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AN INVESTIGATION INTO THE VALIDITY OF SUBATMOSPHERIC PRESSURE RECORDINGS FROM SYNOVIAL FLUID AND THEIR DEPENDENCE ON JOINT ANGLE

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SUMMARY

1. Synovial fluid hydrostatic pressures were measured in normal knee (stifle) joints of anaesthetized rabbits using perforated cannulae. Pressures were subatmospheric in seventy out of seventy-two joints, with a mean value of $-4{\cdot}6$ cm H_2O (range 0 to -12 cm H_2O) at joint angles of 120–150°.

2. Similar values were obtained by a wick-in-needle technique (mean $-4{\cdot}0$ cm H_2O), which along with several other tests indicated that the subatmospheric values were not artifactual.

3. A slow rise in pressure of 1–2 cm H_2O per hour in the motionless joint was attributed to a net filtration of fluid into the joint space.

4. Pressure increased as a curvilinear function of joint angle when the joint was flexed passively, the slope of the relationship depending on synovial fluid volume. Pressure also increased on active flexion of the joint, or on passively increasing the tension of soft peri-articular tissues.

5. Positive (above atmospheric) pressures in acutely flexed joints declined with time. Pressures declined less rapidly when synovial fluid was replaced by non-absorbable paraffin oil. It was concluded that joint fluid can be absorbed by the synovium during acute flexion.

6. Processes which might generate subatmospheric pressures are discussed. The hypothesis is advanced that the flexion-dependent 'trans-synovial pump', possibly in series with a lymphatic pump, may account for the maintenance of a small synovial fluid volume and subatmospheric pressure in the face of a net filtration of fluid from synovial capillaries into the joint space.

INTRODUCTION

Although the patho-physiology of inflamed joints has been the subject of much investigation, the physiology of normal synovial joints has received less attention. Thus, while there is widespread agreement that the hydrostatic pressure of inflammatory joint effusions is above atmospheric pressure, no agreement exists as to the magnitude of normal synovial fluid pressure in healthy joints. Several investigators have reported that fluid pressures, recorded through hollow needles inserted into the joints, are subatmospheric in healthy mammalian knee joints (Müller, 1929; Reeves, 1966; Jayson & Dixon, 1970*a*), but Caughey & Bywaters (1963) have

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suggested that such subatmospheric pressure recordings are artifactual, being created by a blockage of the needle tip. The issue assumes a wider interest in view of current controversy over reports of subatmospheric interstitial fluid pressures (Guyton, 1963), for synovial fluid is separated from interstitial fluid merely by a loose layer of connective tissue permeable to fluid (Levick, 1978).

There is also little agreement as to the effect of joint angle on synovial fluid pressure in the healthy as opposed to pathological joint. In human knee joints containing demonstrable effusions, acute flexion of the joint always causes a marked increase in intra-articular fluid pressure (e.g. Caughey & Bywaters, 1963). In normal human joints however, Reeves (1966) observed no change in fluid pressure upon altering knee joint angle, whereas Jayson & Dixon (1970*b*) observed marked increases in pressure on active or passive flexion of the knee.

The present investigation was carried out on normal knee joints of rabbits and attempted to record synovial fluid pressure, to investigate the suggestion that subatmospheric pressure recordings are artifactual and to re-assess the influence of changes in joint angle on pressure. The observations presented below may help reconcile the apparently conflicting observations of Reeves and of Jayson & Dixon, and may also contribute towards an understanding of how synovial fluid volume is regulated in normal joints.

METHODS

General procedure. New Zealand white rabbits of either sex weighing 2–4 kg were anaesthetized by I.V. sodium pentobarbitone (30 mg kg^{-1}) with urethane (0·5 g kg^{-1}). Anaesthesia was maintained by hourly administration of one tenth the dose of the above mixture. Tracheostomies were performed, and in ten out of the sixty-five rabbits investigated the right common carotid artery was cannulated and connected to an SE 488 transducer to record arterial blood pressure. Body temperature was monitored by a rectal thermistor and maintained at 38–39 °C by means of a heated body-blanket.

Cannulation of the knee (stifle) joint. The right hind limb of the supine animal was secured by ankle ties with the knee joint extended to 120–150°, at which angle the soft tissues investing the joint appeared to be under least tension (the 'position of ease', Barnett, Davies & MacConnaill, 1961). The skin overlying the patella was incised and reflected. A steel cannula was then inserted through the lateral aspect of the patellar ligament and advanced gently into the supra-patellar region of the articular cavity. A small additional dose of anaesthetic was usually administered for this procedure, as the peri-articular tissues appeared to be exquisitely sensitive to mechanical stimulation. A silk purse-string suture was placed around the cannulation site to eliminate leakages and the dissected area was covered by a Ringer-soaked gauze swab. Correct positioning of the cannula tip within the joint cavity was confirmed by dissection at the end of the experiment.

Measurement of synovial fluid hydrostatic pressure. In most experiments the cannula consisted of a saline-filled 21-gauge hypodermic needle with a 45° bevel. The cannula tip was pierced by five or six lateral perforations to minimize the possibility of tip obstruction. The cannula was connected by inextensible saline-filled tubing to an SE 1150/WG differential transducer level with the joint, the total compliance of the recording system being 0·1 μl. cm H_2O^{-1}. The transducer output was fed into an SE 3006 ultra-violet oscillograph to record synovial fluid hydrostatic pressure (accuracy $\pm$ 0·2 cm H_2O; drift $<$ 0·2 cm H_2O/hr and corrected hourly).

Because of the small bore of the cannula, insertion was possible without causing bleeding in most cases: in a few cases a rapidly rising pressure and development of a bloody effusion indicated a significant intra-articular bleed, and such experiments were abandoned.

Wick-in-needle technique. In six experiments designed to assess the validity of pressures recorded by the above technique, synovial fluid pressure was recorded via a wick-in-needle (Fadnes, Reed & Aukland, 1977). The wick-in-needle has been demonstrated to yield 'reasonable' estimates of subatmospheric pressures in the minute interstitial fluid spaces of connective

tissue. A lateral aperture 2–3 mm long was cut into the tip of a 19-gauge hypodermic needle, and long Sea Island cotton fibres drawn into the tip by means of a fine steel-wire loop. When this cannula was filled by Ringer solution, a very large number of small fluid-filled channels existed between the loosely packed cotton fibres. These allowed many contacts to be made with the synovial fluid and hence transmitted the synovial fluid pressure into the needle lumen with little possibility of tip blockage. For a more detailed exposition of the principles underlying the wick technique, see Scholander, Hargens & Miller (1968). The system was calibrated by recording graded subatmospheric fluid pressures existing between the fibres of fluid-soaked vertical strips of filter paper, as described by Scholander *et al.* (1968) and Snashall, Lucas, Guz & Floyer (1971).

Measurement and changes of joint angle. The medial inter-articular line was located by palpation and the axis of a pair of dividers placed over its midpoint. The arms of the dividers were adjusted to lie over the medial surface of the tibia and femur 6 cm from the inter-articular point. Joint angle was defined as the acute angle between the arms and was measured with a protractor.

The relationship between joint angle and synovial fluid hydrostatic pressure was determined for six different joints. Joint angle was varied between 160° (maximal extension) and 55° (maximal flexion, with calf pressing against thigh) by adjustment of the ankle ties and by foam supports to the paw. Pressure was recorded after the joint had been in its new position for 2 or 3 min.

RESULTS

Synovial fluid hydrostatic pressures recorded by perforated cannulae. Sustained subatmospheric (negative) pressures were recorded in seventy out of seventy-two synovial cavities in sixty-five rabbits, pressure being atmospheric (zero) in the other two cases. The time course of a pressure measurement is shown in Fig. 1*A*. Insertion of the cannula created initially a highly negative pressure, which decayed rapidly to reach an almost steady value by 8 min. When synovial fluid hydrostatic pressure was followed for a long period (46 min) a slight positive trend became evident, of the order +1 to +2 cm H_2O/hr. Higher speed recording of such pressure sometimes revealed small regular oscillations of ~0·1 cm H_2O at 250–300/min which were attributed to transmission of the arterial pulse to the synovial fluid (Fig. 1*B*).

The average hydrostatic pressure of synovial fluid at 120–150°, 8–10 min after cannula insertion was −4·6 cm H_2O ($n = 57$). The modal value was −3·5 cm H_2O

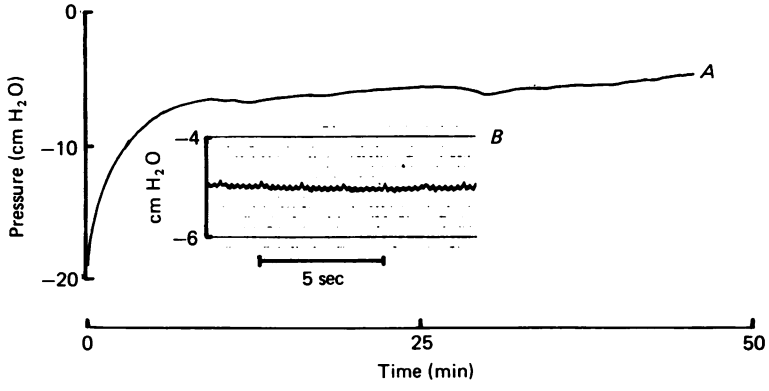

Fig. 1. Intra-articular hydrostatic pressures recorded from knee joints following insertion of perforated cannulae. Joint angle 120°. *A*, pressures recorded for 46 min after insertion of a cannula at time zero. *B*, higher speed and gain recording showing small oscillations in pressure of frequency 250/min, with augmented beats at a rate of 34/min synchronous with respiration.

and the total range 0 to $-12{\cdot}0$ cm H_2O (Fig. 2). In seven animals pressure was compared in the left and right knees. Measurements were made first on the right knee, within 30 min of anaesthetization. In five out of seven cases pressure was more negative in the right knee, which had been in active use until shortly before the measurement, than in the left knee, which had been immobile for an hour or more. The mean difference of $0{\cdot}6$ cm H_2O did not achieve statistical significance. Mean arterial blood pressures were normal during these measurements, being over 100 mmHg.

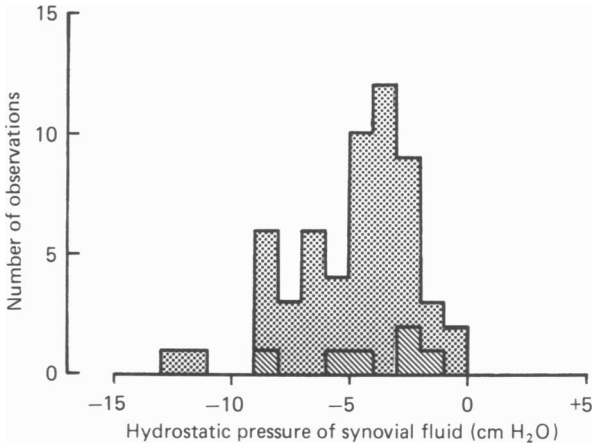

Fig. 2. Histogram showing distribution of synovial fluid hydrostatic pressures recorded through perforated cannulae (stippled areas) or wick-in-needle (crossed areas) after 8–10 min in rabbit knee joints at 120–150°.

Validity of subatmospheric pressure recordings. A number of simple tests were applied to assess the possibility that cannula obstruction had created artifactual pressure recordings.

(1) Digital pressure was applied to the posterior pouch of the synovial cavity in order to displace fluid into the supra-patellar region, mechanical disturbance of the supra-patellar pouch being carefully avoided. A small rise in supra-patellar fluid pressure of about 1 cm H_2O could be evoked in 25% cases. Pressure returned promptly to its former value on removal of the digital pressure.

(2) Infusions of Ringer solution through the articular cannula into the synovial cavity demonstrated that the cannula was patent and caused synovial fluid pressure to rise to above atmospheric pressure (three experiments). Over the next few minutes pressure fell progressively to re-establish a subatmospheric value as shown in Fig. 3. The original value was not fully restored however.

(3) If the hydrostatic pressure of synovial fluid is subatmospheric, it may be predicted that fluid will flow into the joint 'spontaneously' from a fluid-filled cannula connecting the joint cavity to the atmosphere. This phenomenon was observed on every occasion in twenty experiments in which a second fluid-filled cannula was inserted horizontally into the medial supra-patellar pouch; on opening the cannula to the atmosphere the fluid in the hub of the cannula was sucked rapidly into the joint. This observation was analogous to the 'hanging drop' method used by anaes-

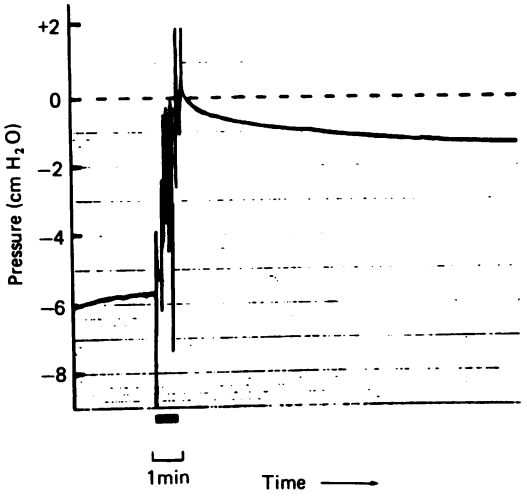

Fig. 3. Time course of intra-articular hydrostatic pressure after injection of 140 μl. Ringer solution through the articular cannula during the interval marked by a dark bar.

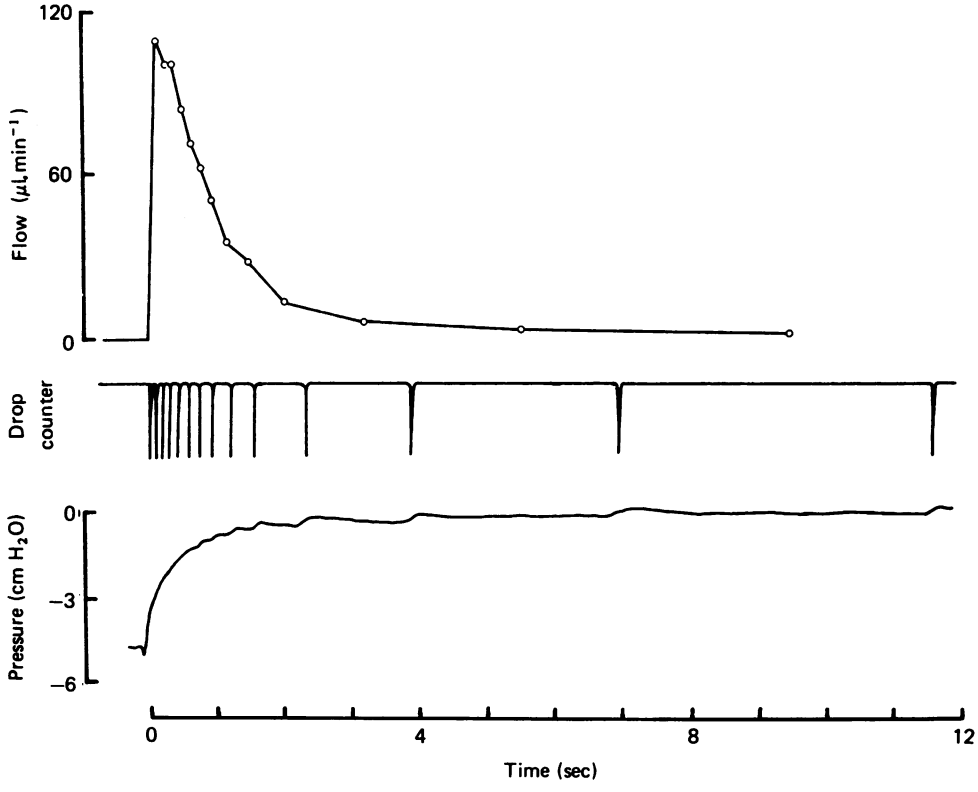

Fig. 4. Flow of Ringer solution into a joint cavity (upper trace) and co-incident joint fluid hydrostatic pressure (bottom trace) when the flow cannula was opened to atmospheric pressure at time zero. Middle trace shows drop counter signal. The small humps on the pressure trace were generated by the fall of each drop.

thetists to locate the epidural space, where pressure is also subatmospheric (Bengis & Guyton, 1977.) By connecting the second cannula to a drop counter, a record of inflow under atmospheric pressure was obtained (Fig. 4). Typically, 120 μl. Ringer solution flowed rapidly into the synovial cavity on connecting the second cannula to the atmosphere, causing synovial fluid pressure to rise from $-4{\cdot}6$ cm H_2O to zero pressure over 5 sec.

(4) The quantitative validity of the perforated cannula measurements was assessed by comparing them with measurements of pressure in six other knee joints by the wick-in-needle technique. The distribution of pressure recorded by this technique is shown in Fig. 2. Mean wick pressure was $-4{\cdot}0$ cm H_2O (range $-1{\cdot}0$ to $-8{\cdot}2$ cm H_2O). These values were not significantly different from those obtained by the perforated cannula technique (Student's t test, $P > 0{\cdot}1$).

Influence of joint angle and fluid volume on pressure. The relationship between joint angle and the pressure of synovial fluid for six different joints is plotted in Fig. 5. The extent to which pressure varied with joint angle depended very much on the magnitude of the pressure. When synovial fluid pressure was low (curves *A*, *B*) pressure changed by 1 cm H_2O or less on moving from extension to maximal flexion or vice versa. When pressure was closer to atmospheric (curve *F*), moderate flexion from 160 to 120° again produced only a small increment in pressure but acute flexion to less than 80° produced a relatively steep increase in pressure to $+7{\cdot}5$ cm H_2O. A similar relationship was obtained by extending the acutely flexed joint. The over-all relationship between synovial fluid pressure and joint angle was curvilinear. Except in one case (curve *F*) a 'position of minimum pressure' (angle above or below which pressure increased) was not observed in these normal joints, in contrast to the relationship observed in inflamed human joints by Eyring & Murray (1964).

Because the pressure of fluid in a closed container depends into alia upon the volume of fluid, it seemed possible that the sensitivity of pressure to joint angle depended on the volume of synovial fluid, relatively 'dry' joints being insensitive to joint angle and relatively 'wet' joints more sensitive. This seemed to be the case, for when an insensitive joint (curve *D*) was converted to a joint of larger volume by the infusion of 200 μl. Ringer solution, fluid pressure became much more sensitive to joint angle (curve *G*). This effect could be rendered even more striking by increasing the size of the simulated joint effusion. Thus the joint which displayed least sensitivity to angle (curve *B*) showed the greatest sensitivity (curve *H*) after the introduction of 950 μl. Ringer solution into the cavity, pressure reaching $+30$ cm H_2O in acute flexion.

The pressure of synovial fluid during spontaneous, voluntary joint motion was not recorded. Repeated passive flexion and extension of the joint between 75 and 110°, however, produced rhythmical increases and decreases in pressure, in five out of five joints with extension pressures greater than -5 cm H_2O. Similar changes were observed when active flexion of the same joint was evoked reflexly from 110° extension (flexion angle not known) by pressure stimulation of the ipsilateral Achilles tendon (Fig. 6). It seemed likely therefore that synovial fluid pressure would oscillate during voluntary motion of such joints. Joints with pressure of -7 cm H_2O or lower showed little sensitivity to dynamic alterations in joint angle ($n = 3$), as might be expected from Fig. 5.

Influence of periarticular tissue tension on synovial fluid pressure. The hydrostatic

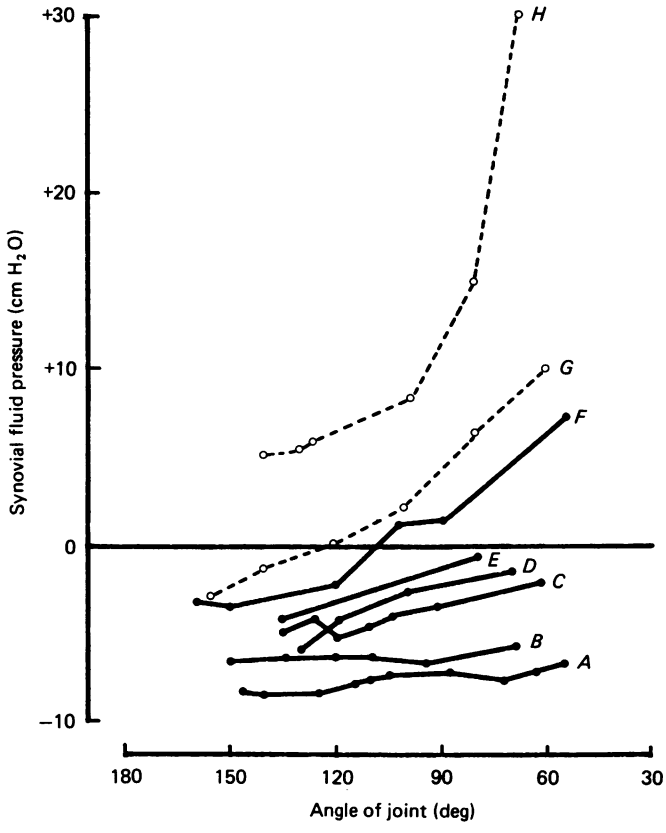

Fig. 5. Effect of static joint angle on synovial fluid hydrostatic pressure in six knee joints. Curves *A–F*: normal joints. Curve *G*: 200 μl. Ringer solution introduced into same joint as curve *D*. Curve *H*: 950 μl. Ringer solution introduced into same joint as curve *B*.

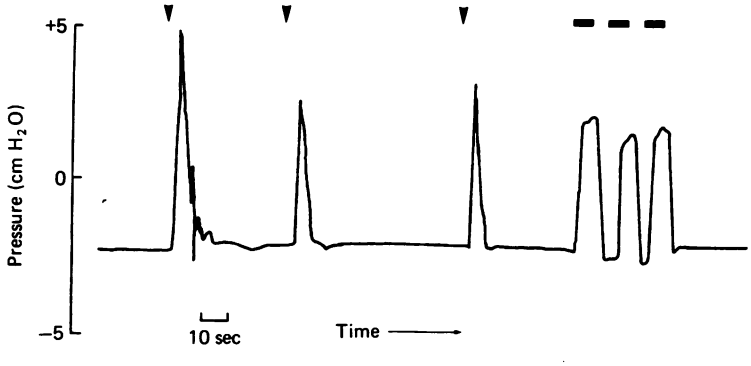

Fig. 6. Changes in synovial fluid hydrostatic pressure induced by active or passive movements of the knee joint. At each arrow the ipsilateral tendo-Achilles was stimulated to evoke a single reflex withdrawal of the limb with flexion of the knee joint. At each bar the joint was flexed passively from 110 to 75°.

pressure of synovial fluid depended not only on joint angle and synovial fluid volume, but also on the pressure being exerted upon the joint capsule by the overlying muscles and ligaments. When the tension in these soft tissues was increased by acute flexion of the ankle joint, fluid pressure in the knee increased by over 2 cm H_2O ($n = 3$), the knee itself being immobilized at 110° (Fig. 7).

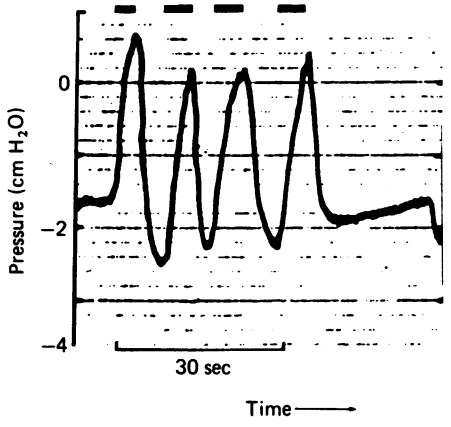

Fig. 7. Changes in synovial fluid hydrostatic pressure of the knee during flexion (dark bars) of the ipsilateral ankle joint. The angle of the knee joint was held constant at 110° throughout.

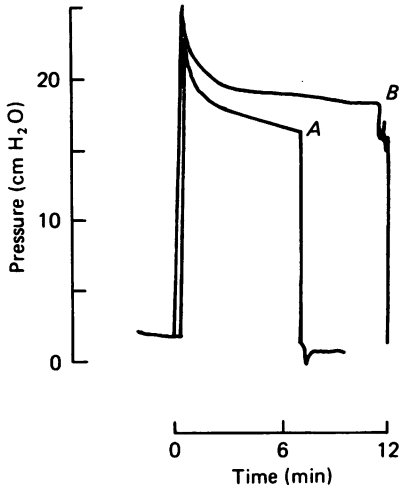

Fig. 8. Changes in joint fluid hydrostatic pressure during two consecutive periods of flexion from 115 to 55°. Traces superimposed to facilitate comparison of slopes. Curve *A*: joint fluid volume enhanced by 200 μl. Ringer solution. Curve *B*: Ringer solution aspirated and 200 μl. light-grade paraffin oil substituted.

Promotion of synovial fluid absorption by joint flexion. When a joint containing an increased volume of fluid (200 μl. Ringer solution) was acutely flexed, fluid pressure was elevated from +1 cm H_2O (115°) to +20 cm H_2O (55°). This high pressure was not maintained, but fell progressively with time (three joints) as illustrated by curve *A* of Fig. 8. On restoration of the joint angle to 115° several minutes later, the pressure was smaller than formerly.

It seemed possible that the fall in fluid pressure was caused by a pressure-induced efflux of fluid from the joint cavity into the surrounding tissue. An alternative explanation however presented itself, namely that the compliance of the joint increased with time while fluid volume remained constant. The flexion experiments were repeated therefore on the same joints after aspiration of the joint fluid and its replacement by 200 μl. light-grade paraffin oil. The latter fluid is unable to leave the joint cavity, because the synovial membrane is impermeable to paraffin oil (Levick, 1979). As is shown by curve *B* of Fig. 8, although pressure still declined with time in the oil-containing flexed joint, the rate of decline was over twice as fast for the Ringer-containing joint as for the oil-containing joint (0·33 and 0·14 cm H_2O min^{-1} respectively at 4 min). It appeared therefore that a substantial part of the decline in fluid pressure in an acutely flexed joint was caused by trans-synovial absorption of the joint fluid.

DISCUSSION

The measurements obtained by the perforated cannula technique confirm previous reports that the hydrostatic pressure of synovial fluid in extended mammalian knee joints is subatmospheric. The mean and range reported here are similar to those obtained by a simple needle technique by Müller, 1929 (-3 to -12 cm H_2O; dog and man) and by Reeves (-2 to -10 cm H_2O; dog, cat, rabbit, man). They are lower than values obtained recently by Jayson & Dixon (1970*a*) ($+3$ to -2 cm H_2O, man) using Braun cannulae, but a central needle must be withdrawn after insertion of such cannulae so the joint cavity was presumably open to the atmosphere for a short time. This may have allowed an influx of fluid from cannula into the joint cavity (cf. Fig. 5) making synovial pressure less subatmospheric than normal. Small regular oscillations in the pressure of synovial fluid coincident with the arterial pulse have been described previously in joints containing effusions (McCarty, Phelps & Pyenson, 1966) and were observed here in some normal joints, but the amplitude of oscillation was considered to be sufficiently small to justify the description of synovial fluid pressure by a single value. Although these fluid pressures were obtained in the non-load bearing joint, they probably exist also in the loaded joint (outside the weight-bearing articular surfaces), for both Müller (1929) and Jayson & Dixon (1970*a*) observed similar fluid pressures in unloaded and loaded knees.

Subatmospheric synovial fluid pressures: fact or artifact? Caughey & Bywaters (1963) proposed that subatmospheric pressure recordings from joint spaces might be artifacts caused by obstruction of the recording cannula. Both the construction of the cannulae and a number of experimental observations preclude this explanation. Neither the small oscillations in pressure, nor the pressure increment resulting from fluid displaced from the posterior pouch would be detected by a blocked cannula; nor would the pressure trace revert to a subatmospheric value following a small fluid infusion if the cannula were obstructed. Perhaps the most convincing observation, however, was the rapid flow of Ringer solution into the joint when a cannula was opened to the atmosphere, for influx could occur only if a pressure gradient existed along the cannula.

A second potential error arises from the pressure of the surrounding solid tissues

on the fluid in the needle tip (Guyton, Granger & Taylor, 1971). This error is slight if sufficient time is allowed for needle and tissue fluid pressures to equilibrate (Brace, Guyton & Taylor, 1975), a condition fulfilled in the present experiments. Moreover, the cotton wick method circumvents the problem of solid tissue pressure (Scholander *et al.* 1968) and yielded values which were similar to those obtained by the perforated cannula technique.

A further criticism might be that the cannula itself created a negative pressure by deforming (expanding) the joint cavity. The highly negative pressure observed immediately after cannulation was indeed attributed to deformation of the synovial cavity around the cannula tip, but the subsequent equilibration at a less negative pressure demonstrated that the fluid around the tip was in continuity with that in more distant undeformed regions. Moreover it has been shown that hypodermic needles of less than 1 mm diameter give a faithful record of fluid pressure in the pleural cavity, a potential space similar to the joint cavity (Agostoni, 1972).

It is reasonable to conclude that the subatmospheric synovial fluid pressures recorded in healthy extended mammalian knees are not artifactual.

Comparison with other extravascular fluid pressures. Joint fluid pressures are similar in sign and magnitude to connective tissue interstitial fluid pressures. Estimates of the latter range from $-6{\cdot}5$ cm H_2O (Guyton, 1963) to $-2{\cdot}8$ cm H_2O (Snashall *et al.* 1971). This similarity supports the concept of Bauer, Ropes & Waine (1940) that the joint cavity is fundamentally a very large but otherwise relatively unspecialized connective tissue space. The similarity also supports the hypothesis that synovial fluid is produced by passive ultrafiltration across the walls of synovial capillaries, in that interstitial fluids are known to be generated by this process, whereas actively secreted fluids such as cerebrospinal fluid have pressures above atmospheric.

Origin of the subatmospheric pressures. Surprisingly, the means by which a subatmospheric synovial fluid pressure might be generated and sustained have received little discussion in the literature. The simplest explanation would be that the joint capsule is in a state of tension and is pulling upon the synovial fluid. This explanation seems inadequate, because the synovial membrane is permeable to fluid (Levick, 1978); extra-synovial tissue fluid would be drawn into the joint cavity until capsular tension became zero. It seems likely that capsular tension can only create transient negative pressures, such as occur during active locomotion (Jayson & Dixon, 1970*b*).

To create a sustained subatmospheric pressure in a permeable container, a sustained force must tend to withdraw fluid from the container. Such a suction force could be created in the knee by either the lymphatic system or plasma colloid osmotic pressure. Smith & Campbell (1928) showed that alternate compression and expansion of the lymphatic plexus of the synovium by joint motion resulted in a withdrawal of fluid from the joint cavity into the lymphatics. Whether a negative pressure can be established by this motion-dependent lymphatic pump is unproven. On the other hand Guyton *et al.* (1971) suggest that negative interstitial fluid pressures arise because the colloid osmotic pressure difference across the capillary wall exceeds plasma hydrostatic pressure, i.e. the capillaries exert a net absorption pressure.

Several considerations indicate that a net filtration pressure rather than an absorption pressure exists from plasma to synovial fluid.

(1) By subtracting from rabbit plasma colloid osmotic pressure (maximum 25 cm H_2O, Levick, 1979) the likely opposing synovial colloid osmotic pressure (12–25 cm H_2O in human and cattle fluid, Jensen & Zachariae, 1959; Bauer *et al.* 1940) and mean synovial fluid by hydrostatic pressure ($-4{\cdot}6$ cm H_2O), the maximum value for synovial capillary hydrostatic pressure compatible with net absorption is estimated to be $+8{\cdot}4$ cm H_2O. Since saphenous and femoral venous pressures were 8–10 cm H_2O in the supine rabbit (unpublished observations) it seems probable that mean synovial capillary pressure is greater than 8·4 cm H_2O. Thus unless rabbit synovial colloid osmotic pressure is considerably smaller than that in cattle and man, a net filtration pressure exists across the synovial capillary wall. A similar argument applies even more forcefully to human knees, for these lie approximately 70 cm below heart level in orthostasis and hence their synovial capillary pressures must often exceed 70 cm H_2O (Levick & Michel, 1978). An effect of gravity on trans-capillary filtration into human knees may help explain the less negative synovial fluid pressures observed by Jayson & Dixon (1970*a*).

(2) In the immobilized rabbit knee, fluid pressure rose slowly towards atmospheric pressure at a rate of $+1$ to $+2$ cm H_2O/hr, indicating a slow increase in synovial fluid volume. The pressure rise is calculated to correspond to the net filtration of 20–40 μl. fluid into the joint per hour, joint space compliance at subatmospheric pressures being 20 μl. cm H_2O^{-1} (unpublished observations). No data could be found in the literature for comparison with this estimate of the minimum rate of synovial fluid formation, but the pressure rise is intriguingly similar to that observed in the interstitial fluid of immobile cat mesentery (Fraser, Smaje & Verrinder, 1978; Clough & Smaje, 1978).

(3) In five out of seven animals pressure was more negative in the knee which had been immobile for the shorter period. Although these differences did not achieve statistical significance, they are again compatible with the hypothesis that a net filtration occurs from the synovial microcirculation into the joint space and that removal of this fluid requires the operation of a motion-augmented pump, probably the lymphatic system.

Structural implications of subatmospheric synovial fluid pressures. Synovial fluid is usually represented as a thin but continuous film of fluid separating the 'parietal' and 'visceral' layers of synovium. This representation may be questioned in view of the subatmospheric nature of synovial fluid pressure. Since the luminal aspect of the parietal synovium is acted upon by a subatmospheric pressure of $-4{\cdot}6$ cm H_2O, a continuous sheet of fluid could only exist if the tissues around the joint exerted a traction of $+4{\cdot}6$ cm H_2O upon the abluminal surface of the parietal synovium. The magnitude of any traction or recoil pressure is unknown. If the recoil pressure is less than $+4{\cdot}6$ cm H_2O, the difference in pressures will cause the parietal synovium to collapse down upon the visceral synovium, making many points of solid contact with it. In this event synovial fluid will exist not as a uniform sheet of fluid but as a series of pools or rivulets separated by pillars of solid tissue, the contact points. Such a physical state might have important implications in the study of both inter-articular and inter-synovial lubrication.

This concept of the possible physical organizations of synovial fluid is analogous to concepts developed previously by Agostoni (1972) and Guyton *et al.* (1971) for pleural and interstitial fluids respectively.

Importance of the influence of joint angle on fluid pressure. The sensitivity of fluid pressure to joint angle depended on the intrinsic pressure or volume. This observation went some way towards reconciling the apparently contradictory observations of Reeves (1966), who observed low pressures which were insensitive to joint angle, and Jayson & Dixon (1970*b*) who obtained less negative pressures which were markedly sensitive to joint angle. In view of the marked effect of soft tissue pressure on fluid pressure (Fig. 7) it seemed possible that increases in fluid pressure during flexion of the joint were partially due to the effects of increased muscle and ligament tension. This suggestion is in accord with evidence presented by Reeves (1966).

The relationship between joint angle and synovial fluid pressure may be of considerable physiological importance, because the rate of absorption of fluid from a joint cavity varies with pressure (Levick, 1978). Increases in fluid pressure during crouching or active use of a joint may simulate a pump which drives fluid out of the joint cavity into the sub-synovial tissue spaces (i.e. towards the lymphatics), and so maintains the volume of synovial fluid at a minimum. The decay of pressure in acutely flexed joints (Fig. 8) supported this pump concept. The trans-synovial pump might not operate in a 'favourable' direction in all joints and species however: for if muscles inserted around the joint capsule were to reduce fluid pressure transiently during movement, the pump might tend to increase synovial fluid volume, as indeed seems to occur in human knees after bicycling (Reeves, 1966).

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