and was joined to the cannula by a short length of tubing. The subject walked on a circular rotating platform. Preliminary studies showed that this produced pressure changes similar to those obtained by walking in a straight line. The period when the foot of the tested limb was on the ground or 'foot-stance' was noted, using an event marker, and was recorded as a horizontal line beneath the trace.

After-effects

In a number of instances the high pressure generated during joint use ruptured the synovial cavity and released the simulated effusion into the tissues of the lower limb. The fluid did not possess the irritant properties of synovial fluid and joint rupture did not produce the syndrome of acute calf pain and stiffness with oedema of the leg and foot that has been ascribed to the pathological effusions of rheumatoid arthritis (Dixon and Grant, 1964; Dixon, 1964; Tait, Bach, and Dixon, 1965; Hall and Scott, 1966; Jayson, Swannell, Kirk, and Dixon, 1969). The only symptom due to the rupture noted by the subject was a sudden loss of the sense of tension in a previously-distended knee joint. No after-effects developed except for some calf aching which disappeared within 24 hours. One of the authors personally confirmed this finding. Joint rupture was diagnosed by a sudden fall in the recorded pressure developed during exercise (Fig. 1*), loss of effusion on clinical examination of the joint, and failure to recover the full volume at the end of the study. It occurred in four of the nine control knees and in three of sixteen rheumatoid knees. The difference in incidence was not of statistical significance. ($\chi^2 = 0.83$; $P > 0.3$). However, the three rheumatoid knees showed only minimal signs of involvement by the disease, and if the comparison had been made of the incidence of rupture between knees which were completely or relatively normal, and those which showed advanced changes of rheumatoid arthritis, all would have been in the former group.

The remaining rheumatoid patients noticed no after-effects after the study, but the controls often suffered a mild synovitis lasting about 24 hours. This may well have been associated with a return to normal activity immediately after the procedure, whereas the rheumatoid patients rested in bed for 24 hours.

Results

**PASSIVE KNEE FLEXION**

This was performed on three control and six rheumatoid knees. The mean intra-articular pressures produced in the empty joints were similar. With additional fluid, knee flexion produced very much higher pressures in both groups. The rheumatoid knees produced higher mean pressures than the controls, but insufficient observations were made to permit a statistical analysis.

**Table 1 Intra-articular pressure (mm. Hg) during passive knee flexion**

<table>
<thead>
<tr>
<th>Volume</th>
<th>Rheumatoid arthritis</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure</td>
<td>No.</td>
<td>Pressure</td>
</tr>
<tr>
<td>0</td>
<td>144</td>
<td>133</td>
</tr>
<tr>
<td>20</td>
<td>291</td>
<td>200</td>
</tr>
<tr>
<td>40</td>
<td>512</td>
<td>370</td>
</tr>
<tr>
<td>60</td>
<td>633</td>
<td>425</td>
</tr>
<tr>
<td>80</td>
<td>777</td>
<td>458</td>
</tr>
<tr>
<td>100</td>
<td>802</td>
<td>478</td>
</tr>
</tbody>
</table>

Volume = Volume of simulated effusion (ml.)
Pressure = Mean intra-articular pressure (mm. Hg)
S.E. = Standard error (mm. Hg)
No. = Number of measurements

The larger volumes of simulated effusion limited the degrees of flexion that were permitted by the subjects. Symptoms experienced were those of a sense of tension or bursting and were similar to those observed during passive joint distension. If full flexion had been carried out at the larger volumes of effusion ignoring the symptoms experienced, very much higher pressures would have developed and ruptures would have been more frequent.

During flexion, variation in the intra-articular pressure was recorded. It was noted that the position for minimal intra-articular pressure was at about 30° of knee flexion. This was in keeping with the finding of Favreau and Laurin (1963), who showed that intra-articular pressures recorded in cadaver knees were lowest with 40° of flexion, and that the flexion deformity in arthritis corresponded...
Intra-articular pressure in rheumatoid arthritis of the knee

403

with the joint positions at which the lowest values were found. Eyring and Murray (1964) similarly showed that the positions spontaneously adopted by patients with effusions were those of minimum intra-articular pressure and that these produced the minimum discomfort.

Pressures during quadriceps contraction (Tables II, III and IV)

Typical traces from a control subject are shown in Fig. 2. The top trace demonstrates a subatmospheric pressure produced in the empty joint and the bottom a positive pressure with a 20 ml effusion. At 10 ml between the two there was no significant variation from the baseline.

The mean intra-articular pressures produced by rheumatoid and control knees at each volume of effusion and with each exercise was calculated and statistical comparisons were made using Students' t test.

In the empty joint a subatmospheric pressure developed in six control knees on elevation, eight on isometric elevation, and four on quadriceps setting. In contrast, positive pressures developed on

![Fig. 2](image)

Control knee. Quadriceps contraction at 0, 10, and 20 ml.

In the empty joint a subatmospheric pressure

Table II Intra-articular pressure (mm. Hg) during leg elevation

<table>
<thead>
<tr>
<th>Volume</th>
<th>Rheumatoid arthritis</th>
<th>Controls</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pressure</td>
<td>S.E.</td>
<td>No.</td>
<td>Pressure</td>
</tr>
<tr>
<td>0</td>
<td>72.4</td>
<td>20.8</td>
<td>16</td>
<td>-43.9</td>
</tr>
<tr>
<td>20</td>
<td>246.4</td>
<td>55.3</td>
<td>15</td>
<td>29.3</td>
</tr>
<tr>
<td>40</td>
<td>346.6</td>
<td>52.0</td>
<td>14</td>
<td>75.3</td>
</tr>
<tr>
<td>60</td>
<td>452.2</td>
<td>48.1</td>
<td>12</td>
<td>126.0</td>
</tr>
<tr>
<td>80</td>
<td>429.3</td>
<td>27.8</td>
<td>8</td>
<td>158.3</td>
</tr>
<tr>
<td>100</td>
<td>552.0</td>
<td>49.9</td>
<td>5</td>
<td>203.0</td>
</tr>
</tbody>
</table>

Table III Intra-articular pressure (mm. Hg) during isometric elevation

<table>
<thead>
<tr>
<th>Volume</th>
<th>Rheumatoid arthritis</th>
<th>Controls</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pressure</td>
<td>S.E.</td>
<td>No.</td>
<td>Pressure</td>
</tr>
<tr>
<td>0</td>
<td>88.6</td>
<td>29.5</td>
<td>14</td>
<td>-106.9</td>
</tr>
<tr>
<td>20</td>
<td>278.9</td>
<td>52.3</td>
<td>13</td>
<td>34.6</td>
</tr>
<tr>
<td>40</td>
<td>399.5</td>
<td>52.0</td>
<td>11</td>
<td>103.4</td>
</tr>
<tr>
<td>60</td>
<td>474.0</td>
<td>42.6</td>
<td>10</td>
<td>176.4</td>
</tr>
<tr>
<td>80</td>
<td>402.5</td>
<td>67.4</td>
<td>6</td>
<td>237.5</td>
</tr>
<tr>
<td>100</td>
<td>385.0</td>
<td>110.9</td>
<td>4</td>
<td>245.0</td>
</tr>
</tbody>
</table>

Table IV Intra-articular pressure (mm. Hg) during quadriceps setting

<table>
<thead>
<tr>
<th>Volume</th>
<th>Rheumatoid arthritis</th>
<th>Controls</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pressure</td>
<td>S.E.</td>
<td>No.</td>
<td>Pressure</td>
</tr>
<tr>
<td>0</td>
<td>72.6</td>
<td>15.5</td>
<td>14</td>
<td>-0.3</td>
</tr>
<tr>
<td>20</td>
<td>295.0</td>
<td>60.5</td>
<td>13</td>
<td>128.9</td>
</tr>
<tr>
<td>40</td>
<td>410.5</td>
<td>72.3</td>
<td>11</td>
<td>247.1</td>
</tr>
<tr>
<td>60</td>
<td>424.2</td>
<td>67.3</td>
<td>10</td>
<td>256.4</td>
</tr>
<tr>
<td>80</td>
<td>381.7</td>
<td>84.3</td>
<td>6</td>
<td>242.5</td>
</tr>
<tr>
<td>100</td>
<td>480.0</td>
<td>164.2</td>
<td>4</td>
<td>179.0</td>
</tr>
</tbody>
</table>

P = Probability of such differences occurring by chance
N.S. = Not significant; P > 0.05
all but one occasion in the rheumatoid joints. The differences in mean pressures between the two groups on all three exercises were statistically significant.

With increasing volumes of simulated effusion, joint use produced higher pressures. In the control joints, pressures rose towards and then above the atmospheric pressure. Fig. 2 demonstrates no significant pressure change at 10 ml but a positive pressure at 20 ml.

At larger volumes, however, with the addition of more fluid, the pressures produced failed to increase further and in several subjects it decreased. These subjects reported difficulty in performing the exercises and it was seen that the quadriceps muscles were not contracting properly. Quadriceps inhibition was not caused by pain as several subjects experienced minimal symptoms but felt physically unable to contract the muscle fully. Others felt marked tension or bursting sensations and were yet able to perform the exercises.

Fig. 3 illustrates the pressures developed on isometric elevation with increasing effusion volumes in a typical control knee, and Fig. 4 the pressures on elevation of the extended lower limb in a typical rheumatoid joint.

![Graph](image)

**FIG. 3** Control knee. Isometric quadriceps contraction.

At each volume and with each movement the mean pressures achieved by the rheumatoid knees were higher than those achieved by the controls. The differences between the two groups were highly significant at all volumes on elevation. Isometric elevation produced differences that were highly significant up to 60 ml but not at 80 or 100 ml. With quadriceps setting, the differences were of statistical significance at 0 and 20 ml but not at larger volumes.

The control knees were virtually empty at initial puncture whereas most of the rheumatoid joints contained effusions. Statistical comparisons of the pressures developed at these particular volumes were performed (Table V). For all three movements the differences were highly significant.

**PRESSURES DURING WALKING**

Only satisfactory traces in which the intra-articular pressures fluctuated in regular patterns during repeated steps were analysed. Typical traces obtained with increasing effusion volumes in control and rheumatoid knees are shown in Figs 5 and 6 (opposite and overleaf).

The peak and trough pressures for each of five consecutive paces were measured and the average values calculated. Observations were made to see whether the peak or the trough coincided with foot- stance or swing.

In the empty control joints, foot-stance coincided with a phase of subatmospheric intra-articular pressure. With the addition of simulated effusion, the magnitude of the subatmospheric pressure diminished and this phase became positive and also became the peak pressure. No negative phase of intra-articular pressure was found in the rheumatoid joints.

**Table V** Intra-articular pressure (mm. Hg) produced at initial effusion volume

<table>
<thead>
<tr>
<th>Movement</th>
<th>Rheumatoid arthritis</th>
<th>Controls</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pressure</td>
<td>S.E.</td>
<td>No.</td>
</tr>
<tr>
<td>Elevation</td>
<td>202·6</td>
<td>42·2</td>
<td>15</td>
</tr>
<tr>
<td>Isometric</td>
<td>223·3</td>
<td>44·9</td>
<td>13</td>
</tr>
<tr>
<td>Quadriceps</td>
<td>207·2</td>
<td>43·0</td>
<td>13</td>
</tr>
</tbody>
</table>
joints, however, and foot-stance always coincided with peak pressure. This increased in magnitude with increasing volumes of simulated effusion.

Statistical comparisons were made for the mean foot-stance pressures in control and rheumatoid knees at 0, 10, and 20 ml. of effusion. At each volume the rheumatoid pressures were considerably higher than the controls and the differences were of high statistical significance (Table VI, overleaf).

The pressure changes in the stance phase of walk-
TABLE VI  
**Intra-articular pressures (mm. Hg) during foot-stance**

<table>
<thead>
<tr>
<th>Volume</th>
<th>Rheumatoid arthritis</th>
<th>Controls</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pressure</td>
<td>S.E.</td>
<td>No.</td>
<td>Pressure</td>
</tr>
<tr>
<td>0</td>
<td>85.8</td>
<td>22.1</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>162.5</td>
<td>31.1</td>
<td>4</td>
<td>-15.3</td>
</tr>
<tr>
<td>20</td>
<td>257.0</td>
<td>54.6</td>
<td>4</td>
<td>-3.0</td>
</tr>
</tbody>
</table>

Discussion

The activity of the muscles in the lower limb was investigated using a telemetering technique by Battye and Joseph (1966). They found that the quadriceps muscle contracts during the first half of the support phase of walking with sometimes a second shorter period of contraction just before the end of this phase. This was consistent with the experimental finding of similar pressures during foot-stance and formal quadriceps exercises. On some occasions a bifid peak was observed on the pressure trace during...
the stance phase (Figs 5 and 6), suggestive of two periods of quadriceps contraction.

The rheological properties of the simulated effusion differed considerably from those of normal and diseased synovial fluids. However, the pressure measurements during formal exercises were obtained during periods of sustained quadriceps contraction and knee flexion when joint movement was not actually taking place and it would seem unlikely that the results would differ significantly from those to be expected with the various types of synovial fluid.

The normal subjects were relatively young, healthy, and usually male, whereas the rheumatoid patients were older, usually female, frail, and often with weak and wasted quadriceps muscles. It would seem likely that the controls would develop the higher intra-articular pressures. However, the mean pressures were always lower in the controls and the differences were often statistically highly significant. With better matching, the differences may well have been even greater.

The empty control joints developed subatmospheric pressures on quadriceps contraction. Dixon (1966) similarly found that the pressure in the normal human knee fell below the atmospheric level by up to 30 mm Hg during walking, but Reeves (1966) observed no marked variation on active or passive use of the joint. Careful observation of the knee of an athletic normal subject will often support the observation of a subatmospheric pressure on quadriceps contraction. When the subject contracts his quadriceps, the soft tissues appear sucked in between the tendon bands as they stand up from the joint. The mechanism of this subatmospheric pressure is in doubt, but it would seem possible that it is due to quadriceps contraction distracting the relatively lax capsule and tending to enlarge the joint space. The distended joint will however be compressed by quadriceps contraction so that positive pressures develop. These were also found by Caughy and Bywaters (1963) and DeAndrade, Grant, and Dixon (1965). The higher pressures in the rheumatoid knees may have been produced by two mechanisms. The rheumatoid joints contained hypertrophied synovium. This is semi-fluid at body temperature and could act as a functional effusion in addition to any fluid present. Alternatively, the inflammatory process alters the joint lining, producing contraction and fibrosis resistant to traction. Quadriceps contraction could distort the joint making it smaller and increasing the intra-articular pressure.

Several subjects showed the phenomenon of quadriceps inhibition with large volumes of simulated effusion (DeAndrade and others, 1965). This was probably due to stretch of the joint capsule rather than to the intra-articular pressure achieved, because above the critical volume at which inhibition occurred there was often a fall in the pressures produced. Again, some subjects ascribed their failure to contract the muscles to states of tension within the joints, but they described these during the resting phases between the exercises and not during the actual manoeuvres.

This inhibition appears to be part of a reflex protecting the joint against possible injury and is similar to those described by Blockey (1954), Swearingen and Dehne (1964), and Stener (1969). Receptors within the joint capsule have been demonstrated by Skoglund (1956) and by Wyke (1967). It is likely that joint distension stimulates the type I corpuscles and produces afferent impulses (Andrew and Dot, 1953; Eklund and Skoglund, 1960) which lead to quadriceps inhibition (Ekholm, Eklund, and Skoglund, 1960). This may be the mechanism producing quadriceps wasting in arthritis of the knee. Quadriceps exercises are often prescribed in order to restore muscle power, but, in the presence of marked distension, they would appear to be of little value and the volume of the effusion would have to be reduced before useful contraction could occur.

Use of the knee joint in the presence of rheumatoid arthritis and of effusions produces high intra-articular pressures in place of the normal subatmospheric pressures. These must place unphysiological strains on the articular surfaces and the synovial lining.

If the articular cortex is weakened by osteoporosis or by rheumatoid granulation tissue, these pressures may be sufficient to disrupt the surface and burst through into the marrow below, producing rheumatoid bone cysts. Castillo, El Sallab, and Scott (1965) found a significant correlation between physical activity and cystic bone lesions in the hands.

These pressures may also be sufficient to produce acute joint rupture. Experimentally, it was shown that this occurred fairly readily in normal or early involved knees but not in knees showing advanced degrees of involvement. This is compatible with the findings of Jayson and others (1969) that the clinical syndrome of acute joint rupture occurred in joints with only short histories of preceding arthritis. Instead of rupturing the joints, the pressures may be sufficient to 'blow out' the synovium and produce synovial cysts. The volume of effusion is reduced and any pressure rise during joint use is limited so that the joint is protected against pressure increases.

Summary

Measurements of pressures within the knees of control and rheumatoid subjects were obtained during passive knee flexion, quadriceps contraction, and walking, in the absence and presence of effusions. Passive flexion produced positive pressures in both
groups of knees, the levels of which increased with volume of fluid. Quadriceps contraction and foot-stand during walking both caused similar changes. A subatmospheric pressure was commonly produced in the empty control knee, but a positive pressure in the rheumatoid knee. With increasing effusion volumes, increasing positive pressures were developed by both groups, but the mean pressures produced by the rheumatoid knees were always higher than those produced by the controls and the differences were usually statistically significant. These high pressures sometimes produced acute joint rupture, but this occurred only in control or early involved knees and not in joints showing advanced changes of rheumatoid arthritis. Knee distension led to quadriceps inhibition in some subjects. This appeared to be due to stretch of the joint capsule rather than to the intra-articular pressure achieved.

We thank Dr. J. Cosh and Dr. G. D. Kersley for allowing us to examine their patients. We wish to express our gratitude to the Arthritis and Rheumatism Council and Association of Friends of the Royal National Hospital for Rheumatic Diseases, Bath, for grants for equipment and technical assistance. One of us (M.I.V.J.) is in receipt of a Research Grant from the Medical Research Council.

References


Intra-articular pressure in rheumatoid arthritis of the knee. 3. Pressure changes during joint use.

M I Jayson and A S Dixon

*Ann Rheum Dis* 1970 29: 401-408
doi: 10.1136/ard.29.4.401

Updated information and services can be found at:
[http://ard.bmj.com/content/29/4/401.citation](http://ard.bmj.com/content/29/4/401.citation)

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
[http://group.bmj.com/group/rights-licensing/permissions](http://group.bmj.com/group/rights-licensing/permissions)

To order reprints go to:
[http://journals.bmj.com/cgi/reprintform](http://journals.bmj.com/cgi/reprintform)

To subscribe to BMJ go to:
[http://group.bmj.com/subscribe/](http://group.bmj.com/subscribe/)