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PHYSIOLOGICAL COMPARTMENTATION OF FLUID WITHIN THE SYNOVIAL CAVITY OF THE RABBIT KNEE

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SUMMARY

1. The relationship between pressure in the suprapatellar region (P_{sp}) of the synovial cavity of the rabbit knee, and volume of fluid (oil or saline) infused into that region displayed a pressure plateau (15·5 cm H_2O) between 1·2 and 2·0 ml.
2. No pressure plateau occurred when fluid was infused simultaneously into the suprapatellar and posteromedial regions of the synovial space.
3. Pressure in the posteromedial region (P_{pm}) did not respond to suprapatellar fluid infusions until onset of the suprapatellar pressure plateau. During the plateau phase, P_{pm} rose steadily towards P_{sp}. At the end of the plateau phase the two pressures were almost equal and rose in parallel.
4. The plateau phenomenon also occurred during aspiration of volume-expanded joints; and was present at all joint angles.
5. It was concluded that the joint space, although anatomically continuous, is divided into two hydraulically separate compartments at physiological pressures.
6. The sites of communication between the two compartments at pathological pressures were explored by casts of the synovial cavity.

INTRODUCTION

The relationship between fluid volume and pressure in a synovial cavity is one of several factors influencing trans-synovial flow and volume homeostasis. In a previous study it was found that the pressure–volume curve was sigmoid for the synovial cavity of the rabbit knee (Knight & Levick, 1982). In those experiments fluid was infused into the synovial cavity at two sites, one anterior and one posterior, in order to ensure an even distribution of fluid throughout the space. This was necessary because, as was discovered at the beginning of the study, a pressure–volume curve of different shape was obtained when only one infusion site was employed. Dependence of the shape of the curve on the infusion site seemed incompatible with the conventional view of the synovial cavity as a single, hydraulically continuous space. The observation therefore excited the present investigation.

This paper describes the synovial pressure–volume curve obtained with a single infusion site; and experiments to find an explanation for this unusual curve. A preliminary account of some of the findings has been given to the Physiological Society (Knight & Levick, 1981).

METHODS

Pressure and volume measurements

These have been described previously in detail (Knight & Levick, 1982). In brief, the knee of a supine, New Zealand rabbit under pentobarbitone–urethane anaesthesia was held at a measured angle by ankle ties; the joint was extended (110–140°), except where otherwise specified under Results. After incision and reflexion of the skin over the anterior and medial aspects of the knee,

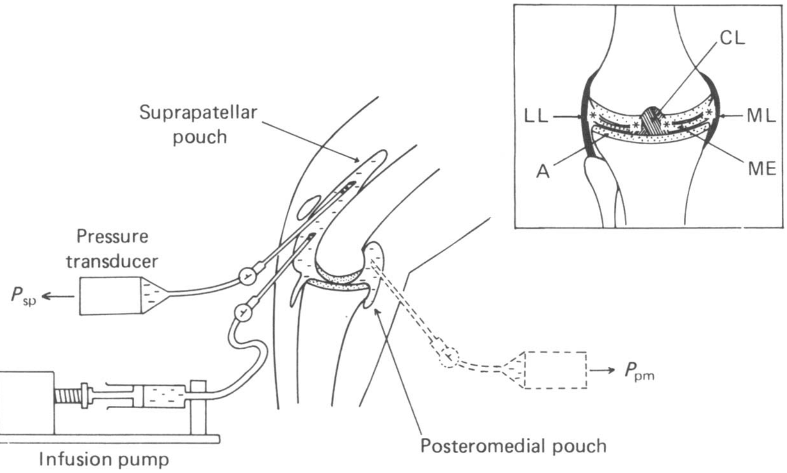

Fig. 1. Schematic representation of the experiment. The right knee joint is shown in oblique section in the main diagram. Pressure was measured in the suprapatellar pouch (P_{sp}) and also in some cases in the posteromedial pouch (P_{pm}). The inset diagram shows a coronal section through the knee to illustrate the principle anatomical structure: A = articular cartilage; ME = meniscus; LL = lateral ligament; ML = medial ligament; CL = cruciate ligaments, occupying the intercondylar gap of the femoral epiphysis. The four asterisks indicate the sites of the channels of communication between anterior and posterior regions of the joint cavity (see Results).

up to four steel 21-gauge cannulae were inserted into the joint cavity and secured by purse-string sutures. Two of the cannulae were inserted into the suprapatellar region of the synovial cavity (Fig. 1). One of these was connected to a pressure transducer level with the joint; transducer output was recorded on a two-channel potentiometric recorder (± 0.5 cm H_2O). The second suprapatellar cannula was connected to an infusion pump, by means of which the volume of the synovial cavity was increased in steps of 92–200 μl., at regular intervals of between 1 and 8 min. This period allowed fluid distribution throughout the available space. Since pressure decayed slowly with time, owing to stress relaxation of the cavity walls, standardization of the time between volume increments within an experiment was important. Suprapatellar pressure at the end of each period was plotted against volume.

In some experiments a third cannula was inserted, into the posteromedial region of the joint space, and was connected to a second pressure transducer to record posteromedial fluid pressure. Also in a few experiments a fourth cannula was inserted, again into the posteromedial region, and was connected to the infusion pump. The number of cannulae and their locations in a given experiment are specified in the Results section.

The infusate was either an inert oil (75 % Dow Corning silicone oil 200/0·65 c.s. and 25 % liquid paraffin B.P.) of low viscosity (1·8 centipoise or mPa . s); or else a physiological salt solution, either Krebs solution or normal saline. The low-viscosity oil mixture was preferred for most experiments, since it is not absorbed from the joint cavity (Levick, 1979; Knight & Levick, 1982): hence the volume inside the joint equalled the known infused volume. This was not so for saline solutions, which were readily absorbed by the synovial tissue lining the joint space.

Casts of the synovial cavity

The morphology of the cavity of the knee joint is complex, and proved to be relevant to this investigation. Although the space is anatomically continuous, the large anterior region (principally suprapatellar) is potentially separated from the smaller posterior region by closely opposed articular cartilages, menisci and ligaments (Fig. 1). In order to demonstrate the principal channels of communication across this central barrier, resin casts were made of the joint space. Acrylate resin (10 g, Trylon CL 201 PA) was mixed with thinner (2 ml., Monomer C), activator (0·3 ml., Accelerator E), catalyst paste (1·2 g) and pigment, and injected at 22–24 °C into the suprapatellar space of the knee. Some joints were flexed and extended ten times by hand immediately after the injection; others were left untouched. The animal was killed and the resin allowed to set. The hind limb was removed, and the tissues macerated in concentrated sodium hydroxide solution or hydrochloric acid to liberate the cast. Twelve resin casts were made of volume 1–3·5 ml. at joint angles between 120° and 90°. One latex rubber cast of 4 ml. was also made for an extended joint.

RESULTS

The pressure–volume curve for a single infusion site

Fig. 2 shows an example of the pressure–volume relationship observed in the extended knee (120°) when low-viscosity oil was infused into the suprapatellar region. The curve resembled in several respects the relationship reported previously for dual infusion sites (Knight & Levick, 1982). Initially pressure was subatmospheric, and pressure rose steeply with volume at subatmospheric pressures; the curve became less steep just above atmospheric pressure; and steepened again at high pressures. However, an additional feature, a pressure plateau, was observed at supra-atmospheric pressures with the single infusion site; this feature was not present with dual infusion sites. As volume and pressure increased, there came a point at which a volume increment failed to produce a sustained pressure increment: indeed pressure often fell slightly, by ~1 cm H_2O. The continuous pressure trace showed that although the volume increment produced a transient increase in pressure, this quickly decayed to its former level or even slightly lower (see inset, Fig. 2). This pattern repeated itself for several subsequent volume increments. Thus a plateau (or in many cases a small peak followed by a shallow trough) developed in the pressure–volume curve. The pressure plateau eventually terminated when volume increments again began to produce sustained increases in pressure.

For twenty-one joints the mean pressure at which the plateau occurred was 15·5 cm H_2O (S.E. of mean ±1·8 cm H_2O; range 5–28·5 cm H_2O). The plateau began at a mean volume of 1·2 ml. (S.E. of mean ±0·1 ml.; range 0·5–2·2 ml.); and ended at a mean volume of 2·0 ml. (S.E. of mean ±0·1 ml.; range 1·0–3·1 ml.). The pressure plateau thus persisted in spite of an average infusion of 0·8 ml. fluid – a substantial volume increase in such a small joint. There was no significant correlation between plateau pressure and time-averaged infusion rate, which ranged from 12·5 to 100 μl./min.

The pressure plateau was not caused by volume-induced reflex inhibition of periarticular muscle tone for it persisted when the limb was subjected to ring denervation at upper femoral level (one experiment).

Pressure gradients within the synovial cavity

The cause of the plateau phase became clear when pressure was measured simultaneously in both the posteromedial region and the suprapatellar region of the joint space, during oil infusion into the suprapatellar space (six joints). Initially, as the oil was infused, suprapatellar pressure increased. Simultaneous pressure in the posteromedial region, however, consistently showed no response to the anterior volume increments (Fig. 3*A*): there was no detectable fluid transfer between the two regions at low volumes and pressures, even after cumulative periods of 24 min. At the onset

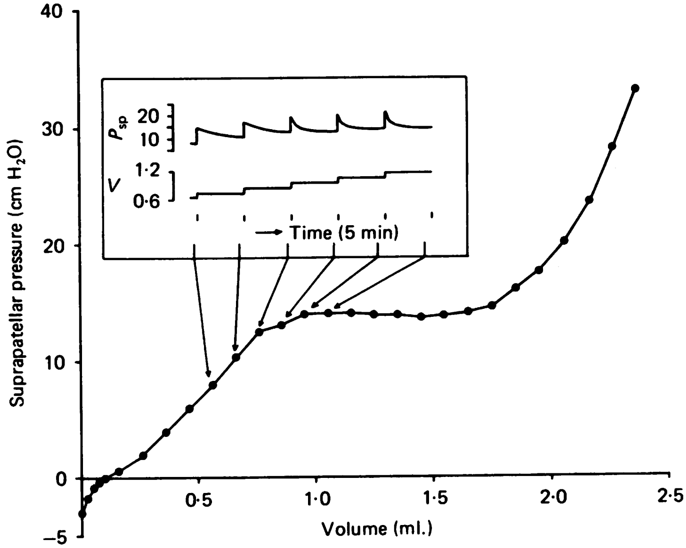

Fig. 2. Relationship between suprapatellar pressure (P_{sp}) and the volume (V) of low-viscosity oil infused into an extended rabbit knee. Fluid was infused through a single, suprapatellar cannula in aliquots of 0·1 ml. at 5 min intervals, and pressure at the end of the 5 min period was plotted (see inset). The curve shows a pressure plateau at 13–14 cm H_2O, extending from 0·75 ml. to 1·65 ml. The inset shows a section of the pressure record (P_{sp}) before and during the plateau. The slow decay in pressure with time prior to onset of the plateau is attributed to viscous creep of the cavity walls. At onset of the plateau, however, the shape of the pressure-decay curve changes: there is a steeper initial fall in pressure. This is attributed to flow into the relatively empty posterior region of the joint space.

of the suprapatellar pressure plateau, however, posteromedial pressure began to increase in response to suprapatellar infusions i.e. fluid transfer had begun. The plateau phase ended when posteromedial pressure had reached or almost reached suprapatellar pressure (Fig. 4). Thereafter both pressures continued to respond to infusions (Fig. 3*B*), and climbed in parallel (Fig. 4), the posteromedial pressure being usually a few cm H_2O lower than suprapatellar pressure. Thus the plateau of suprapatellar pressure was due to the infused oil flowing away from the suprapatellar region into the relatively empty posterior region of the joint space, through a newly-patent communication. The average pressure difference (ΔP) between the anterior and posterior region at commencement of this 'run-off' was 13·5 cm H_2O (S.E. of mean $\pm 4{\cdot}4$ cm H_2O; range 6·0–28 cm H_2O).

When fluid was subsequently aspirated in aliquots from the suprapatellar region (three joints), the suprapatellar pressure and posteromedial pressure declined together until a certain pressure was reached (Fig. 5). Thereafter, further withdrawal of suprapatellar fluid reduced suprapatellar pressure but not posteromedial pressure; the two regions had become hydraulically separated again. The entrapment of fluid in the posteromedial region created a second plateau, but this time a plateau of posteromedial pressure. These experiments demonstrated that the establishment of hydraulic communication across the joint cavity was a reversible phenomenon.

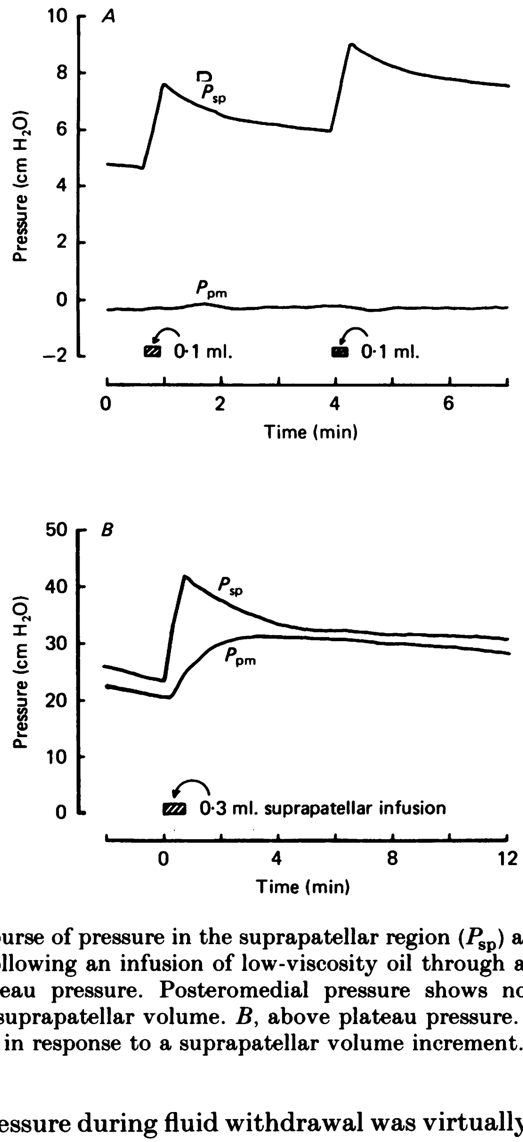

Fig. 3. Time course of pressure in the suprapatellar region (P_{sp}) and in the posteromedial region (P_{pm}) following an infusion of low-viscosity oil through a suprapatellar cannula. *A*, below plateau pressure. Posteromedial pressure shows no response to repeated increments of suprapatellar volume. *B*, above plateau pressure. Posteromedial pressure rises promptly in response to a suprapatellar volume increment.

The plateau pressure during fluid withdrawal was virtually the same as the plateau pressure during infusion, in all three joints. The pressure difference across the joint, however, was much larger at onset of the plateau during infusion (average ΔP 14·7 cm H_2O, $n = 3$) than at onset of the plateau during fluid withdrawal (average ΔP 4·5 cm H_2O, $n = 3$). Thus pressure *per se* rather than pressure difference across the joint space seemed to be the crucial factor determining hydraulic communication.

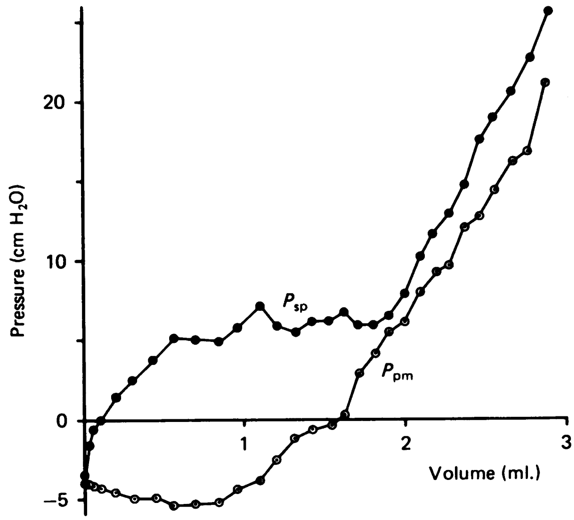

Fig. 4. Response of suprapatellar pressure (P_{sp}) and posteromedial pressure (P_{pm}) in the same extended knee to repeated 0·1 ml. infusions of low-viscosity oil at 3 min intervals through a single, suprapatellar cannula.

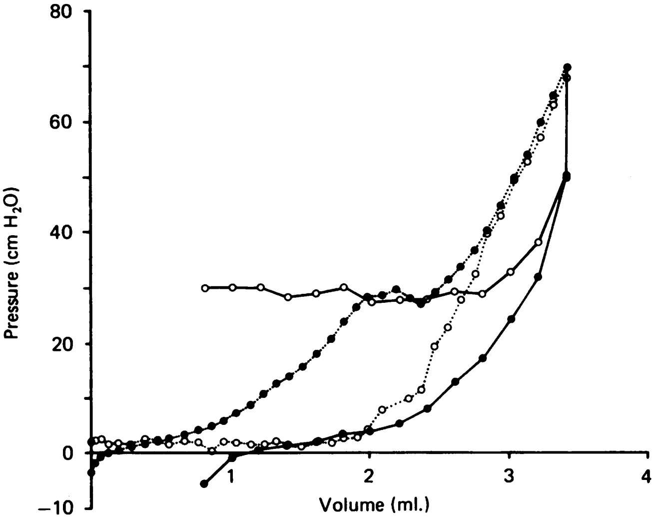

Fig. 5. Response of suprapatellar pressure (●) and posteromedial pressure (○) to fluid withdrawal from the suprapatellar region of the volume-expanded knee. Low-viscosity oil was first infused (dotted lines) through a suprapatellar cannula to a total volume of 3·4 ml. The fluid was then aspirated in aliquots; the resulting pressures are joined by continuous lines. Posteromedial pressure shows a plateau at 28–30 cm H_2O during fluid withdrawal: suprapatellar pressure plateaued at the same pressure during fluid infusion.

Pressure plateaux with aqueous infusates

It seemed possible that the hydraulic non-communication between anatomically continuous regions might be an artifact, created perhaps by a high surface tension at an oil–synovial fluid interface in mid-joint. To investigate this possibility the experiment was repeated with a physiological salt solution as infusate rather than

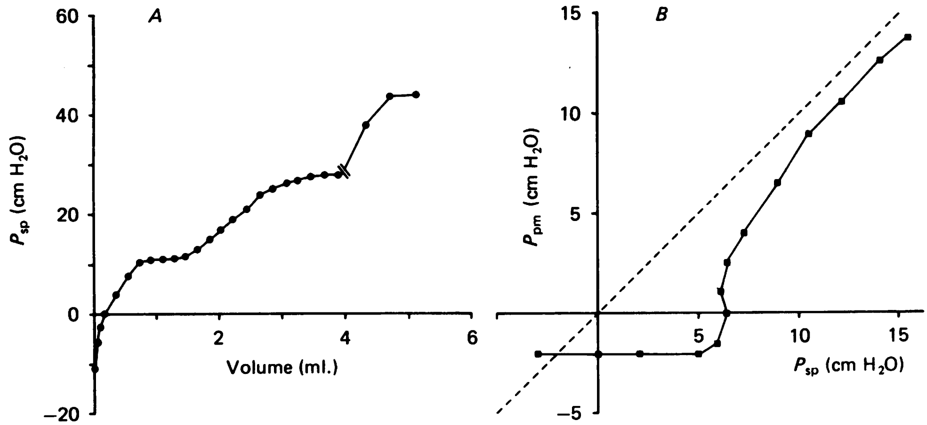

Fig. 6. The pressure response to the infusion of a saline solution into two extended rabbit knees. *A*, suprapatellar pressure (P_{sp}) when volume was increased by the infusion of normal saline into the suprapatellar pouch in aliquots of 0·2 ml. at 2 min intervals. A pressure plateau (11 cm H_2O) occurred between a volume of 0·7 ml. and 1·4 ml. A second plateau developed at 27·5 cm H_2O because absorption rate at this pressure equalled infusion rate. When infusion rate was doubled to 0·4 ml. every 2 min (break in curve), pressure rose again, until a third plateau developed at 44 cm H_2O. Since saline is absorbed by the synovial lining of the joint cavity, the abscissa only represents volume infused, which is greater than the volume within the joint cavity. *B*, relationship between suprapatellar pressure (P_{sp}) and posteromedial pressure (P_{pm}) in another knee whose volume was progressively increased by the infusion of normal saline into the suprapatellar region. P_{sp} displays a plateau at 6·5 cm H_2O. The dashed line through the origin has a slope of 1 and is the line of equality.

the low-viscosity oil. The aqueous fluid was infused through a single cannula into the suprapatellar region. Pressure was recorded in either the suprapatellar pouch alone (three joints) or in both suprapatellar and posteromedial regions (two joints). Relatively high infusion rates were used (200 μl. increments at 2 min intervals) because aqueous fluids infused at low rates can display pressure plateaux for a quite different reason *viz.* that trans-synovial absorption rate can equal infusion rate (Knight & Levick, 1982).

A pressure plateau, similar to that produced by oil infusions, was observed in all five joints infused with a saline solution (Fig. 6*A*). Mean plateau pressure was $10{\cdot}6 \pm 1{\cdot}53$ cm H_2O (range 6·5–15·0 cm H_2O). In the saline-infused joints, however, a second plateau subsequently developed at higher pressures. Such secondary plateaux arise with an absorbable fluid because the rate of absorption from the joint space grows with pressure until it equals the time-averaged infusion rate (Knight & Levick, 1982). In the two saline-infused joints in which both suprapatellar and posteromedial pressures were recorded, posteromedial pressure behaved as for oil infusions (Fig. 6*B*). It showed no detectable response to suprapatellar infusions below plateau pressure; gradually increased during the suprapatellar plateau; and increased by the same amount as suprapatellar pressure after termination of the plateau. These observations seemed to exclude the possibility that the plateau phenomenon was an artifact caused by oil.

Pressure plateaux during posteromedial infusions

The above experiments demonstrated that anterior infusions opened a channel into the posteromedial region. It seemed worthwhile to test whether hydraulic communication could also be created by a pressure gradient in the opposite direction, since this would establish whether the system was unidirectional (like a flap valve). Oil was therefore infused through a single cannula into the posteromedial region in two joints. Pressure was recorded simultaneously from both the posteromedial and suprapatellar regions. In each joint the pressure–volume curve for the posteromedial region resembled the curve in Fig. 2, and showed a plateau (at 13 and 7 cm H_2O respectively). Suprapatellar pressure showed no response to the increments of posteromedial volume until onset of the posteromedial pressure plateau. During the plateau, suprapatellar pressure gradually increased to within a few cm H_2O of posteromedial pressure. The plateau then ended and both pressures rose in parallel. The response to posteromedial infusions was thus closely analogous to the response to suprapatellar infusions, i.e. the properties of the channel of communication were symmetrical.

Abolition of pressure plateaux by dual infusion sites

The above experiments indicated the existence of two hydraulically separate regions of the joint cavity at physiological pressure; but they did not exclude the possible existence of more than two subcompartments. To investigate this possibility, either oil (three joints) or a saline solution (two joints) was infused simultaneously into the suprapatellar and posteromedial compartments. The suprapatellar and posteromedial infusion cannulae were connected to the same infusion line, so that fluid was infused at the same pressure into each region. Pressure was recorded in both the suprapatellar region and the posteromedial region through two additional cannulae.

Similar results were obtained in all five joints; an example is presented in Fig. 7. Posteromedial and suprapatellar pressures increased as sigmoid functions of volume. No pressure plateaux developed. Thus no additional subcompartments seemed to be present.

Influence of joint angle on fluid compartmentation

All the above experiments were carried out on extended joints (110–140°). To discover whether fluid compartmentation occurred only at such joint angles, two types of experiment were carried out: (1) determination of the pressure–volume curve in an immobile, flexed joint; and (2) determination of the curve in joints moved through a continuous range of motion.

(1) In one animal the pressure–volume curve for each knee was determined by the infusion of oil through a single, suprapatellar infusion site; suprapatellar pressure was

recorded. One knee was held stationary in a flexed position (80°); the other was extended to 138° as a control. The resulting pressure–volume curves are shown in Fig. 8. It is seen that both curves displayed a pressure plateau, i.e. fluid compartmentation was present in the flexed as well as in the extended position. The curves also show that flexion increased the pressure at which hydraulic communication

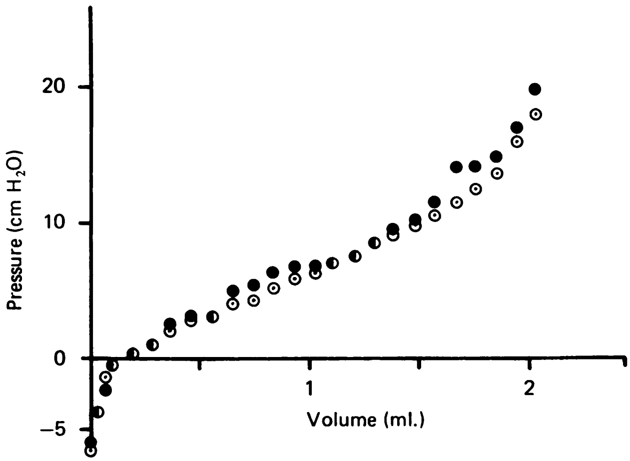

Fig. 7. Pressure responses to the infusion of low-viscosity oil simultaneously into the suprapatellar and posteromedial regions of the extended rabbit knee through two cannulae. Pressures in the suprapatellar region (●) and in the posteromedial region (○) were recorded via two other intra-articular cannulae.

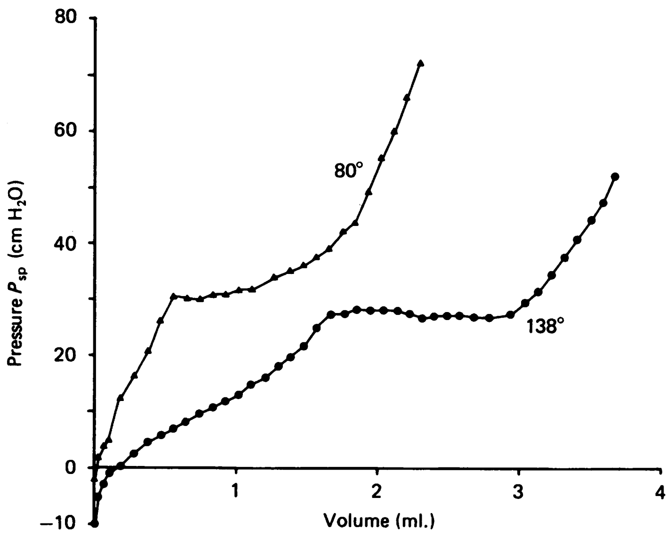

Fig. 8. Effect of joint angle on the plateau phenomenon. The two curves represent the two relationships between suprapatellar pressure and volume of low-viscosity oil, infused into either knee of one rabbit. One knee was immobilized in extension (138°) and the other in flexion (80°). Oil was infused through a suprapatellar cannula alone. Infusion rate was the same for each joint (0·092 ml. at 3 min intervals).

commenced, from 25 cm H_2O to 30 cm H_2O. The volume required to establish hydraulic communication was reduced by flexion, however, because flexion increases the slope of the pressure–volume relationship (Knight & Levick, 1982).

(2) The possibility remained that there might be intermediate angles at which the two regions were normally in hydraulic communication. Three joints were therefore investigated over a full range of motion. Again oil was infused through a single suprapatellar cannula and suprapatellar pressure was recorded. After each volume increment the joint was flexed and extended slowly by hand five times, then returned to its control angle for a pressure measurement to be taken. One joint was flexed to 75° from a control angle of 100°; the other two were extended to 150° from a control angle of 75°. In all three joints a pressure plateau occurred. The plateau pressures were higher in the initially flexed joints (19 and 22 cm H_2O) than in the initially extended joint (5 cm H_2O), as expected. The presence of a plateau in each case showed that the anterior and posteromedial regions did not communicate hydraulically, at physiological volumes, at any angle between 150° and 75°.

Anatomical location of the channels of communication

Casts of the joint cavity were obtained for ten extended knees (120°) and three flexed knees (90°). The injected volume of casting material (1·0–4·0 ml.) was sufficient to obtain casts of the channels of communication. After maceration of the animal tissue, six intact casts were obtained (three flexed, three extended): the remainder fragmented because of the thinness of the channels connecting anterior and posterior regions. The fragmented specimens were weighed: from these weights the relative volumes of the various compartments of the joint cavity were determined.

Two views of an intact 3·5 ml. acrylate resin cast are shown in Figure 9. Four main volumes may be recognized: (1) a large anterior plate, comprising supra- and infrapatellar regions; (2) the synovial sheath curled around the head of extensor digitorum longus; (3) a posterolateral hollow hemisphere cupped around the lateral femoral condyle; and (4) a smaller, posteromedial hollow hemisphere cupped around the medial femoral condyle. The posteromedial region made direct contact with the anterior infrapatellar region by three thin channels. One passed under (lateral to) the medial ligament of the joint, and was very thin (0·3–0·5 mm × 0·4–0·6 mm). The other two channels took intercondylar routes, and wound around the cruciate ligaments; again the channels were very thin (0·4 mm × 0·7–1·2 mm). Dissection of an oil-injected joint indicated that the intercondylar channels pass respectively lateral and medial to the pair of cruciate ligaments i.e. round the outside of the ligaments rather than in between them (see inset, Fig. 1). The posteromedial region of the cast was almost completely separated from the posterolateral region by a deep groove, but a very thin, short channel connected the two across the base of the groove. The posterolateral region was in contact with the anterior compartment by a more substantial channel (1·2–1·7 mm × 0·5–1·3 mm) which passed under (medial to) the lateral ligament. A striking negative observation was that no sheets of resin ever extended through the intercartilaginous plane between the menisci, tibial and femoral articular cartilages i.e. the casts did not demonstrate any fluid communication through the plane. This contrasted with the conventional textbook diagram which depicts a continuous sheet of fluid throughout the interarticular plane.

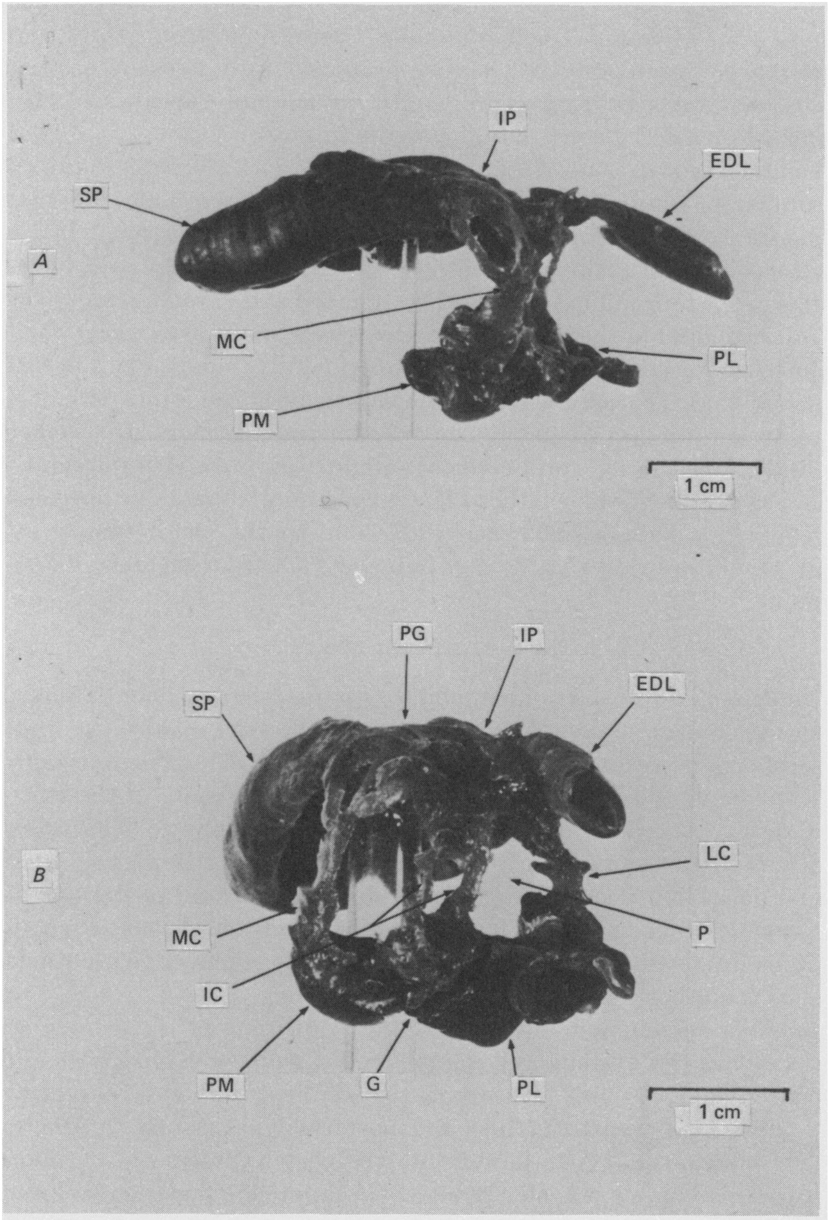

Fig. 9. Two views of an intact, 3·5 ml. acrylate-resin cast of the cavity of a rabbit knee. *A*, lateral view. The femur, which was removed by maceration, would be to the left of the picture; the tibia to the right. *B*, axial view, slightly oblique, looking at the cast from its tibial aspect. SP = suprapatellar pouch: the impression of the quadriceps muscle is visible; IP = infrapatellar region; PG = patellar groove; EDL = prolongation of joint space around head of extensor digitorum longus; PM = posteromedial pouch; PL = posterolateral pouch; G = deep groove separating the two posterior pouches; MC = medial channel; IC = intercondylar channels around the cruciate ligaments; LC = lateral channel; P = plane between femoral and tibial articular surfaces.

The above description applied to casts from extended joints, whether moved immediately after injection or left immobile. Casts from flexed joints were similar, except that each intercondylar channel branched into two (occasionally three) thinner channels for a portion of its length, presumably because of the different positions or tensions of the cruciate ligaments in flexion.

For seven fragmented casts of volume between 1 and 4 ml., from joints at 120°, the anterior compartment constituted 58·6 ($\pm$2·0) % of the total volume; the posterolateral compartment formed 24·0 ($\pm$2·1) %; the posteromedial compartment 14·3 ($\pm$1·0) %; and the extensor digitorum longus sheath 2·0 ($\pm$0·5) %. These volumes of distribution were similar in the four joints which had been flexed and extended immediately after acrylate injection and in the three joints which were not moved. Also, the fractional distribution of material between the various regions was independent of total volume between 1 and 4 ml. To obtain comparable values for flexed joints, two of the flexion casts were broken up and weighed. The anterior compartment was slightly smaller (50·6 %) and the posterior compartments slightly larger (posterolateral = 26·1 %: posteromedial = 20·1 %) than in the extended position. The significance of these small changes could not be assessed statistically due to the small sample size, but if significant, would indicate that flexion pumped fluid from the anterior to posterior compartment.

DISCUSSION

Posteromedial pressure did not respond to suprapatellar infusions at low pressures, even when cumulative periods of up to 24 min had elapsed since the first infusion. The failure of posteromedial pressure to respond after such long periods indicates that no significant flow occurred between the two regions, in spite of the existence of a pressure gradient; the resistance to flow was effectively infinite. This observation is incompatible with the conventional view that the joint cavity is a single, hydraulically continuous space. It is concluded that the suprapatellar and posteromedial regions are not normally connected by a low-resistance, fluid-filled space, even though the regions are anatomically continuous (i.e. a seeker can be passed, with difficulty, from one space to the other without broaching any tissue).

The plateau phenomenon represented the transition phase from a two-cavity system to a one-cavity system, and is explained as follows. Each infusion during the plateau caused suprapatellar pressure to rise above a critical level which caused a transient opening of a channel into the posteromedial pouch. A fraction of the pressurized, anterior fluid then flowed into the posteromedial pouch, and increased its pressure. This volume transfer reduced suprapatellar pressure, however, and so caused the channel to close again before anterior and posteromedial pressure had equilibrated. This process repeated itself until the anterior–posterior pressure difference became negligible. An alternative explanation of the suprapatellar pressure plateau may be considered: that the plateau represents a period during which endogenous synovial fluid is being absorbed from the joint cavity. This explanation is dismissed on the grounds that (1) it does not explain the changes in posteromedial pressure and (2) the volume over which the plateau extends (0·8 ml.) is far greater than the volume of synovial fluid in a rabbit knee.

The above two-compartment explanation is supported by previous evidence of compartmentation. Evans Blue solution infused anteriorly into the rabbit knee at 5 cm H_2O, does not reach the posteromedial pouch; but at 22 cm H_2O, the posteromedial pouch is filled (Levick, 1979). The present experiments with two infusion sites (Fig. 7) indicate that no more than two discrete compartments exist. Thus the posterolateral pouch must be hydraulically continuous with either the posteromedial pouch or with the anterior compartment, at pressures below plateau level. But with which? The earlier Evans Blue study indicated a connection with the anterior compartment below plateau pressure. But in the present experiments the average volume absorbed during the plateau phase (0·8 ml.) was 40 % of the total volume infused by the end of the plateau: this is greater than posteromedial volume alone (14·3 %) and corresponds closely to the combined volume of the posterolateral and posteromedial pouches (38 % of total volume). This implies that the posterolateral pouch, like the posteromedial pouch, is separated from the anterior region until onset of the plateau. The matter could be resolved in a future study by simultaneous pressure measurements in all three regions of the joint space.

Fluid compartmentation in other joints

It is not clear whether fluid compartmentation is unique to the rabbit knee. No plateau occurs in the pressure–volume curve for the dog knee (McCarty, Phelps & Pyenson, 1966). Most published pressure–volume curves for the human knee do not display a plateau (e.g. Jayson & Dixon, 1970*a*; Myers & Palmer, 1972): but at least one does. In Fig. 3 of the paper by DeAndrade, Grant & Dixon (1965) a suprapatellar plateau at ~20 mmHg occurred over a volume increment of ~20 ml. The cavity of the human knee is anatomically similar to that of the rabbit (i.e. anterior, posteromedial and posterolateral regions are recognizable), so the explanation of the plateau could be similar; unfortunately there seem to have been no pressure measurements in the posterior region of the human knee. Fluid compartmentation, albeit of a rather different type, has been clearly demonstrated in a pathological condition of the human knee: popliteal (Baker's) cysts are anatomically continuous with the posterior joint space, but can contain fluid at a different pressure to that in the suprapatellar space (Jayson, 1968; Jayson & Dixon, 1970*b*). Unlike the communication across the rabbit joint, however, the communication between the human joint space (suprapatellar region) and popliteal cyst is unidirectional. It would be of interest to measure pressure in the intervening posterior pouch in such cases.

Mechanism of opening and closure of the communication

For hydraulic non-communication to occur in an anatomically-continuous space, some obstruction to flow must exist. Since the obstruction can be overcome by pressure, it may be termed a valve. The experiments provided three clues to the nature and properties of the valve.

(1) The resin casts showed that, when the valve was open, three narrow channels passed between anterior and posteriomedial regions. Each of these channels wound under a thick, tense ligament (medial, anterior cruciate, and posterior cruciate ligaments), and bore the impression of the ligament. (2) Flow could occur from the posteromedial to the anterior region, as well as vice versa. Thus the channel of communication once open, was bidirectional, and did not have the properties of a

flap or ball valve. (3) The opening of the channel depended primarily on pressure, rather than on the pressure difference across the valve. These observations led to the view that the channels of communication are closed at low pressures because the normal (90°) stress exerted by the tense ligaments upon the channels exceeds the opposing fluid pressure: a channel opens when fluid pressure in either region exceeds normal stress due to the ligaments. (The situation may be likened to the opening of a collapsed blood vessel once intraluminal pressure exceeds extramural stress.) This mechanism could explain the observation that the pressure required to open the channels (plateau pressure) was increased by flexion of the joint: flexion presumably increases ligament stress.

Ogston & Stanier (1953) and later Roberts (1971) have demonstrated that synovial fluid in very thin films does not behave as a fluid but can withstand a considerable force without flowing. Therefore the question arises whether the barrier to flow might be a very thin film of synovial fluid rather than ligaments. This seems unlikely, because the barrier to flow is easily re-established by reduction of pressure (Fig. 5); whereas the channels contain oil at this point rather than synovial fluid. Ogston's observation might, however, explain the failure of the resin casts to reveal the thin sheet of synovial fluid which is assumed to occupy the intercartilaginous plane.

The hydraulic communication above plateau pressure

A puzzling feature of the pressures above plateau pressure was that, although anterior and posteromedial pressures were virtually equal in a few joints (e.g. Fig. 5), in most cases the pressure in the infused compartment was 1–4 cm H_2O greater than in the distant compartment, even after several minutes had been allowed for fluid transfer (see Figs. 3*B*, 4, 6*B*). While pressure differences of 1 cm H_2O could be accounted for by errors in the estimation of the zero positions of the cannula tips, differences of 4 cm H_2O could not be so explained. It is possible therefore that, even above plateau pressure, fluid communication between the anterior and posterior regions is intermittent in many joints: opening of the valve may require not only that a critical pressure be exceeded, but also that a small pressure difference exists across the channel. Intermittent closure of the links between anterior and posteromedial regions might also explain why the posterior portions of seven out of ten casts of extended joints were detached. The irregularity of the slope in some posterior pressure–volume curves (e.g. Fig. 4) indicates that the operation of the valve mechanism is somewhat 'sticky'.

Relevance to the synovial pressure–absorption relationship

It is considered improbable that the establishment of a communication between anterior and posteromedial regions of the joint could explain the breaking-point phenomenon, which is an increase in the absorptive capacity of the joint at $\sim 9{\cdot}6$ cm H_2O (Levick, 1979, 1980). First, the plateau pressures are mostly higher than breaking pressures. Secondly, the plateau phenomenon is immediately reversible whereas the breaking point phenomenon is not. Thirdly, the increase in absorptive area achieved by communication with the posteromedial region is $\sim 14\,\%$, whereas absorptive capacity increases by $> 500\,\%$ above breaking pressure. Finally, infusion of fluid simultaneously into suprapatellar and posteromedial pouches does not abolish the breaking point phenomenon (Levick, 1979).

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REFERENCES

DEANDRADE, J. R., GRANT, G. & DIXON, A. ST. J. (1965). Joint distension and reflex muscle inhibition in the knee. *J. Bone Jt Surg.* **47***A*, 313–322.

JAYSON, M. I. V. (1968). Study of a valvular mechanism in the formation of synovial cysts. *Ann. phys. Med.* **9**, 243–245.

JAYSON, M. I. V. & DIXON, A. ST J. (1970*a*). Intra-articular pressure in rheumatoid arthritis of the knee. I. Pressure changes during passsive joint distension. *Ann. rheum. Dis.* **29**, 261–265.

JAYSON, M. I. V. & DIXON, A. ST J. (1970*b*). Valvular mechanisms in juxta-glomerular cysts. *Ann. rheum. Dis.* **29**, 415–420.

KNIGHT, A. D. & LEVICK, J. R. (1981). Pressure–volume relationships and fluid compartmentation in synovial joints. *J. Physiol.* **319**, 36–37*P*.

KNIGHT, A. D. & LEVICK, J. R. (1982). Pressure–volume relationships above the below atmospheric pressure in the synovial cavity of the rabbit knee. *J. Physiol.* **328**, 403–420.

LEVICK, J. R. (1979). The influence of hydrostatic pressure on trans-synovial fluid movement and on capsular expansion in the rabbit knee. *J. Physiol.* **289**, 69–82.

LEVICK, J. R. (1980). Contributions of the lymphatic and microvascular systems to fluid absorption from the synovial cavity of the rabbit knee. *J. Physiol.* **306**, 445–461.

MCCARTY, D. J., PHELPS, P. & PYENSON, J. (1966). Crystal-induced inflammation in canine joints. I. An experimental model with quantification of the host response. *J. exp. Med.* **124**, 99–127.

MYERS, D. B. & PALMER, D. G. (1972). Capsular compliance and pressure–volume relationships in normal and arthritic knees. *J. Bone Jt Surg.* **54**B, 710–716.

OGSTON, A. G. & STANIER, J. E. (1953) The physiological function of hyaluronic acid in synovial fluid; viscous, elastic and lubricating properties. *J. Physiol.* **119**, 244–252.

ROBERTS, A. D. (1971). Role of electrical repulsive forces in synovial fluid. *Nature, Lond.* **231**, 434–436.