The patellar pain syndrome

In this study the term patellar pain (PP) is used to describe pain in the knee region, characteristically provoked or accentuated by a set of manoeuvres involving either motion of the patella, increased pressure of the patella against the femoral condyles, or a combination of both.

The main object is to describe and analyse the results of intraosseous patellar phlebography, pressure measurements and isotope scanning of the knee joint, and to compare the findings with histological changes in bone marrow, patellar cartilage and adjoining synovium.

PP may be due to a variety of causes (Table 1), patients with manifest arthrosis excluded. Objective signs may vary in the subgroups, but the pattern of pain is fairly uniform.

Subjective symptoms

Pain is the cardinal symptom. It is usually focused in the anteromedial aspect of the knee, but may occasionally be more pronounced laterally or in the popliteal region. Most patients talk of continuous discomfort that may increase to aggravating pain at rest, especially after unusual activity. At all stages of the disorder, pain is provoked or accentuated by loaded extension or flexion of the knee, the classical pain-provoking daily activities being climbing stairs, squatting, and riding a bicycle, especially uphill. In severe cases even prolonged sitting with bent knees, as in a cinema or car, may become intolerable. Sitting with the legs up and slightly flexed is the favourite resting position.

Most patients feel a momentary increase of pain during knee extension at the point when 30° flexion is passed. At this moment a click behind the kneecap is usually felt or heard. Many patients refer to these episodes as locking of the knee.

Table 1. Generally recognized causes of patellar pain

1	Malalignment syndromes, especially the "excessive lateral pressure syndrome" (Ficat et al. 1975).
2	Overuse syndromes. Patellar pain in structurally normal knees, related to occupation or athletics.
3	Reflex sympathetic dystrophy. Usually a posttraumatic, occasionally a postoperative, disorder (Ficat & Hungerford 1977).
4	Patellar arthrosis

Clinical signs

Most of those suffering from PP are young, between the ages of 15 and 40, and many are or have been active in sports. Women seem afflicted more often than men, and the preponderance of women seems to be correlated to their increasing participation in the more physically active sports.

In the majority of those with non-arthritic PP the range of knee motion is normal and, if performed without resistance, painless. Wasting of the quadriceps muscle is generally observed if the patient has had knee surgery, but is not a constant finding in untreated cases. Synovitis of the knee joint may be present, but relatively rarely. Crepitation on movement of the patella may be felt or heard and this manoeuvre is often painful. The knee is stable.

Two clinical tests, usually positive (i.e. provoking pain) in all severe PP cases regardless of etiology, have been used during our measurements of intrapatellar pressure: the pressure test and the sustained flexion test.

The pressure test is performed with the patient lying supine with the knee extended. The patella is placed in the trochlea and kept there with one hand, while the examiner puts weight on the patella by the palm of the other hand.

The sustained flexion test (Hejgaard and Arnoldi 1984). The patient is placed supine with the knee extended and relaxed. The knee is then flexed fully and kept firmly in sustained flexion for up to 45 s; the movement to full flexion is pain-free in the absence of arthrosis. The test is positive if the patient complains of increasing pain after a pain-free interlude of 15–30 s (Figure 1). The pain may become extremely severe and caution is advocated.

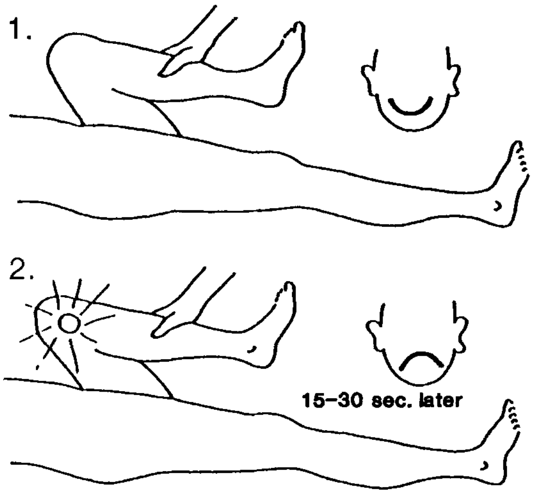


Figure 1. Positive sustained flexion test: pain-free movement into maximal flexion (1); after 15–30 seconds in maximal flexion the knee becomes increasingly painful.

Radiography

As a rule, routine radiography is normal in non-arthrosis cases, but tangential exposures and arthrography may show a variety of abnormalities most of which are of doubtful importance as causes of pain. These changes and their possible significance have been described in detail by Wiberg (1941), Stougaard (1975), Ficat and Hungerford (1977) and Lund (1978).

In this context, only the appearance of cancellous and subchondral bone is discussed.

Vascular circulation

Arterial supply

The patella is richly supplied with arterial blood. The patellar ring (rete patellae) is formed by contributions from six arteries: the supreme genicular, the medial and lateral superior genicular, the medial and lateral inferior genicular, and the anterior tibial recurrent arteries.

Björkström and Goldie (1980) described the extraosseous and intraosseous arterial supply thus: in the extraosseous patellar ring a transverse infrapatellar artery runs posterior to the patellar ligament. The other parts of the rete patellae and the branches forming the prepatellar arterial network lie in the thin layer of loose connective tissue that covers the fibrous extension of the quadriceps tendon on the anterior aspect of the kneecap. This part of the extraosseous arterial system is thus only covered by the subcutaneous fascia and the skin.

In addition to these main sources of arterial supply, Björkström and Goldie (1980) described further supply from the quadriceps tendon to the base of the patella, and from the synovial tissue to the margins of the bone and to the apex. They described two main intraosseous arterial systems. One is the mid-patellar branches that stem from the prepatellar network and enter obliquely upwards from below, through the central part of the anterior surface. They are distributed to the chondro-osseous junction in the upper part of the patella. The second system consists of apical branches from the transverse infrapatellar artery posterior to the patellar ligament. This system supplies the lower part of the patella and communicates with the midpatellar arteries. Considerable variations in size and importance were observed in the various groups of intrinsic arteries.

Normal cartilage. Björkström and Goldie (1980) observed that the subchondral arteries arose from the apical and mid-patellar arteries as straight or slightly curved branches radiating towards the subchondral region. In all specimens with normal cartilage, the calibre of the arteries gradually diminished to fine terminals. After age 60 the main arteries were thinner than in the young, and the endings did not reach out as far towards the periphery.

Chondromalacia. Björkström and Goldie (1980) observed that branches reaching normal cartilage zones resembled those of normal patellae, whereas arteries reaching the zones of degenerated cartilage became wider than normal, reached further towards the periphery, ending either straight and abruptly or were bent almost parallel to the cartilage. In the zone of degeneration the arterial pattern might take on an arcade-like design. Within these arcades the arteries were tortuous and wide at the peripheral margin. These vascular changes correlated with the grading of chondromalacia.

Arthrosis. Björkström and Goldie (1980) found that the design of the intrinsic arterial supply resembled that in severe stages of chondromalacia, but was more pronounced.

Figures 2 and 3 are microangiographs from cadaveric patellae (Bridgeman and Brookes 1990); Figure 2 confirms the description of the intrinsic arterial network given by Björkström and Goldie (1980), with an arcade-like design near the osteochondral border in chondromalacia (Figure 3). Further, a cluster of arterial branches is seen penetrating far into a patch of cartilage softening.

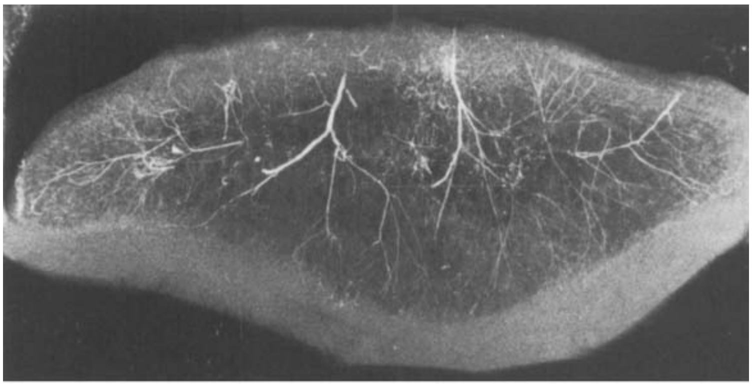


Figure 2. Microangiograph of a transverse section of a normal human patella showing arteries radiating towards the articular cartilage and recurrent branches supplying the patellar cortex centrifugally (Bridgeman and Brookes 1990).

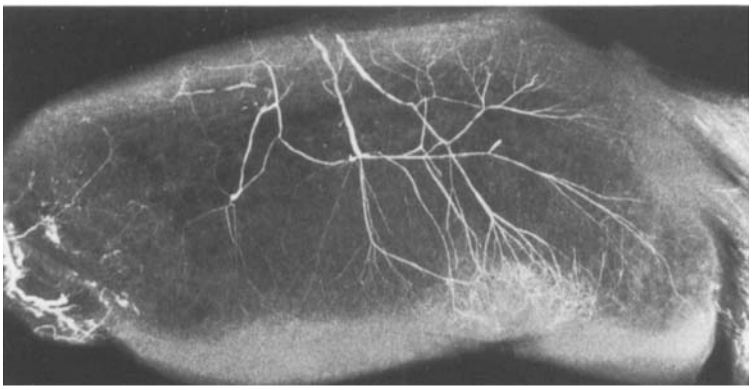


Figure 3. Microangiograph of a sagittal section of an aged human patella showing the auxillary inferior marginal blood supply. A patch of cartilage softening is associated with marked hypervascularity (Bridgeman and Brookes 1990).

Venous drainage

Venous flow from the patella has mainly been studied by means of intraosseous phlebography. The contrast-filled veins seem to correspond to the arteries in the patellar ring as will be described in detail below.

Pain sensors in bone

Cortical and trabecular bone is richly supplied with myelinated, as well as unmyelinated, nerves (Ottolenghi 1901, Sherman 1963). The nerves accompany the arterial branches into the Haversian system in cortical bone and into the medullary spaces.

In certain pathological states the nerves in bone marrow and periosteum seem to increase in number. Thus, Reimann and Christensen (1977) noted a higher density of nerves in arthrotic subchondral bone than in normal bone of the femoral head and ascribed the increase to the hypervascularity of arthrosis. Levine et al. (1984) ascribed an important role to the peripheral nervous system, especially the peptide neurotransmitter, substance P, in the severity of experimentally induced arthrosis. They found that joint capsules of rats with severe arthrosis are more densely innervated by sensory neurones that contain this substance. Badalamente and Cherney (1989) observed that in chondromalacial patellae the number of vessels at the osteochondral junction was much higher than in normal patellae. They penetrated the junction, including the duplicated or reduplicated tidemark, and small myelinated nerves containing substance P and serotonin were constantly associated with these minute vessels.

Cartilage abnormalities

Various degrees of patellar cartilaginous degeneration are frequently observed in even quite young PP patients, and *Chondromalacia patellae* is still the term used for this condition in most textbooks. However, post mortem studies, intraoperative observations and, later, the increasing use of the arthroscope for preoperative diagnosis have revealed that chondromalacia is observed in many subjects without a history of PP. Conversely, in a large number of patients with typical PP, the cartilage appears normal and its texture responds normally to touch or probe. Add to that the fact that normal cartilage does not contain nerves and it is understandable that most recent publications have discarded chondromalacia as the etiological factor for PP.

Staging of chondromalacia

The changes observed in knee joint cartilage in PP are usually confined to the patella, except in the last stages of chondromalacia when the cartilage of the femoral trochlea may show mirror changes. However, at this stage it is difficult to distinguish between chondromalacia and patellar arthrosis.

We have used the Ficat and Hungerford (1977) staging system:

Stage 0: no discernible changes in patellar cartilage.

Stage 1: local softening ("the blister lesion") of a circumscribed area, commonly, but not always, located in the "danger zone" on the lateral facet near the median ridge. At this stage the surface of the cartilage is intact. It is still a closed chondrosis.

Stage 2: open chondrosis. At this stage the degenerative cellular and ground-substance changes predominate. In particular, the superficial cartilage layer becomes involved by increasing proliferative changes alternating with signs of necrosis and ending in surface ulceration.

Stage 3: open chondrosis with denudation of bone and progression to arthrosis.

Bone abnormalities

In reflex sympathetic dystrophy, radiography shows a varying degree of *osteoporosis,* and arthrosis is characterized by *subchondral sclerosis.*

There is considerable difference of opinion about typical radiographic findings in the malalignment and overuse syndromes. Stougaard (1975) assessed the incidence of chondromalacia in autopsy material before age 50. He determined the site and extent of cartilaginous changes and their relationship to radiographic change using A-P and tangential exposures, as well as radiographs of 2-mm thick horizontal slices. The chondromalacial changes were far less marked on the lateral than on the medial facet, but subchondral sclerosis occurred only laterally and beneath normal, as well as degenerated, cartilage. Lund (1978), in a thorough statistical analysis of radiographic findings, found no differences between normal, chondromalacial or arthrotic joints in any radiographic variables, including subchondral sclerosis. The same view was expressed by Perrild et al. (1982).

Other authors have suggested that subchondral patellar sclerosis is a diagnostic sign of chondromalacia (Fürmaier 1952, DePalma 1954, Ficat et al. 1972). Ficat and Hungerford (1977) describe increased lateral subchondral density together with medial osteoporosis as being typical of the lateral pressure syndrome.

Synovial membrane

At the base of the patella the synovium, extending from the suprapatellar pouch, is densely adherent to the insertion of the quadriceps femoris tendon, while it is separated from the medial and lateral vasti by loose connective tissue (Ficat and Hungerford 1977). The medial, lateral and distal cartilaginous borders are surrounded by a small synovial wall or fold, separated from the patella by a fossa. In patients with PP syndromes, and especially those with the severe stages of chondromalacia, the peripatellar synovial wall and ditch often show characteristic signs of localized synovitis with inflammatory oedema that covers the entire peripheral area of cartilage. A synovial plica is often involved in this process and may adhere to the upper medial corner of the patella.

Such inflammatory changes may become very pronounced, but rarely exceed the immediate vicinity of the patella. Generalized synovitis of the knee joint is not common in the PP syndromes, arthrosis excepted.

Hoffa's fat pad

Covered by synovial membrane where it faces the joint cavity, the centre of the infrapatellar fat pad lies behind the ligamentum patellae, but extends beyond the borders of the ligament medially and laterally. During extreme flexion the centre is compressed by the ligament with the medial and lateral parts appearing as bulges on either side.

Proximally, the fat pad covers the entire posterior part of the apex below the border of the patellar cartilage, and distally it has a broad connection with the anterior proximal surface of the tibia.

Vessels and subsynovial tissues

Intraosseous phlebography of the patella indicates that the posterior surface of the apex is the most important exit for veins draining the bone. A number of relatively large veins invariably lead from this area to the fat pad where they anastomose with veins leaving the anterior part of the proximal tibia. Drainage from both sources reaches the inferior venous circle (Ficat and Hungerford 1977) with connections to the deep popliteal and the superficial saphenous veins.

Venous drainage from the patella, which takes place through thin-walled vessels with low intraluminar pressure, could thus be influenced by the following factors:

1) high intraosseous bone marrow pressure;
2) extraosseous compression by the skin and subcutaneous tissue during stretching of these structures (veins leaving the anterior aspect of the patella);
3) compression at the base of the patella by contraction of the quadriceps muscle;
4) compression by synovial and subsynovial inflammation (drainage from the medial and lateral margins);
5) compression by inflammatory or mechanical processes in Hoffa's fat pad, affecting the veins emerging from the apex.

Patellar circulation

Pressure measurements

Ficat and Hungerford (1977) found that the intramedullary pressure in normal human patellae, measured with subjects in the horizontal position was 10–15 mmHg. Björkström et al. (1980) found a mean normal pressure of 19 mmHg. In puppies, Bünger et al. (1982) found the patellar pressure to be 12 (8–15) mmHg and observed that the pressure rose to about 30 mmHg when the intra-articular knee pressure was raised by injection of fluid. If the fluid-injected knee was flexed beyond 90°, the mean pressure in the patella rose by approximately 20 mmHg.

In painful knees with reflex sympathetic dystrophy, base line pressure in the patella ranged from 30–50 mmHg (Ficat and Hungerford 1977). Björkström et al. (1980) measured the intramedullary pressure in chondromalacia, graded according to Collins (1949), and found mean values of 41 mmHg in Grade 1, 37 mmHg in Grade 2, and 64 mmHg in Grade 3. The mean pressure in their normal control group was 19 mmHg, and in patellar arthrosis 37 mmHg. In all groups the pressures varied within wide limits.

None of these examinations recorded extraosseous venous pressure simultaneously with intraosseous pressure. These studies agree about intraosseous pressure in normal human and animal patellae and they also indicate that painful patellar arthrosis and chondromalacia seem to be associated with increased intrapatellar pressure in the resting horizontal position.

Material

We studied patients with severe PP and loss of function, resistant to at least 6 months strict conservative treatment. The various etiological entities (Table 1) were all represented, but as typical persistent pain was the only criterion for invasive diagnostic procedures and operations no attempt was made to group the patients by etiology. This would also have been difficult as half the affected knees had already had operations elsewhere, a typical history being persistent pain leading to arthrotomy without improvement, followed by chondrectomy with exacerbation, and then lateral release causing disablement.

We studied the relationship between characteristic pain, the venous drainage system of the patella, and the pressure and pressure variations in the three bones of the knee joint. ^{99m}Tc-phosphate scintigraphy was also used to identify possible changes in bone marrow metabolism.

After pressure measurements and phlebography, arthroscopy was performed on the painful knees using a standard infero-lateral approach. The articular surfaces were examined by eye and by probe. Patellar chondromalacia was graded according Ficat and Hungerford (1977).

The material was 24 men, median age 33 (20–48) years and 37 women, median age 34 (16–60) years. The symptoms were bilateral in 5 men and 15 women. There were thus 81 painful knees and 41 knees (control) without pain. Patients with radiographic gonarthrosis were excluded. The mean duration of symptoms was 3 (1–12) years.

Techniques

The patients were examined supine under general anesthesia. Premedication was oral benzodiazepine, and anesthesia was induced by intravenous thiopentone and suzamethonium chloride, maintained, after endotracheal intubation, by halothane and NO_2, supplemented with intravenous fenatyl citrate. Relaxation was achieved with gallamine.

Bone-marrow biopsy needles, 4 cm long with an outer diameter of 3 mm and a lumen of 1.5 mm, were drilled percutaneously into the patella close to its base, with the tip directed slightly distally. Similar needles were drilled 2 cm into the medial femoral and tibial condyles. Extraosseous venous pressure was measured through a tube introduced into the internal saphenous vein of the affected leg at the level of the medial malleolus. The tip of the tube was placed at the junction of the saphenous and femoral veins. Heart level, arbitrarily fixed at 5 cm posterior to the sternum at the level of the fourth intercostal space, was used as reference point for all measurements. The needles and the intravenous catheter were flushed with 1 mL of heparine saline solution and connected to a four-channel pressure recording system. Within the range of measurement, the recording system gave proportional deflection according to the pressure applied and responded accurately in frequency and amplitude to sine-wave pressure as high as 15 Hz.

Intraosseous pressure was defined as the difference between the measured intraosseous pressure and the extraosseous venous pressure. The intramedullary pressure was measured simultaneously in the patella and the femoral and tibial condyles.

All patients were examined bilaterally with the knees in relaxed extension and in sustained forceful flexion. As noted in the phlebographic studies, the veins leaving the apex patellae through Hoffa's fat pad seem to be the most important drainage complex from the patellar bone marrow. It was therefore of interest to determine what effect their compression would have on intraosseous pressure in the relaxed extended knee. Groups were therefore further examined bilaterally in the supine position and during compression of Hoffa's fat pad, by pinching with the thumb and index finger on both sides of the patellar ligament, and during forceful pressure on the patella against the femur, simulating the pressure test.

Table 2. Pressures in the patella, and the femoral and tibial condyles in the relaxed, extended knee in controls and in knees with patello-femoral pain. Mean (range) mmHg above the extraosseous venous pressure

Group	No	Patella	Femur	Tibia
Controls	41	19.1 (0–68)	17.6 (0–46)	13.4 (2–38)
With pain	81	24.4 (3–62)	17.9 (0–54)	15.0 (2–38)

Difference in patellar pressure, 5.3 mmHg ($P < 0.01$, Mann-Whitney *U* test)

Table 3. Pressures in the patella, and the femoral and tibial condyles, during compression of Hoffa's fat pad and during compression of the patella against the femoral trochlea in 10 control knees. Mean (range) mmHg above the pressure in the saphenous vein

Compression	Patella	Femur	Tibia
Hoffa's pad	44.0 (22–53)	25.3 (8–44)	26.6 (13–47)
Patella	72.7 (44–98)	44.0 (25–76)	35.6 (10–57)

Results

Relaxed extended knee

Total material comprised 61 patients, 40 of whom had long-standing pain, severe enough to warrant operation. In this group Hejgaard and Arnoldi (1984) found a higher patellar pressure in painful knees than in controls ($P < 0.001$, Mann-Whitney) and those with pain at rest had higher pressure than those without ($P < 0.05$).

The difference in mean patellar pressure between painful and control knees was significant, whereas the differences observed at the two other points of measurement were not (Table 2). The mean extraosseous venous pressure was 8 (2–18) mmHg. No difference between pain-free and painful extremities was observed.

Figure 4 shows a typical intraosseous pressure reaction at the three points of measurement and Table 3 the mean pressure rise above rest level in the patella, femur and tibia.

Compression of Hoffa's fat pad. Intraosseous pressure tracings are normally pulsatile. Non-pulsatile tracings during rest with the knee extended were always artefacts caused by obstruction of the cannula and could be corrected by injection of heparine-saline or by further insertion of the needle, or both. With the pressure measuring system used during this period it was not possible to compare pulse excursions on tracings from different points of measurement, but it was always possible to follow their variations in size on the tracings from each individual measurement point.

Table 4 gives the mean values of intraosseous pulse

Figure 4. Pressure tracings from the patella and the femoral and tibial condyles, before during, and after compression of Hoffa's fat pad. Pain-free knee in relaxed extension.

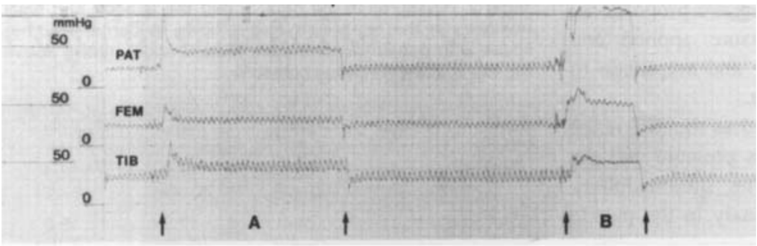


Figure 5. Pressure tracings from the three points of measurements in control knees:
A. During compression of Hoffa's fat pad.
B. During compression of the patella against the femoral trochlea.

Figure 6. Pressure tracings from the patella and the femoral and tibial condyles, before during, and after a period of sustained maximal knee flexion in a pain-free knee.

Table 4 gives the mean values of intraosseous pulse amplitudes before, during and after compression of Hoffa's fat pad (Figures 4 and 5). The size of pulse pressure waves increased in the pain-free patella ($P < 0.001$) and in the tibia ($P < 0.01$). There was a tendency to even greater excursions in painful knees, but the difference between control and painful knees was significant only in the patella ($P < 0.05$, Mann-Whitney).

Manual compression of the patella. The effects on intraosseous pressure of compression of Hoffa's fat pad and of the patella against the femoral trochlea (pressure test) were compared in 10 controls. The rise in pressure was greater at all points of measurements during compression of the patella than during blockage of the apical veins (Table 3).

The traceable effects of these two manoeuvres on the pulsatile excursions from the patella are compared in Table 4. As observed earlier the size of the pulse waves increased during fat pad compression. Patellar compression, however, was always accompanied by total or almost total disappearance of pulse synchronic excursions (Figure 5). In the femur and tibia, where the rise of intraosseous pressure during patellar compression was modest, the tracings remained pulsatile.

Measurements of pressure were followed by arthroscopy in 68 PP knees. The presence or absence of chondromalacia was noted and graded (Table 5). Taken as a group, the pressure in chondromalacial patellae was not higher than in painful knees without.

Sustained knee flexion

Forced maximum flexion of the knee caused a sudden rise of intraosseous pressure, in control as well as PP knees, at all three points of measurement (Figure 6). After peaking, the pressures fell to a plateau which, as a rule, was level until the knee was stretched; they then fell steeply to a point somewhat lower than the initial resting level. Pressure recovery to this level generally took less than one second in the patella and femur, and up to 3 seconds in the tibia.

Table 4. Mean pulse amplitudes (mmHg) in 10 control knees in extension, before, during, and after compression of Hoffa's pad and patella against the femur

Compression	Before	During	After
Hoffa's pad	8	13	8
Patella	8	0	8

Table 5. Pressure in patella at rest with the knee in extension; and plateau pressure during sustained maximal knee flexion in knees with patellar pain, with and without arthroscopically verified chondromalacia (staging system, Ficat & Hungerford 1977). Mean (range) mmHg

Group	No	At rest	Maximally flexed
Without chondromalacia	27	22.9 (3–46)	79.6 (26–255)
With chondromalacia	41	24.9 (5–62)	86.1 (16–161)
Stage I + II	14	27.5 (5–62)	88.5 (29–148)
Stage III	24	23.6 (3–46)	77.5 (16–161)

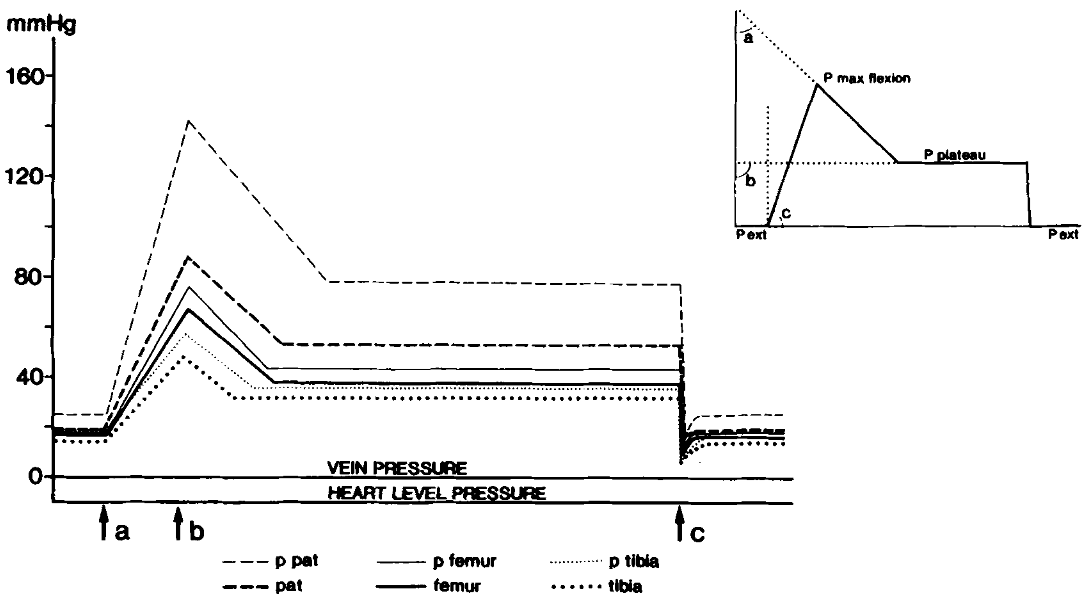


Figure 7. Pressures at rest—peak values and plateau pressures—before, during, and after sustained maximal knee flexion. Mean values from the three points of measurements in pain-free knees and in knees with patellar pain. The method of assessing the rapidity of pressure rise and fall is illustrated in the upper right section. Mean pressure in the internal saphenous vein is 8 mmHg. The letter "p" in front of the measurement indicates pressure tracing from painful knees. Mean interval a–b was 1.2 sec. The interval b–c varied within wide limits.

Mean pressures during sustained knee flexion in control knees and PP knees are shown in Figure 7, which also gives the mean angles of pressure increase and decrease. Table 6 gives the detailed results. The differences in maximum and plateau pressure between patellae in control and painful knees were highly significant ($P < 0.001$, Mann-Whitney). In the femur and tibia there were no pressure differences between control and painful knees.

Plateau pressures. Clinical results of the sustained flexion test in 66 PP knees were compared with the pressure in the patellar bone marrow during sustained flexion. As painless movement into flexion, arthrosis excluded, followed by a pain-free interlude of 15–30 seconds is characteristic of a positive test (Figure 1), it was of special interest to compare plateau pressure with the outcome of the test. The plateau pressure was higher in painful knees with positive clinical tests, than in those with a negative test ($P < 0.001$, Mann-Whitney; Table 7).

During sustained knee flexion, the bone marrow pressures seemed to be largely equal in painful joints with and without chondromalacia (Table 5). As was the case in relaxed extension, the plateau pressure seemed to be slightly higher in the early stages than in Stage 3 ($P < 0.08$).

Table 6. Peak and plateau pressure in the patella, and the femoral and tibial condyles during sustained maximal knee flexion in controls and in knees with patellar pain. Mean (range) mmHg

Group	No	Peak pressure	Plateau pressure
Patella			
Control	27	88.6 (29–172)	53.8 (17–92)
Painful	69	143.2 (36–256)	77.5 (16–256)
Femur			
Control	27	67.1 (19–118)	38.2 (14–62)
Painful	69	76.7 (20–248)	43.4 (10–88)
Tibia			
Control	27	48.0 (14–84)	32.9 (8–52)
Painful	69	56.5 (18–120)	35.4 (7–72)

Table 7. Patellar plateau pressure during sustained maximal knee flexion in 66 knees with patellar pain. The group is divided according to the results of the sustained flexion test. Mean (range) mmHg

Test	No	Plateau pressure
Negative	12	46.0 (16–86)
Positive	54	89.8 (29–256)

Pressure waves. Pulse waves in the patella disappeared completely from the pressure tracings during sustained flexion in 27 of 33 control knees. In 6 knees, they became visible when the pressure had reached plateau level. In this subgroup, the plateau pressure was 30 (28–60) mmHg, compared with 54 (17–92) mmHg in the whole group. Of the 69 PP knees, 6 showed pulsatile plateau pressure, with a mean of 34 (26–61) mmHg, compared with 78 (16–256) mmHg in the whole group. In the femur and tibia all pressure tracings were pulsatile at plateau level.

Intraosseus phlebography

Intraosseous phlebography of the normal patella was performed by Ficat and Hungerford (1977) on subjects lying supine with the knee extended. A-P and lateral projections were used. They found the apical region to be the main hilus of venous drainage from the patella and that the contrast-filled veins seemed to follow the course of the arteries supplying the bone. In reflex sympathetic dystrophy they found intramedullary stasis in the femoral condyles. They do not seem to have performed patellar phlebographs in these cases.

Material

Our material comprised 24 men and 34 women with typical PP. In 6 men and 13 women the disorder was bilateral. Intraosseous phlebographs were performed on both patellae and thus consisted of 39 examinations of patellae from pain-free control knees and 77 of patellae from PP knees. The patient material was almost identical with that examined by pressure measurements; its characteristics are further described in the section on pressure measurements.

Methods

The examinations were performed on the operating table under general anesthesia after the pressure measurements. Contrast material (2–3 mL) was injected into the patella, and the first exposure was made 1 second after completion of the injection. Further exposures were made at 1 and 5 minutes and in a number of cases at 10 or 15 minutes. Lateral projections were used.

In 30 patients, the examination of the extended knee was followed by a second series of bilateral phlebographs with the knee in sustained maximal flexion. The same amount of contrast material was used in this series and the exposures were made at the same time intervals.

Lateral projections were used. Lateral projections were preferred to A-P exposures, as we were particularly interested in the study of apical, anterior, and superior drainage vessels under various conditions. In our experience, these vessels are more clearly discerned in lateral exposure.

The following details were particularly noted:

1) the position and course of drainage veins from pain-free control patellae;
2) their presence or absence in phlebographs from painful knees;
3) the structure and appearance of the network of intrapatellar bone marrow vessels in control and painful knees;
4) the emptying time (i.e., the number of minutes from the end of contrast injection to the first contrast-free exposure) was determined for extraosseous, as well as intraosseous veins and comparison was made between painful and pain-free knees;
5) the changes wrought by sustained maximal flexion of the knee.

Results

Patellar drainage in the extended knee

The extraosseous course of the veins was observed on the exposures at 1 second after injection of contrast. On all phlebographs the contrast in extraosseous veins of both control and PP knees had disappeared within 1 minute of injection (Figure 8). The following vessels were always observed in pain-free knees:

1) a cluster of 5–12 veins leaving the apex in a distal posterior direction, draining into the saphenous system and into the popliteal vein (Figure 8);
2) a varying number of veins leaving the anterior surface of the patella and following it closely in a proximal direction to superficial femoral veins;
3) a varying number of slender veins leaving the basis patellae in a proximal direction to deep and superficial veins of the femur;
4) a few slender veins leaving the medial and lateral margins of the patella.

In painful knees the overall picture corresponded to the description given above. However, in 21 of them, one or several of these systems were missing from the phlebographs, usually the veins leaving the basis and the lateral and medial margins. Only in 2 cases had the apical veins disappeared completely, while they were unusually slender in 8 others.

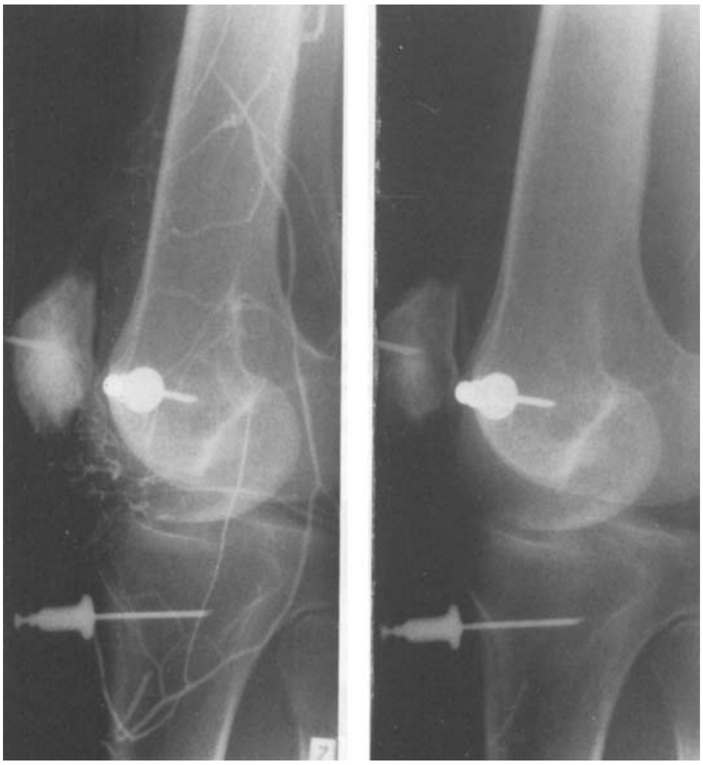


Figure 8. Intraosseous patellar phlebograph from a pain-free knee.

Left phlebograph exposed 1 sec after contrast injection showing filled intra- and extraosseous veins, apical drainage, and a few veins leaving the basis patellae.

Right phlebograph, the same knee exposed 5 min later showing complete evacuation of contrast from intra- and extraosseous veins.

Patellar drainage in sustained flexion

In almost all knees, with or without pain, the veins emerging from the anterior aspect, the base and the medial and lateral borders had disappeared from the phlebographs (Figure 9). The apical veins thus seemed to be the only drainage channels functioning in this position, and in many patients the picture suggested a decrease of vein diameters. In all cases the extraosseous veins were empty on the 1 minute exposure.

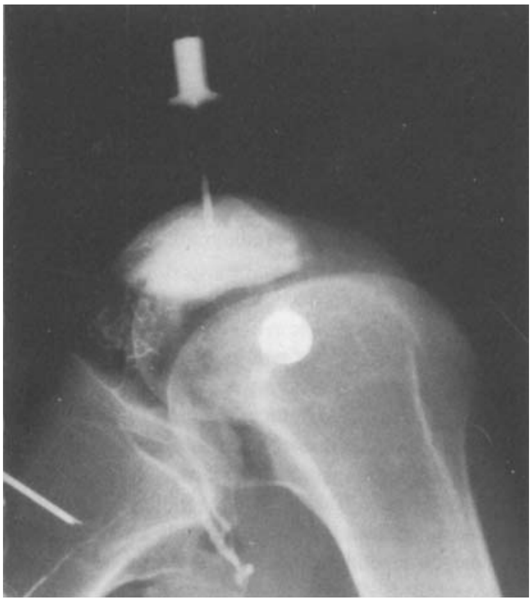


Figure 9. Intraosseous patellar phlebograph, from the same pain-free knee as shown in Figure 8, exposed 1 sec after contrast injection. Apical veins are the only escape route for the injected contrast.

Intraosseous venous patterns

Two intraosseous venous patterns could be clearly distinguished as: 1) a tightly woven mesh of minute vessels (Figure 10); and 2) a loose network of larger venules and veins (Figure 11).

Their distribution between control and painful knees was: Type 1, 36 of 39 controls and 32 of 77 PP knees; Type 2, 3 of 39 controls and 45 of 77 PP knees. Patients with painful knees and Type 1 phlebographs were younger, 27 (16–48) years, than those with Type 2, 39 (22–60) years.

An analysis of case histories revealed that most, but

Figure 10. Type 1 intraosseous phlebograph from a knee with patellar pain exposed 10 min after contrast injection with the knee in extension. Extraosseous veins are empty and contrast is only retained in a fine-meshed intraosseous network.

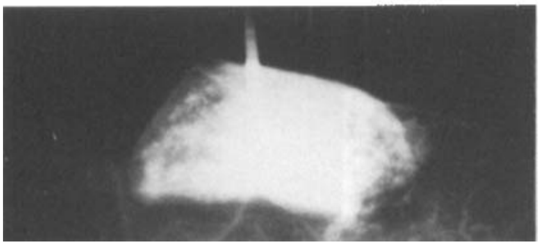


Figure 11. Type 2 intraosseous phlebograph from a knee with patellar pain exposed 1 sec after contrast injection with the knee in extension. Extraosseous drainage is visible. The contrast fills a loose network of larger venous channels in the patellar bone marrow. The contrast was completely evacuated within 5 min.

An analysis of case histories revealed that most, but not all, with a history of overuse of the knees (e.g. sport) had Type 1 phlebographs, while most, but not all, patients with a history of direct knee trauma had Type 2 phlebographs; however, most Type 2 patients had normal routine skyline radiographs.

Extended knee

Pain-free knees. Total or nearly total emptying of intramedullary contrast occurred within 10 minutes from 28 patellae (Figure 8); in the remaining 11 the contrast distribution remained unchanged. All 3 patellae with Type 2 intraosseous patterns emptied completely.

Painful knees. Taken as a group, 27 emptied completely within 10 minutes, while no change in contrast distribution could be observed in the remaining 50. A difference was noted between intramedullary Types 1 and 2 patterns; 25 of 45 Type 2 patellae emptied within 10 minutes, the majority within 5 minutes. In contrast, only 2 of 37 patellae with Type 1 pattern were empty within 10 minutes.

Sustained flexion

From the 39 painful and 21 control knees examined phlebographically in sustained flexion, no emptying of contrast material from the patellar bone marrow was observed during the 10-minute observation time. There was no difference between pain-free and painful knees or between the two intramedullary vessel patterns.

^{99m}Tc-phosphate scintigraphy

Lin et al. (1981) found in 130 patients, referred for "a variety of reasons", that bone scan showed increased uptake in 38 percent of the knees examined. In a prospective study, Kipper et al. (1982) found increased isotope uptake in 20 of 100 consecutive examinations. Most of those with increased patellar uptake had malignant systemic or degenerative diseases; only 3 had knee pain. Fogelmann et al. (1983) found that increased patellar isotope uptake was especially characteristic of metabolic bone disorders and malignant disease.

Darracott and Vernon-Roberts (1971), using ^{87m}Sr, examined 11 patients with chondromalacia patellae. Seven cases with unilateral symptoms exhibited reduced isotope uptake in the region of the affected patellae, whereas the other two had moderately increased patellar uptake. Of the two patients with bilateral symptoms, one had reduced uptake in the region of both patellae, the other had markedly increased uptake in one patella and reduced uptake in the other. In all cases lateral scans showed the changes in uptake to be maximal in the region of the patellae, although similar changes of lesser degree were occasionally recorded in the femur or tibia.

Investigations of present material

Hejgaard and Diemer (1986) used ^{99m}Tc-diphosphonate scintigraphy, intraosseous pressure determination, radiography, arthroscopy and physical diagnostic tests to examine 80 PP patients. Their material was largely identical with that described in the sections on intraosseous pressure and phlebography. Their bone scans showed that half of the painful knees had an increased uptake, compared with one tenth of controls. A highly significant correlation was evident between an increased uptake and painful Stage 3 chondromalacia.

increased uptake and painful Stage 3 chondromalacia.

The relative values of radiography, bone scintigraphy and the sustained flexion test were compared for diagnosis of "high pressure" patellae. The sensitivity of radiography was only 7 percent, compared with 44 percent for bone scintigraphy and 78 percent for the clinical sustained flexion test. The positive predictive value of a bone scan for detecting high pressure patellae was 0.72. The best predictor was a positive sustained flexion test with a predictive value of 0.85.

Histology in chondromalacia

Darracott and Vernon Roberts (1971) in studies of chondromalacia and control patellae found a thinning of the subchondral osseous plate in all cases of chondromalacia. The trabecular bone showed either diffuse osteoporosis or isolated osteopenic foci. There was also vascular invasion through the osseous plate, and where this was present, the deep zone of the patellar cartilage showed hyperplasia of the chondrocytes. Osteopenia was most marked in the regions of vascular supply; this could suggest that the changes in bone were secondary to disturbances of blood supply.

Badalamente and Cherney (1989) compared the vascular and periosteal innervation of the human patella in the normal state and in chondromalacia. In control patellae arterial capillaries and thin-walled venous capillaries were present at the osteochondral junction and deep to the single-layer tidemark. In chondromalacia arterial and venous capillaries consistently penetrated the osteochondral junction, the basal calcified layer of cartilage as well as the duplicated or reduplicated tidemark. Further, the number of vessels increased from 1.5 arterial capillaries/10^5 μm^2 in control patellae, to 6.0 vessels/10^5 μm^2 in chondromalacia. In all cases, small myelinated nerves containing substance P and serotonin were consistently associated with capillaries in subchondral and medullary trabecular bone and in the periosteum. Badalamente and Cherney (1989), suggest that substance P and serotonin, by their location, may influence the vasoactivity, substance P as a potent vasodilator and serotonin as a peripheral vasoconstrictor and neurotransmitter of sensory pain signals (Hökfelt et al. 1975). They also suggest that the increase in bone marrow pressure and resultant pain in chondromalacia and arthrosis may be related to the increased number of vessels.

Goodfellow et al. (1976) found two distinct lesions affecting the articular cartilage of the patella. Surface degeneration, which is age dependent, becomes increasingly more frequent with increasing age. In their opinion it does not cause patellar pain in youth, but may predispose to degenerative arthrosis in later years. Goodfellow et al. (1976) introduced the term "basal cartilage degeneration" to describe a lesion in which there is fasciculation of collagen in the middle and deep zones of cartilage without, at first, affecting the surface. It was found astride the ridge separating the medial from the odd facet in adolescents who had complained of prolonged patellar pain.

The diagrams given here (Figure 23) are modifications of the original drawings by Goodfellow et al. (1976). They described the state of cartilage in fasciculation thus: "the articular surface is smoothly intact and the disorder can only be detected by palpation. The cartilage has then an appreciably spongy consistency and exhibits what can be fairly described as pitting edema. They also found that in all fasciculated specimens the matrix stained less well with P.A.S. than in normal cartilage.

Further, they noticed that synovial effusion was only present where the superficial layer had ruptured, and in these cases the synovial fluid was found to contain "innumerable small pieces of shed cartilage". Special histologic examinations of the synovium in chondromalacia (PP knees) do not seem to have been reported however.

Our histological investigations were directed at normal cartilage and synovium and the changes observed in the different stages of chondromalacia patellae in PP knees and in gonarthrosis. As for the bony structures the interest was centered on vascular changes in or near the osteochondral junction.

Material

Normal patellae: 6 specimens removed from 5 subjects at autopsy. These patellae and knee joints appeared normal to the naked eye and probe, and the histories did not suggest any previous knee trouble. Three of these subjects were women, two men with median age 30 (16–62) years.

PP patellae: 4 patellae removed by patellectomy, 3 women and 1 man with median age 35 (31–61) years.

Patellae from arthrotic joints: 11 kneecaps removed at autopsy from 4 women and 7 men with median age 70 (58–81) years.

In all cases, the patella was removed together with the adjacent synovial membrane and part of Hoffa's fat pad. In PP knees a biopsy was also taken at an average 3 cm from the medial patellar margin.

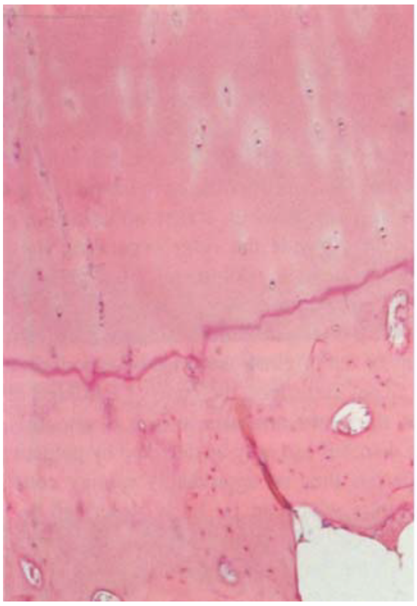


Figure 12. Area of osteochondral junction and tidemark from a normal patella, HE ×100.

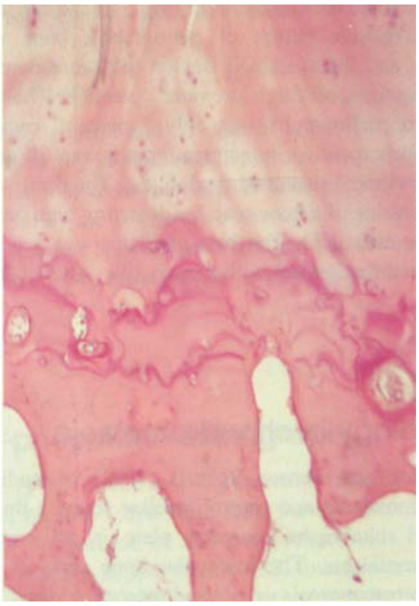


Figure 13. Same area as in Figure 12 but from a painful patella (chondromalacia, Stage 2). Multiplication of tidemark. Lacunae with vessels between tidemarks, HE ×100.

Methods

All specimens were kept in formalin solution until preparation. Hematoxylen-eosin was used for all joint components. MSB (Martius scarlet blue), the modification by Pusey and Edwards (1978), was used to visualize erythrocytes in interstitial tissues, intravascular erythrocytes, agglutination and fibrin thromboses, and the presence and structure of collagen.

Results

Bone marrow and osteochondral junction

In the centre the patella of PP knees was clearly osteopenic, compared with normal and arthrotic patellae, and our findings did not differ from those of Darracott and Vernon-Roberts (1971). The thinning of the osteochondral end plate observed by these authors and Badalamente and Cherney (1989) was also confirmed (Figures 13–16), as was the increased vascularity of this part of the bone.

In normal and in most arthrotic patellae the thickness of the end plate compared with PP patellae was quite striking (Figures 12 and 13), but the most noticeable difference was the almost universal finding of vascular penetration through the bone into the basal calcified layer of cartilage and through the tidemark. Where this had happened, a second tidemark had appeared, looking almost like a second line of defense. Up to six tidemark lines were observed, but occasionally vessels with erythrocytes and fibrin appeared peripheral to the outermost tidemark in the matrix of the middle layer of cartilage. In our numerically very small material this configuration appeared in all cases.

In two cases of arthrosis, duplication of tidemark and vascular invasion into the basal calcified layer were noted. However, these were autopsy cases and the history of the knee disorder was not known. Duplication of tidemark and vascular penetration from bone were never observed in normal patellae.

Matrix

In the normal patella the matrix (collagen) is stained a uniform clear blue by the MSB method (Figur 14). In PP patellae stained by this technique, the characteristic picture was a smaller or greater area where the middle layer had lost its blue colour and taken on a grayish hue, often with reddish variations (Figure 15). The demarcation between these areas was often sharply defined. The superficial layer retained its dark blue collagen colour as long as it remained intact (Figure 15).

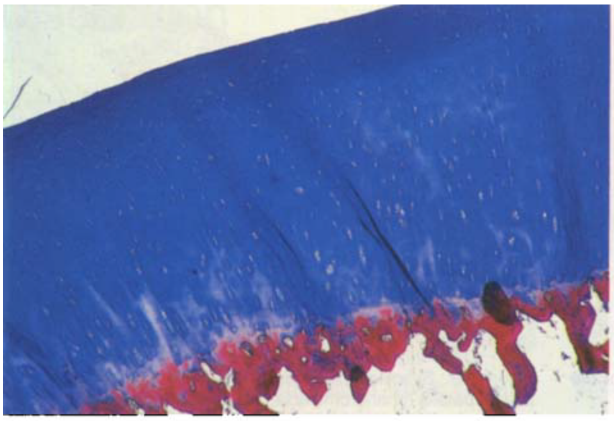


Figure 14. Chondromalacia patellae. Macroscopically normal cartilage on the lateral facet, MSB ×25.

Figure 15. Same patella as in Figure 14. Section from "blister lesion", MSB ×25.

Figure 16. Section of the osteochondral junction in a painful patella (chondromalacia, Stage 2). Vessel penetration from bone marrow into cartilage through several tidemarks. Erythrocyte stasis to aggregation, MSB ×250.

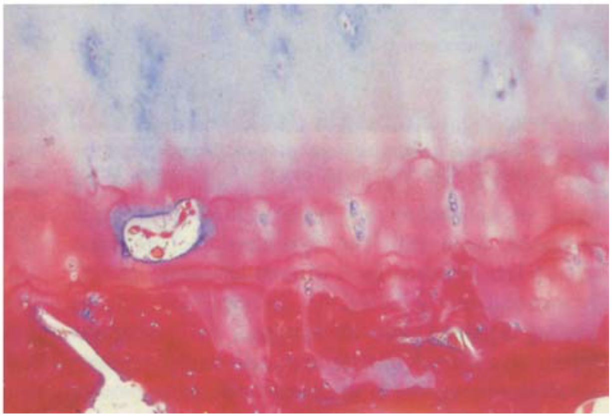


Figure 17. Section of the osteochondral junction in a painful patella (chondromalacia, Stage 1). Triplication of tidemark. Intravascular fibrin thrombi (red) in canal between second and third tidemark, as well as in intraosseous vessel (lower right), MSB ×100.

Chondrocytes

Compared with normal patellae the chondrocytes of PP cartilage and their territorial matrix did not appear affected, where the matrix had kept its normal blue colour by MSB staining. In the areas of decolouration the clusters and pillars of chondrocytes seemed more widely dispersed than under normal circumstances and in certain areas they were very scarce (Figures 15–17). In our material we could not demonstrate the chondrocyte hyperactivity mentioned by Darracott and Vernon-Roberts (1971).

Synovium

Normal knees. The biopsies from these specimens had the same appearance as normal synovium in any other normal joint (Figure 18): 1–2 rows of synoviocytes

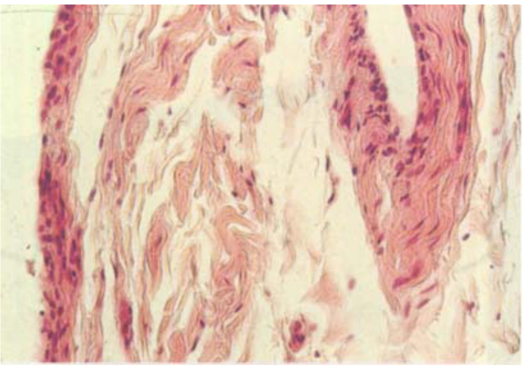


Figure 18. Synovial membrane from the medial border of the patella in a normal knee, HE ×250.

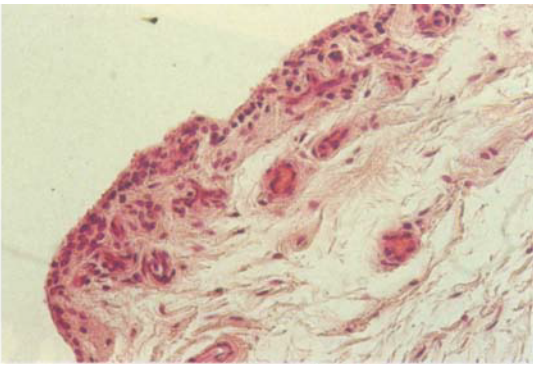


Figure 19. Synovial membrane from the medial border of a painful patella (chondromalacia, Stage 3 to arthrosis). Increased vascularisation and erythrocyte stasis, HE ×250.

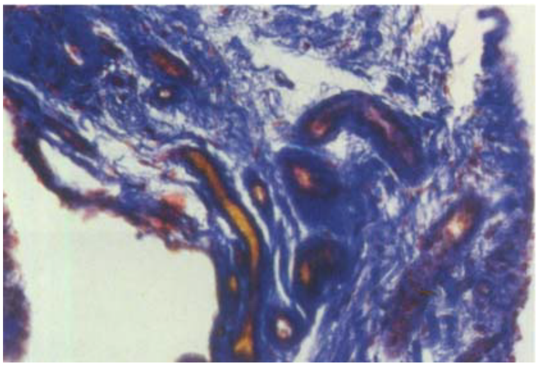


Figure 20. Synovial membrane from recessus suprapatellaris in a painful patella (chondromalacia, Stage 3 to arthrosis), MSB ×250.

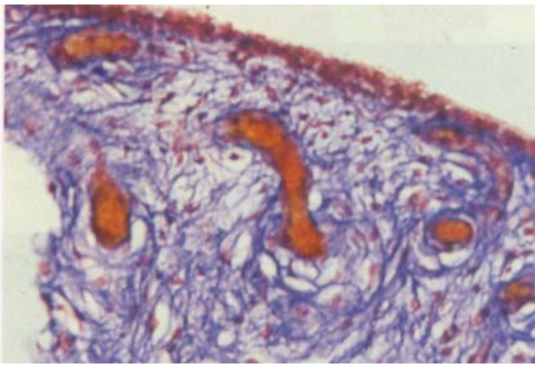


Figure 21. Synovial membrane from recessus suprapatellaris in a patient with gonarthrosis, MSB ×250. In Figures 19 and 20, intravascular erythrocyte stasis, agglutination, and fibrin thrombi are characteristic features.

covering a rather loose stroma, poor in vessels. In Hoffa's pad several smaller and some larger normal vessels were observed in all cases.

Gonarthrosis. In arthrosis the synovium was characterized by an increased number of synoviocytes, 4–6 rows not being unusual. Collections of inflammatory cells were seen occasionally. The stroma was oedematous with more or less collagen. Fibrosis could dominate the picture and, generally, the synovial membrane was much thicker than normal. The most striking abnormality was, however, the greatly increased vascularity of the membrane with intravascular stasis, structureless aggregations of erythrocytes filling long sections of small and medium-sized vessels, and sometimes turning into fibrin thrombi (Figure 21). Most vessels seemed dilated and MSB staining showed numerous extravascular interstitial erythrocytes. This general picture was independent of the location of the biopsy site.

PP knees. In all biopsies taken from the parapatellar "wall and ditch", most of the changes (except hemosiderin deposits) described under arthrosis were present, although mostly in a more modest degree as long as the patellar cartilage surface was intact. In these cases synovial biopsies taken at a distance from the patellar margin were all normal. In the two Stage 3-cases the outlying biopsies had the same general appearance as in arthrosis (Figure 20).

Hoffa's fat pad

In two PP cases (chondromalacia Stage 0–1), intravascular stasis and structureless erythrocyte aggregations were observed. In the other PP specimens the vascular structures did not differ from those observed in normal knees.

Discussion

Causes of increased intraosseous pressure in the knee

Increased intraosseous pressure is nearly always due to high resistance to the blood flow in the veins draining bone marrow. Intraosseous hypertension may be intermittent, chronic, or a combination where chronically high pressure is increased periodically.

The cause of abnormally high resistance to venous flow from the bone marrow may be found proximal to the joint structures, i.e. between the joint and the heart, in the joint or in the bone marrow (Arnoldi 1990).

Supra-articular causes of increased pressures

The results of pathological changes in supra-articular drainage are best known from patients with severe and long-standing venous insufficiency of the lower limb (Arnoldi et al. 1971). In those patients intraosseous hypertension is only present during walking and thus strictly intermittent. When measured in the horizontal position and at rest, bone marrow pressure at the ankle is normal.

Articular and intraosseous changes influencing pressure in joint-bearing bone marrow

Figure 22 shows in schematic form the known and suspected factors influencing bone-marrow pressure:

1) Intra-articular compression of veins draining juxta-articular bone.
2) Compression of the draining veins by structural stretching or torsion as they pass through the fibrous capsule.
3) Compression deformation of closed or semi-closed intraosseous compartments.
4) (?) Intra- or extravascular blockage of venous circulation inside the bone marrow.

Intra-articular compression of veins draining juxta-articular bone

Synovitis is a characteristic early manifestation of the intraosseous engorgement-pain syndromes, arthrosis and rheumatoid arthritis. Increased intra-articular volume causes increased intra-articular pressure, the rise depending on the degree of volume increase and the strength of the fibrous capsule (Eyring and Murray 1964). In the joints most commonly affected in the lower limb, the hip and the knee, the proximal and distal ends of the femur are ensheathed by large synovial joint recesses, and the most important drainage from the femoral head and neck and a large part of the femoral condyles is via subsynovial channels. That compression and closure of these veins—with intact arterial inflow—leads to intraosseous engorgement and hypertension is well documented (see e.g. Arnoldi and Reimann 1979, Bünger et al. 1981, 1982, 1983, Lucht et al. 1981).

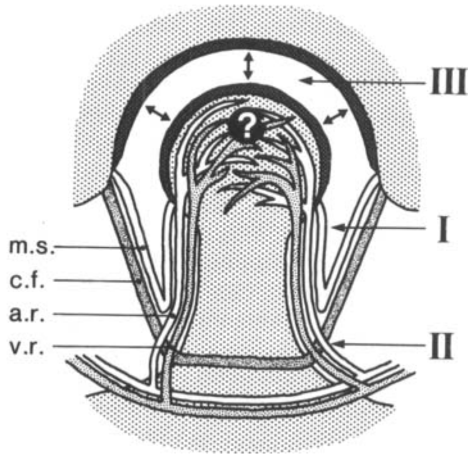


Figure 22. Schematic representation of the relationships between the retinacular vessels and the synovial and fibrous capsules of the hip joint. m.s. synovial membrane, c.f. fibrous capsule, a.r. retinacular artery, and v.r. retinacular vein.

Compression of draining veins during their transcapsular course

The effect of this mechanism has been demonstrated in horses (Arnoldi et al. 1980), and is probably also one of the factors responsible for the steep rise in pressure in the femoral head on inward rotation of the hip joint (Arnoldi 1990).

Compression deformation

This occurs when a joint is under load, and pressure variations under such conditions have been studied previously in the femoral head (part of a tubular bone). The rise in pressure during these manoeuvres seems to depend on the height of the resting pressure and the degree of cartilage destruction (Arnoldi 1990).

Intra- or extravascular blockage of venous circulation inside the bone marrow

The first stages of non-traumatic femoral head necrosis are characterized by high bone-marrow pressure of the femoral head yielding pulsatile pressure tracings (Ficat and Arlet 1980), and the pressure reaches its highest values just under the osteochondral junction. Intraosseous phlebography shows impeded venous drainage and intraosseous stasis, and there is increased uptake of bone-seeking isotopes (Ficat and Arlet 1980). In the later stages, the necrotic area, most often starting as the crescent sign near the osteochondral border, becomes pulseless but the pressure inside the necrosis is still high, while the flow is slow. At all stages, the pressure in the vascularized bone seems to react to rotation and loading in the same way as in arthrosis. The cause and site of intraosseous vascular blockage are not known, although both intra- and extravascular blockage have been reported.

Pressure differences between the patella and the femur and tibia

The measurements generally showed higher pressure in the patella than in the femur and tibia in pain-free control knees, as well as in PP knees. Sustained flexion accentuated these pressure differences. The results of the various experiments and manoeuvres performed during our investigations indicate that the difference in pressures and pressure variations observed are due mainly to the following circumstances:

1) difference in bone structure;
2) different conditions for venous drainage in the three areas of measurement;
3) different effects on bone marrow pressure of the mechanical forces affecting the skeletal components of the knee joint during loaded flexion and extension.

Anatomy

The patella belongs to the group of short bones. It consists of a densely woven cancellous network surrounded on all sides by a solid, but still elastic cortical shell and, in the age group with which we are concerned, it is highly vascularized. In contrast, the femoral and tibial condyles are parts of tubular bones, and their cancellous bone marrow merges into loosely structured diaphyseal bone marrow extending far beyond the knee region (and our points of measurement). These anatomical facts ensure that an increase of intramedullary pressure, caused by bone deformation for example, may disperse over a large area, from the marrow of the condyles upwards through the femur and distally through the tibia. In the closed marrow of the patella this pressure dispersion is negligible. Release of intraosseous pressure is only possible through drainage to extraosseous veins, not as in the two other areas of measurement through both intra- and extraosseous drainage channels.

Venous drainage

The drainage systems from the patellar bone marrow are, with one exception, highly exposed to extraosseous blocking forces. As seen from the phlebographic studies, maximal flexion of the knee usually compresses the veins leaving the anterior surface and the proximal border of the patella. This happened in both control and painful knees. The veins leaving the medial and lateral borders are exposed to changes in the peripatellar synovium through which they pass, and they, and all other veins mentioned, may be compressed by capsular stretching during flexion.

The large cluster of veins leaving the apex patellae is generally visible during flexion, but may apparently become affected by compression of the patellar ligament during extreme flexion and by changes in the fat pad. Narrowing of these veins was a frequent observation in PP knees during flexion and was sometimes also observed with the knee in extension. The majority of phlebographs indicated that of the patellar veins the apical drainage had the largest capacity.

Intraosseous venous drainage

Phlebographic study of the knee in extension gave no clearcut picture of intraosseous venous stasis in patellae of PP knees. Extraosseous veins always disappeared very quickly from the radiographs. It is true that delayed emptying was considerably more common in painful than in control knees, but the flow from the bone marrow was unimpeded in one third of cases, almost all of whom had a loose network of relatively large intraosseous vessels. The lack of consistency may have something to do with the character of the patient material. All patients had one symptom in common: typical, severe and long-standing patellar pain. While arthrotic knees were excluded, no other effort was made to divide the material according to (possible) etiology. However, retrospective study, resulting from the different intraosseous vessel patterns, suggests that different etiology may produce different phlebographic pictures. Thus, the patients with Type 1 pattern, which generally shows delayed intraosseous retention of contrast material, was predominantly a younger group with overuse or malalignment syndromes, while the Type 2 group was dominated by post-traumatic cases, including cases with manifest osteopenia or reflex sympathetic dystrophy. This observation may deserve further investigation.

The phlebographic results from maximal flexion are more uniform: sustained maximal flexion closes all drainage channels, except the apical veins and may even affect these vessels. This exit is either too narrow to allow drainage of contrast medium, or there may be other, intraosseous, factors that are co-active in the prevention of drainage from bone in sustained maximal knee flexion.

Physiological considerations

Blockage of the apical veins by compression of Hoffa's pad always caused varying degrees of elevation in intrapatellar and tibial pressure. In the patella it reached a level of 40-50 mmHg above the extraosseous vein pressure. This was also true in cases where the pressure and sustained flexion tests were positive. At this intraosseous pressure level the pulse waves had increased in size, as they always seem to do when intraosseous pressure is raised by increased resistance to flow in extraosseous drainage vessels (Arnoldi et al. 1972). Non-pulsatile pressure tracings were never observed when the intraosseous pressure rise was due to extraosseous venous blockage alone. Such tracings are similarly always pulsatile from the femoral head and from the femoral and tibial condyles in arthrosis, or the intraosseous engorgement-pain syndromes (Arnoldi et al. 1971, Arnoldi et al. 1975). These tracings and the very high maximum and plateau pressures in the patella during sustained knee flexion indicate that factors other than blockage of extraosseous venous drainage are influential.

Patellar compression and sustained knee flexion

In pain-free, control knees, manual compression of the patella against the femoral trochlea caused an immediate rise of patellar pressure to approximately twice that obtained in the same patellae by blockage of apical veins. This reached the same level in controls as the plateau pressure in PP knees (70–80 mmHg above extraosseous vein pressure). At this level the patellar tracings were always non-pulsatile. Plateau levels in the femur and tibia might come very close to the patellar pressure, but non-pulsatile tracings were never observed from these points. It is interesting to note that in the first stages of non-traumatic osteonecrosis of the femoral head, where pressure in the head frequently reaches 70-80 mmHg, the pressure tracings are always pulsatile in the unloaded joint (Ficat and Arlet 1980).

Thus, the pulse amplitudes in PP patellae and in juxtachondral bone marrow in arthrosis increase when the intraosseous pressure rises above a certain level, due to compression of the drainage veins in their extraosseous course (Figure 4). This phenomenon is probably caused by a release of the veno-arterial reflex with compensatory dilation of arterioles (Nielsen 1984).

Tissue tension

Burch and Sodeman (1937) defined tissue tension as the pressure with which tissue structures resist changes in their anatomical relations. Such pressure varies considerably from tissue to tissue. Thus, in the lower leg tissue tension is always lower subcutaneously than intramuscularly, and among the muscles those with a tight fascia have higher tissue tension than those with loose fascial coverings, both at rest and during muscle contraction (Wells et al. 1937). Apart from the structure of the muscle fascia, muscular tissue tension was found to depend on the amount of extravascular fluid, and the degree of filling of the blood vessels.

Apart from measurements of intraosseous pressure, no measurements of tissue tension in bone and cartilage seem to have been made. Cartilage has a low compliance, and tissue tension must increase to considerably higher levels than tension in subcutaneous and muscular tissues, at even small increases in tissue volume.

Influence of tissue tension on blood flow

High resistance to capillary flow due to increased pressure at the venous side of the circulatory system leads to oedema of the tissue involved. Edema increases tissue tension and it is generally agreed that such increased tension tends to reduce blood flow, either due to arteriolar closure at high extravascular pressure (Burton and Yamada 1951), or to a reduction of local arteriovenous pressure difference and, hence, blood flow, as local venous pressure increases equal to the rise in the surrounding tissue pressure (Ryder et al. 1944).

In studies of patellar pressure under various conditions it was noted that in this limited and non-compliant bone-marrow space, increasing intraosseous pressure (tissue tension) affected the pulsatile excursions of the pressure tracings from the marrow. Under the circumstances of the experiments, an increase of intraosseous pressure to the range 40–60 mmHg enlarged the pulse waves. However, if the pressure increased further, the tracings became non-pulsatile.

Nielsen (1984), in experiments on soft tissue arteries of the lower limb, observed that pre-capillary vessels collapsed with cessation of blood flow when the (effective) diastolic transmural arterial pressure was reduced to zero, and Sejrsen (1990) stated: "When the tissue pressure reaches values corresponding to diastolic arterial pressure, the arterioles will be compressed during diastole. Under these circumstances the arterioles present a very high resistance to flow and they will not be refilled during systole. The result is a cessation of blood flow".

In the patella this state is intermittent and the death of osteocytes from prolonged anoxemia is not characteristic, as in bone marrow with chronically high juxtachondral pressure at rest, abetted by periods of even higher deformation pressure (arthrosis, non-traumatic femoral head necrosis, etc.).

Pressure at rest, intermittent hypertension, pain and chondromalacia

Pressure at rest

In patients with PP and chondromalacia, the intraosseous pressure of the patella at rest and with the knee extended is higher than in pain-free controls (Björkström et al. 1980). Hejgaard and Arnoldi (1984) found in a material with severe PP that those with pain at rest had a higher pressure than those without. The histological findings may indicate that this pressure rise may be due to resistance to venous drainage through some of the drainage systems caused by the circumscribed parapatellar synovitis. However, this patellar rest-pressure rise rarely reached the levels observed in painful arthrosis and non-traumatic femoral head necrosis.

The material comprised cases both with and without chondromalacia. It is of interest that in the enlarged material presented here there was no patellar rest pressure difference between painful knees with or without visible chondromalacia. This fact, in combination with the histologic findings suggest that increased intrapatellar pressure is the cause, rather than the effect of the cartilage changes.

Intermittent patellar hypertension

Sustained maximal flexion of the knee joint simulating situations that provoke typical pain, is accompanied by a considerable increase in patellar pressure, greater in painful than in control knees. With plateau pressure above a certain level the sustained flexion test becomes positive, i.e. painful, again independently of the presence or absence of demonstrable chondromalacia.

Thus, at rest in extension, as well as during maximal sustained knee flexion, the pain felt seems to be dependent on the height of pressure in the patellar bone marrow, but independent of the state of the cartilage, at least as judged by the criteria used for staging of chondromalacia.

Changes in patellar microcirculation, pain and chondromalacia

The microscopical observations of Darracott and Vernon-Roberts (1971) on patellae with and without visible signs of chondromalacia and those of Badalamente and Cherney (1989) on chondromalacia patellae showed the same picture of vascular penetration of the osteochondral junction by arterial and venous vessels superficial to a duplicated or reduplicated tidemark as reported in this study.

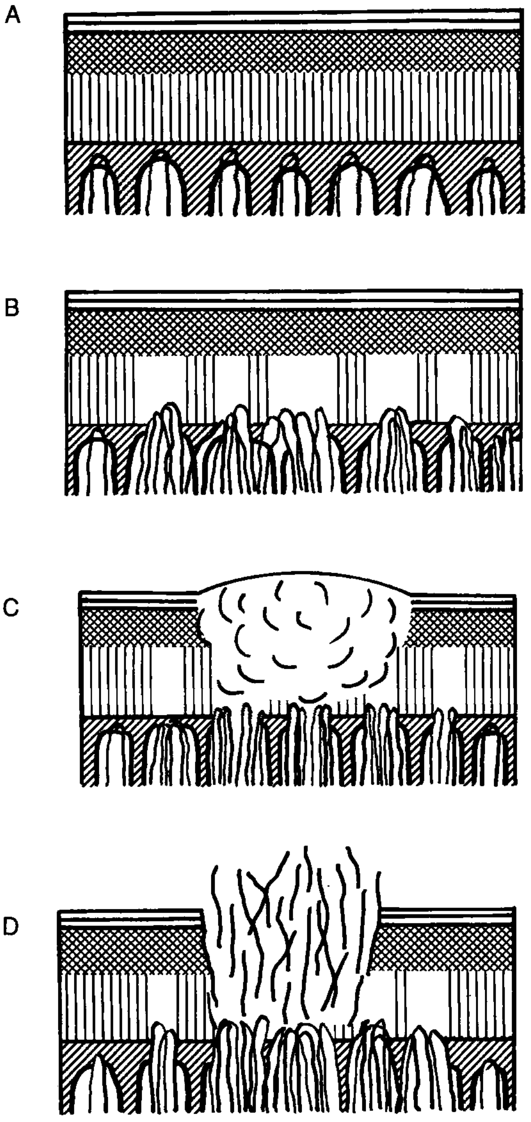
Pain

The results of our pressure measurements, in combination with the histological findings, indicate intermittent local intraosseous venous hypertension and vessel dilation as the cause of capillary proliferation. Vessel dilation is known as a pain inducer (Arnoldi 1989) and our results indicate that high transmural pressure in the increased number of vessels during periods of high non-pulsatile intraosseous pressure (and probably stagnant blood flow) may trigger release of neurotransmitters of sensory afferent pain. The pain-free interlude during the sustained flexion test would indicate that the release of these transmitters is gradual.

Basal cartilage degeneration

Ficat and Hungerford (1977) located the first signs of abnormality in the intermediate layer of articular cartilage, whereas Goodfellow et al. (1976) pointed to the basal layer as the site of the first degenerative changes. The authors of both papers ascribed the development of chondromalacia patellae exclusively to mechanical causes, such as abnormal contact pressure and shearing during movements of the patellae against the femoral trochlea.

At this point it would, however, seem appropiate to note that during the first stages of chondromalacia the changes are confined to the cartilage of the patella. They do not occur in the femoral contact area, where pressure on cartilage must be equally high, until Stage



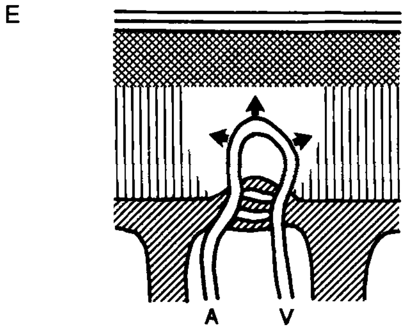


Figure 23. Development of chondromalacia

A. Normal stage. The osteochondral bone plate is solid. The relatively few vessels in the area reach the Haversian canals, but do not penetrate into cartilage. The tidemark forms a single layer (Darracott and Vernon-Roberts 1971, Goodfellow et al. 1976, Badalamente and Cherney 1989); own observations, see section on histology.

B. Indicates the osteoporosis of cancellous bone, the thinning of the osteochondral bone plate (Darracott and Vernon-Roberts 1971), the multiplication of vessels in this region (Badalamente and Cherney 1989), the penetration of vessels and nerves into the basal cartilage layer (Darracott and Vernon-Roberts 1971, Badalamente and Cherney 1989, Bridgeman and Brookes 1990; own histologic observations) and the fasciculation of collagen fibres perpendicular to the joint surface (Goodfellow et al. 1976, Ficat and Hungerford 1977) by matrix edema (own assumption).

C. Expanding edema and changes in matrix and cell quality lead to the circumscribed "blister lesion", soft to the touch and probe.

D. The weakened edematous cartilage explodes, due to compressive and shear forces during loaded knee joint movements.

E. "The patologic unit". A vessel loop in cartilage during a moment of high resistance to venous flow, e.g. sustained maximal flexion of the knee joint. The increased hydrostatic pressure at the venous end of the loop is responsible for edema, and water retention in the matrix is augmented by raised protein osmotic pressure in the interstitial water compartment. The composition of the matrix and chondrocyte environment is probably severely affected and this may well be the fundamental reason for the succeeding cellular degeneration.

3, the stage of patellar arthrosis. Up to this point the disorder is apparently "asymmetrical". Undoubtledly, mechanical forces of the type mentioned play an important role in the development of chondromalacial changes, but the asymmetrical development should make us consider whether other factors are influential.

The reports by Darracott and Vernon-Roberts (1971) and Badalamente and Cherney (1989), the observation by Bridgeman and Brookes (1990) and our own histological findings suggest that the initial changes do in fact occur in the basal layer and that the disorder may be triggered by vascularisation with associated changes in chondrocyte and matrix environment.

What these changes are and how the disorder progresses through the various stages is still far from clear. However, vascularisation of cartilage exposes this tissue to intermittent venous hypertension and oedema. We know that intermittent venous and intraosseous hypertension is responsible for localized soft tissue degeneration and skeletal changes at the ankle in patients with chronic venous insufficiency of the lower limb (supra-articular intraosseous hypertension; Arnoldi et al. 1972) and the very severe damage caused by intermittent capillary hypertension, in contrast to chronically increased pressure was demonstrated by Boersma and van Limborgh (1967) and Fagrell (1972).

Stages of basal cartilage degeneration

The author's interpretation of the early changes and the development leading to manifest chondromalacia patellae (Figure 23) is based on the concept of Goodfellow et al. (1976), but modified by recent additions to our knowledge.

Conclusion

It is in accordance with clinical observations, the pressure measurements and histological findings presented here, that PP, due to venous and capillary hypertension and distension, is the initial symptom of a vascular disorder, and that the changes in articular cartilage leading to gross chondromalacia are secondary manifestations of this. In the femoral trochlea, which is exposed to the same forces of compression as the patella, the open structure of the bone marrow prevents a build-up of intraosseous pressure equal to that in the patella. This would be the most probable reason for the rare and late cartilage involvement of the femur.

Therapy

The evidence presented here points to two mechanisms: impeded venous drainage through extraosseous veins and intraosseous pressure increase by deformation-compression of the patella. Both are involved in the pathogenesis of PP by increasing the resistance to venous and capillary flow in and from the patellar bone marrow and accompanied by vessel distension. The pressure increase by compression of the patella would appear to be the more important and to give rise to the characteristic accentuation of pain on loaded knee flexion. Other forms of venous blockage may play a role in inducing increased intraosseous pressure in the relaxed knee and, thus, inducing pain at rest. Both mechanisms may provoke pain via release of pain transmitters, when the minute vessels are distended. Histological evidence indicates that one of the main changes is vascularisation and innervation of the normally avascular and nerveless cartilage.

Conservative therapy

Clinical experience indicates that the pain in PP may be reversible if the high intermittent intraosseous pressure is lowered for a prolonged period. The initial management of PP should therefore always be conservative, particularly in young people with pain arising from overuse caused by excessive sports activity. A pause of at least six months during which all pain-provoking exercise of the knee is studiously avoided will bring relief in most cases, as long as the patients, their parents and their trainers understand the reasons for the regimen and follow it strictly. Today it is extremely important that young patients are convinced of the absolute necessity of these measures. The social life (and status) of many young athletes is intimately interwoven with their direct and indirect sports activities and many cannot accept the idea of relegation to the side lines, merely on the advice of their doctor. The increasing number of people under the age of 35, mostly women, who are now permanent invalids with a history of several unsuccessful operations is, in my experience, primarily the result of insufficient information, instruction, support and follow-up during the period when recovery would still have been possible.

Isometric, painless, quadriceps exercises and patellar bracing are often effective conservative measures, although how exercising the quadriceps can be effective, when the majority show no signs of quadriceps atrophy, is poorly understood; a venous pump effect on the drainage through the proximal and marginal veins cannot be excluded. However, the psychological benefits of a training program during such a period of forced athletic inactivity may make it easier for the sports-orientated patient to follow doctor's orders. The effect of these excercises may thus be indirect.

In the few cases of non-arthrotic PP presenting with signs of general knee synovitis, a period of treatment with anti-inflammatory medication may be useful, but in the majority of cases this treatment has no effect.

Surgery

It should be repeated that surgery should not be undertaken as the primary therapy for PP. Controlled conservative treatment of at least six months duration may lead to full recovery or at least to a state where the level of discomfort is acceptable to the patient. The main reason for this very necessary conservatism is that the ultimate results of surgery are unpredictable, and one unsuccessful operation too often is followed by a second, third and fourth with increasingly poor results (see the description of our patient material).

Surgical methods

The surgical procedures en vogue in recent decades are legion, but in principle they fall into these groups:

A. Procedures that are performed in order to diminish abnormally high local or general contact pressure between the patella and femur (in our terminology, to reduce compression-deformation of the patella).

B. Core decompression (fenestration) of the patella in patients with high intraosseous pressue and abnormal intraosseous phlebographs (improving the drainage from the patellar bone marrow).

C. Local or general removal of patellar cartilage. Resurfacing of the patella comes into this category.

D. Patellectomy.

E. Patellar osteotomy (improving the drainage from the patellar bone marrow and opening of the closed patellar bone marrow space).

A. Joint decompression and correction of malalignment

The common aim of the many surgical interventions to correct malalignment is to reduce the pressure on the lateral patellar joint facet

Lateral release (division of the lateral retinaculum and the lower fibres of the vastus lateralis) is a relatively simple procedure, especially when done through the arthroscope. It has been extensively used and the initial results show considerable success (Larson et al. 1978, Ceder and Larson 1979). Ficat and Hungerford (1977) employed lateral release, often combined with surgery on patellar joint cartilage and/or drilling of the subchondral bone plate. They reported 76 percent excellent and good results in a series of 174 cases of "excessive lateral pressure syndrome". Their patients were followed for at least 6 months. Insall (1982) was less optimistic, stating that in his experience only about half of the knees treated with lateral release will improve.

Proximal realignment. Insall et al. (1976) combined an extensive lateral release with a vastus medialis advancement in order to alter the line of pull of the quadriceps muscle. In their opinion this procedure should be considered for the most disabled patients. Six years later, Insall (1982) stated that when used under these circumstances a 90 percent incidence of good and excellent results can be expected.

However, a recent paper by Abraham et al. (1989) reported only 55 percent satisfactory or excellent results in patients with chondromalacia (follow-up 5-11 years). These authors are of the opinion that the operation should be reserved for recurrent patellar dislocation and very advanced chondromalacia. Further, their studies suggested that pain relief was due mainly to patellar denervation.

Distal realignment. Most of the variants of this surgery involve medial transposition of the tibial tuberositas (Roux 1888, Hauser 1938, Trillat et al. 1964, Hughston and Walsh 1979). In all reports, early results were highly promising, but other authors have reported a high incidence of late arthrosis after the once very popular Hauser's operation (Hampson and Hill 1975, Crosby and Insall 1976). This may be due to the fact that medial transposition of the tibial tubercle, because of the triangular shape of the proximal part of the tibia, is always accompanied by posterior displacement. This actually increases the articular pressure in the patellar articulation (Ficat and Hungerford 1977). Modifications of this procedure by Emslie-Trillat (Trillat et al. 1964) and Goutallier and Debeyre (1974) avoid excessive medial-posterior transfer. Again, the early results of these modifications were reported to be good.

Advancement of the tibial tuberosity. Maquet (1963, 1976) described a procedure with anterior advancement of the tibial tubercle for PP patients. In a theoretical model, he demonstrated that by moving the tibial tuberosity 20 mm anteriorly, the pressure of the patella against the femur was reduced by approximately half.

Ferguson et al. (1979) argued that a 10 mm elevation is sufficient. The results reported after the Maquet operation vary considerably. The rate of excellent and good results after a short follow-up ranged from 85 to 100 percent (Maquet 1970, Sudman 1980, Ferguson 1982, Svartveit et al. 1983). However, three Scandinavian studies (Lund and Nielsen 1980, Uppheim et al. 1982, Engebretsen et al. 1989) with longer follow-up showed decreasing effect of the operation with time. Thus, Engebretsen et al. (1989) reported that only 10/33 patients showed lasting improvement, while 7 others had experienced temporary improvement for up to two years after surgery.

B. Core decompression

Ficat and Hungerford (1977) used this procedure in a series of 15 patients with reflex sympathetic dystrophy and recorded diminution of pain and improvement of mobility in the majority. These patients displayed increased intraosseous pressure at rest with the knee in extension and delayed emptying of contrast medium after *femoral* phlebography. Postoperative reduction of intraosseous pressure was demonstrated, and the improvement was stated usually to persist, but no length of follow-up was given.

C. Patellar cartilage surgery

Patellar shaving has been used for many years (Wiles et al. 1960) and is still performed in many clinics as the sole intervention. The realisation that chondromalacia is often present in a joint without pain, while many knees with typical PP have apparently intact cartilage, has resulted in widespread scepticism of the usefulness of this operation.

Debridement (excision of the cartilage lesion and drilling of the subchondral plate) can be useful in arthrosis (Insall 1982), and Ficat and Hungerford (1977) used the procedure in patients with painful chondromalacia in combination with surgery for malalignment. They believe the cartilaginous lesion to be the result of mechanical wear and tear and the source of the symptoms. Of 38 operations including debridement (chondrectomy), all but three had excellent or good results. Patients were followed for 6 months or more.

Resurfacing of the patella is used with satisfactory results as an integral part of total knee replacement in arthrosis. In younger patients with non-arthrotic PP the results of these procedures are unpredictable and the operation should not be used (Insall 1982).

D. Patellectomy

As seen from the description of our patient material, a large proportion had already been through multiple operations with steadily increasing disablement as the result. Patellectomy remains the ultimate option for intractable PP although the costs may be heavy, especially weakening of knee extension, and most surgeons are unwilling to consider patellectomy as a primary procedure. Bentley (1970) found, however, that primary patellectomy gave better results than patellectomy performed as a salvage procedure in knees with multiple operations, and Jensen and Hansen (1989) came to the same conclusion. However, these authors and most others recommend preservation of the patella whenever possible.

E. Patellar osteotomy

Arnoldi et al. (1971) demonstrated that high intraosseous pressure in the femoral head and neck in patients with painful coxarthrosis, fell abruptly when the cortex was opened, either by osteotomy or by fenestration (forage, core decompression), and that these procedures were followed by relief of the characteristic pain at rest within 24 hours.

Morscher (1978) reported a favourable response to longitudinal osteotomy of the patella in the sagittal plane in patients complaining of severe PP. He attributed the effect of the operation to correction of incongruities in the patella articulation, resulting in an increased area of cartilage contact during certain phases of knee flexion, and an improvement of nutrition of patellar cartilage. He also mentions the possible benefit of reduced intrapatellar pressure.

Nerubay and Katnelson (1984) reported 15 cases of PP resulting from patellar malalignment on whom they performed coronal plane osteotomy. The results after three years were favourable in 14. They attributed the relief of pain both to the reduction of intraosseous pressure, including associated biological adaptations, and to the mechanical realignment of the patella made possible by a shift between the anterior and posterior patellar fragments.

Material and methods

The group studied by Hejgaard and Arnoldi (1984) comprised 40 patients with very severe PP of differing etiologies. They are included in the total patient material reported above and were examined preoperatively in the same manner and by the same methods.

Through an anterior incision, approximately 4 cm long, the patella was osteotomised longitudinally using a chisel. The articular cartilage was preserved and allowed the osteotomy to be opened up. A distance of 3–4 mm between the anterior rims of the ostetomy was maintained by insertion of a small block of cortical bone taken from the superficial part of the osteotomy. The patients were mobilized immediately (several were operated under local anesthesia as outpatients), as fully weight bearing. At the end of two weeks, increasing exercise against resistance and movement exercises were prescribed. Full activity, including sports, was allowed at the end of six weeks.

Results

The immediate result of the osteotomy was an abrupt fall of bone marrow pressure in the patella (Figure 24).

As the indication for the operation in all cases was pain, the result was evaluated solely by quantifying the residual pain, using a visual analogue scale. Scott and Huskisson (1976) have demonstrated that this method is reproducible. Zero was selected as the lowest value (no pain) and one hundred as the highest.

The patients were reviewed 5–19 months after the operation. At that time (1984) we found a substantial relief of pain following osteotomy. The three patients who did not improve after operation had normal patellar pressure in extension and a negative sustained flexion test. A follow-up of this material in 1989 showed, however, that only 16 of 37 patients who five years before had been catalogued as painfree results, still considered themselves to be so; in the remainder, the patella-related pain had recurred.

Comments

The results published by Morscher (1978), Nerubay and Katnelson (1984) and Hejgaard and Arnoldi (1984) agree on the beneficial effect of patellar osteotomy on severe PP and Nerubay and Katnelson (1984), as well as Hejgaard and Arnoldi (1984) attribute this effect to a decrease of intraosseous pressure in the bone marrow

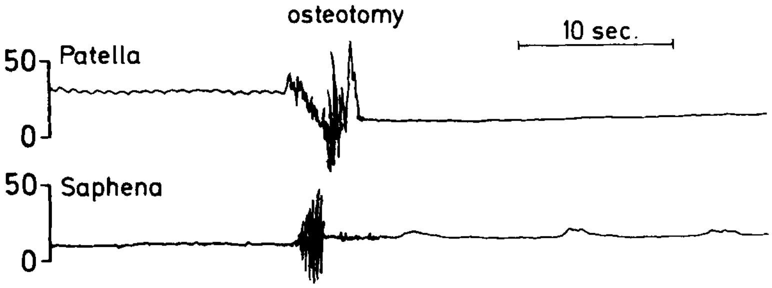


Figure 24. Pressure tracings from the patella and the internal saphenous vein, before, during, and after patellar osteotomy.

of the patella. The results of the investigations reported above point to a lowering of patellar plateau pressure during knee flexion as the most important factor. Our experiments indicate that the decrease of this pressure is due both to a decrease of resistance to venous outflow from the marrow and especially to a decrease of the effect of mechanical deformation on patellar bone marrow pressure during loaded flexion. The apparently longer-lasting effect of coronal osteotomy on pain from malalignment when compared with the results of longitudinal osteotomy on the mixed PP group of Hejgaard and Arnoldi (1984), may be due to the possibility of malalignment correction inherent in coronal osteotomy. However, in the context of the pathophysiology of pain the interesting fact is that both operations lowered intraosseous patellar pressure and were followed by immediate decrease or disappearance of PP.

Discussion and conclusion

According to the reports cited in this section, the results of surgery seem to be rather unpredictable. While most reports after a short follow-up are optimistic, most series with a follow-up over 3–6 years report success in alleviation or reduction of PP in only 30–60 percent. The exceptions are Insall's (1982) statement that after six years good results can be expected in 90 percent of severely invalidated patients treated by proximal realignment, and perhaps Nerubay and Katnelson's (1984) report of the results of coronal patellar osteotomy.

Looking at the subject of surgery as a whole a consensus seems to be taking form: most surgical interventions are—more or less consciously—directed against excessive compression-deformation of the patella during loaded flexion, and, judging from the immediate results, most methods are undoubtedly effective in reducing the high plateau pressure associated with PP. However, it seems that we still have not found a method to keep this pressure down permanently.

Add to which, many postoperative complications, especially patellar arthrosis and long-standing synovitis, can be traced to two main sources. The first is surgery that has changed the mechanical conditions in a way that increased, instead of diminished, the pressure of the patella against the femoral condyle (e.g. the posterior-medial transfer of the tibial tubercle in Hauser's and related techniques), and secondly, intra-articular interventions leading to traumatic synovitis, perhaps maintained by permanent weakening of the quadriceps muscle which also weakens the pump effect on knee joint fluid.

It is a common clinical experience, especially in overuse and malalignment syndromes, that "active", controlled and prolonged conservative treatment often leads to disappearance of patellar pain. It is also evident from clinical experience that negligence and ignorance of its importance leads to disablement in a large number of patients. This might mean that the process is reversible, at least up to a point. Where this point of no return occurs cannot be decided from the experiments referred to here, and it may differ in different forms of PP. However, the experience gathered indicates that the battle is lost at the time when the blister lesion has rendered the cartilage susceptible to the heavy compression and tearing forces involved in loaded flexion and extension of the knee joint. This is the time when most surgery for PP is performed, and, looked at from this point of view, the generally poor long-term results of most types of surgery are understandable.

The evidence of the investigations referred to here, together with the abundance of clinical experience from the literature, serves to stress the importance of the rigid conservative regimen. Unfortunately, most patients are more influenced by their own ambitions and those of their parents, teachers and trainers, than they are by the often conflicting opinions of the members of the medical profession.

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Carl C. Arnoldi

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