



Review article

ANTERIOR KNEE PAIN

Andrew M. Jackson

From London, England.

Troublesome anterior knee pain is a common problem in active adolescents and young adults and the complex of symptoms is well known.^{1,2} From a clinical viewpoint it is convenient to consider the causes under the headings of 'Distinct' and 'Obscure' (Table I). The first group consists mainly of focal lesions which can be clinically and radiologically defined and for which local treatment can be applied and outcomes predicted. The second includes dynamic problems, such as maltracking and the excessive lateral pressure syndrome, as well as idiopathic chondromalacia and psychogenic pain.

The frequency of anterior knee pain is related to the very considerable compression and shear forces which are transmitted through the patellofemoral joint. One important function of the patella is to displace the patellar tendon away from the centre of rotation of the knee and so increase its moment arm. The contact point between the patella and the trochlear groove is a fulcrum and on the patellar side of the articulation an area of contact sweeps up from the inferior to the superior pole as the knee flexes from full extension to 90° of flexion.³ Static experiments show that at 60° of knee flexion over twice the body-weight is transmitted through the patellofemoral joint, while dynamic studies in serious weight-lifters suggest a patellar tendon tension of 17.5 times the body-weight when pushed to the extreme.^{4,5} It is therefore not surprising that the patellofemoral joint has the thickest articular cartilage of any joint in the body and that peripatellar failure of the extensor mechanism is such a common problem. Acute failure is of course manifest as 'fracture' or 'rupture' while subacute and chronic failure give rise to anterior knee pain. The origin of the pain is a subject of debate since articular cartilage is devoid of nerve endings. Cartilage also has very limited powers of repair and regeneration once fibrillation and ulceration have occurred.

Overuse and repeated minor trauma commonly play a part in the production of anterior knee pain, especially when sufficient time is not allowed for the resolution of subliminal damage. Paradoxically, underuse can also be incriminated as a cause of damage to the articular surface, an example being the chondromalacia which is seen on the medial or odd facet of the patella. This small area does not come into contact with the femur until the knee flexes past 130° and therefore in most patients is a very underused part of the articular surface. Osteoporosis of the patella often accompanies anterior knee pain although it is difficult to know if it is a cause or effect. It reduces the stiffness of the patella and causes thinning of the subchondral bone plate, thus decreasing the ability to cope with the huge forces already referred to.

Distinct causes of anterior knee pain

Overuse

Juvenile traction osteochondritis. Osgood-Schlatter disease is commonly seen in boys aged between 12 and 14 years and less often in girls and at a slightly younger age. The diagnosis is easy if the patient points to the tibial tubercle and a prominence is already obvious. Kneeling is painful. The condition occurs at a time when increasing demands are made on a still immature skeleton. The adult patellar tendon is firmly anchored to bone by Sharpey's fibres, but in the growing child the attachment is more tenuous (Fig. 1).⁶ Repeated microavulsion injuries accompanied by half-hearted fibro-osseous repair results in prominence of the tubercle. Its fragmented appearance on radiography is due to partial separation of chondro-osseous fragments. Very rarely, infection or trauma may masquerade as Osgood-Schlatter disease and plain radiography should be a routine procedure, especially in the unilateral case.

Treatment is conservative in the first instance, reducing activity to a level at which symptoms become manageable. Short-term immobilisation in a cast is very rarely necessary and bilateral application is a definite mistake since it will result in a very disabled child. The natural history is for the symptoms to disappear within one to two years, leaving the bony prominence as a permanent marker of the event. If symptoms remain troublesome into the late teens, radio-

A. M. Jackson, FRCS, Consultant Orthopaedic Surgeon
Trauma and Orthopaedic Department, St George's Hospital, Blackshaw
Road, London SW17 0QT, UK.

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Table I. Causes of anterior knee pain

Distinct	Obscure
Overuse Osgood-Schlatter, Sinding-Larsen-Johansson, jumper's knee, quadriceps tendinitis, bipartite patella, stress fracture	Idiopathic knee pain
Trauma-related lesions Osteochondritis dissecans, bone bruising, post-traumatic degenerative change	Chondromalacia
Syndromes and dysplasias	Maltracking, subluxation/tilt
Tumours Dorsal defect of patella, giant-cell tumour, osteosarcoma, etc	Excessive lateral pressure syndrome
Miscellaneous Plicae, bursae, patellar tendon ossification, posterior cruciate ligament rupture	Reflex sympathetic dystrophy
Iatrogenic Hauser procedure, infrapatellar contracture syndrome, neuroma	Psychogenic

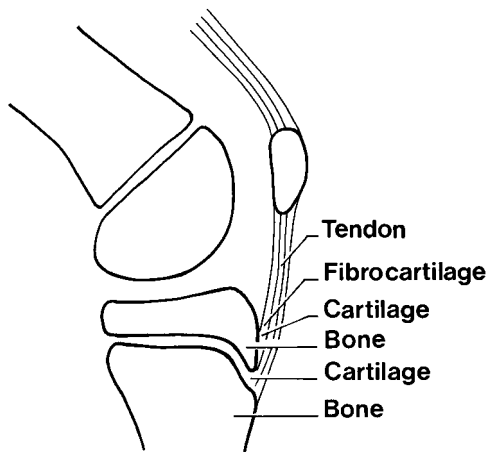


Fig. 1

Diagram showing the tenuous anchorage of the patellar tendon in the immature skeleton (reproduced with permission of Harcourt Publishers Ltd).

graphs will probably show an ossicle in the substance of the tendon adjacent to the tubercle. Removal of the ossicle and sometimes an associated bursa will be curative and most patients are asymptomatic in adult life.⁷

Sinding-Larsen-Johansson disease occurs in the same age group, but the point tenderness and pathology are located at the lower pole of the patella. The lateral radiograph of the patella may be normal or show some speckled calcification in the patellar tendon adjacent to the lower pole. Treatment is as for Osgood-Schlatter disease. Usually the area of speckled calcification heals, leaving the familiar elongated lower pole of the patella which is so often seen in footballers. Occasionally, a troublesome ossicle persists and requires excision. Very rarely, traction osteochondritis similar to Sinding-Larsen-Johansson disease may be seen at the proximal pole of the patella.⁸ The differential diagnosis of abnormal calcification at the lower pole of the patella lies

between Sinding-Larsen-Johansson disease, bipartite patella, stress fracture and impending sleeve fracture.

Jumper's knee. This is the name given to tendinitis at the tendon-bone interface at the lower pole of the patella after skeletal maturity. Again the problem is overuse and jumping is not the only cause. Focal tenderness at the lower pole of the patella is the key physical sign and this can often be eliminated if the patient is asked to do a straight-leg raising test and to hold the leg a few inches off the couch. This shows that the tender area can be protected by the tensed superficial fibres of the patellar tendon and that the problem lies at a deeper level.

Conservative treatment demands a period of rest, perhaps combined with pain-relieving physiotherapy which includes ultrasound, deep friction, isometric exercises and hamstring stretches. Oral anti-inflammatory agents can be helpful but local injections of steroids are contraindicated for fear of rupture of the tendon. If conservative treatment fails and ultrasound or MRI shows abnormalities in the substance of the tendon, surgical exploration may be indicated in that small group of patients who are professional athletes and who refuse to give up sporting activities which exacerbate the condition.

A variety of different procedures has been recommended, but not surprisingly the concept of inflicting surgical trauma on a degenerative tendon in order to stimulate healing does not always work. The simplest operation is to split the tendon at the site of maximal tenderness and, if a granuloma is found, to scrape it away with a curette. If the deeper half of the tendon is involved it can be stripped off the lower pole of the patella and some authors have advocated drilling of the distal pole in order to bring in a new blood supply.⁹ The knee should be protected in a splint for four weeks before beginning a rehabilitation programme. For recalcitrant cases in top athletes, Blazina et al¹⁰ claim success with resection of the lower pole of the patella and reattachment of the tendon. More recently, it has been proposed that the cause of the problem is impingement of



Fig. 2a



Fig. 2b

Radiographs of a 12-year-old boy showing a) a widely displaced sleeve fracture and b) the opposite knee with the characteristic bipartite lower pole. Separation occurs through the line of the synchondrosis. If pain is associated with such a picture there must be concern about impending separation (reproduced with permission of Harcourt Publishers Ltd).



Fig. 3

Radiograph of an adult footballer with acute-on-chronic anterior knee pain. The right patella is tracking better perhaps because the stress fracture has acted as an autolateral release.

the deeper fibres of the patellar tendon against the lower pole of the patella in flexion rather than traction. Accordingly, arthroscopic trimming of the lower pole of the patella has its advocates.¹¹ This procedure not only brings in a new blood supply but also exposes the deep fibres of the patellar tendon at its origin to synovial fluid, which may be beneficial.

Bipartite patella. This is common in childhood. It is often bilateral and usually regarded as a variation of normal ossification. The two centres may proceed to fusion in the second decade. Very rarely, in response to overuse or acute injury, the synchondrosis separating the two centres may become painful and the site of local tenderness. There are three sites at which bipartite patella is found and each has an important soft-tissue attachment:

- 1) The distal pole of the patella with attachment of the patellar tendon.
- 2) The lateral margin of the patella with attachment of the lateral retinaculum.
- 3) The superolateral corner, the insertion of vastus lateralis. This is the commonest site for symptoms.

It is conceivable that in some patients the type-1 lesion may represent the endstage of Sinding-Larsen-Johansson syndrome. In others, there is no doubt about the bipartite nature of the lower pole of the patella and in this context the sleeve fracture of the patella is a fascinating entity.¹² Perhaps patients with a bipartite lower pole with its characteristic appearance are at high risk of sustaining a fracture (Fig. 2). Certainly, if such a patient presents with pain and

tenderness at the lower pole, this should be viewed as indicating separation, and activities should be curtailed. The fracture, when it occurs, is along the line of the synchondrosis; the avulsed distal pole is largely cartilaginous. It is always much larger than the radiographs suggest and must be reattached.

The type-2 lesion, if not bipartite, may be caused by direct injury or by a stress phenomenon related to a tight lateral retinaculum and maltracking of the patella. A mobile lateral marginal stress fracture of the patella with the synovial fluid gap at the site of the injury may perhaps represent an autolateral release of a tight retinaculum (Fig. 3).

The type-3 lesion is the commonest cause of symptoms which occasionally are severe enough to merit surgery. The simplest and most effective procedure is excision of the superolateral fragment. If the excision is performed through an incision in line with the fibres of vastus lateralis and by peeling back the insertion of this muscle to the patella rehabilitation can be rapid. Alternatively, Ogata¹³ has shown that simple detachment of vastus lateralis from the fragment can often remove the stress and allow spontaneous healing.

Stress fracture of the patella. This was first described in the English literature by Devas.¹⁴ It is commonly transverse and is seen in athletes as well as in patients with cerebral palsy who overload the patella by walking with a crouch gait.¹⁵ It is a mistake to assume that deteriorating gait in a spastic who is entering the teenage years is due simply to

an increase in weight combined with a decrease in motivation. The problem, not always obvious, may be the result of a stress fracture and avulsion of the lower pole of the patella (Fig. 4). In these cases management must include release of the hamstrings and correction of the flexed knee deformity. The distinction between a stress fracture and a bipartite patella remains difficult. It is said that the line of a stress fracture passes vertically through the patella while that of a synchondrosis of a bipartite patella runs more obliquely. Histologically, the presence of callus is proof of a genuine stress lesion, and excessive mobility of a fragment with sclerotic margins and a synovial gap would suggest established nonunion of a fracture.



Fig. 4

Radiograph of the knee of a teenage spastic diplegic with deteriorating crouch gait. There is a combination of stress fracture/separation of the lower pole of the patella, Osgood-Schlatter disease and patella alta (reproduced with permission of Harcourt Publishers Ltd).

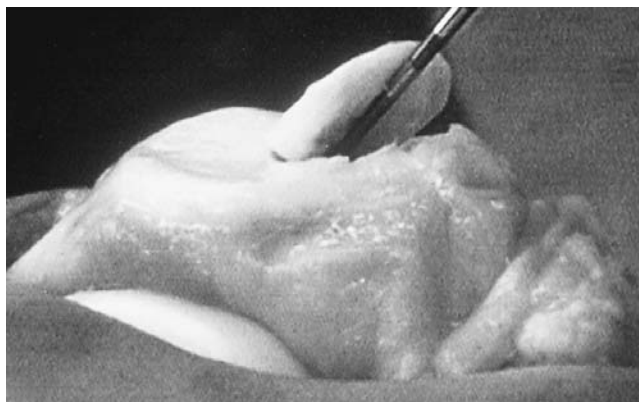


Fig. 5

A large osteochondritis dissecans lesion of the patella. Fixation failed and patellectomy was eventually required (reproduced with permission of Harcourt Publishers Ltd).

Trauma-related lesions

Osteochondritis dissecans of the patella. This is a rare cause of anterior knee pain in the young athlete. Repeated microtrauma and, particularly, shear stress to the articular surface are thought to be the cause.¹⁶ Sometimes the lesion is associated with maltracking. In addition to pain there may be obvious crepitus when the knee extends against load, and perhaps a moderate effusion. Pain inhibition may cause giving-way. If the lesion is small a painful arc may be found as it passes over the articular surface of the femur during movement.

Plain radiographs, including the 30° skyline view, will usually confirm the diagnosis although, if in doubt, more detailed images may be obtained by CT or MRI which will show whether separation has occurred. On the plain film subchondral sclerosis usually indicates loosening of the fragment within the crater. The best way to stage the lesion is at arthroscopy when the surgeon must distinguish between the following conditions:

- 1) An intact lesion with no break in the articular cartilage which on probing reveals no movement of underlying bone.
- 2) A separated lesion in which there is no break in the articular cartilage but the probe shows that beneath this intact layer the bone has separated.
- 3) A trapdoor lesion in which the articular cartilage is breached for part of the circumference of the lesion. The fragment of osteochondritis dissecans is partially detached from the articular surface.
- 4) A totally detached lesion, a loose body.

Conservative treatment with rest is appropriate for intact lesions which will usually show no sclerosis, and in patients presenting under the age of 13 or 14 years when healing is the rule. There is a good case for attempting to stabilise separated lesions, and biodegradable polylactic acid pins may be of value. Lesions which are partially or completely separated from the articular surface have been treated by excision leaving vertical edges to the crater, curettage and drilling, with the aim of healing the defect with fibrocartilage. With smaller lesions of the patella, good or excellent results have been recorded with follow-up as long as 15 or 19 years, but the larger craters still remain a problem^{17,18} (Fig. 5). Autologous implantation of cartilage may offer hope when previously patellectomy was the final solution.

Bone bruising. When seen on MRI of a post-traumatic knee, bone bruising is most often a minor feature of a more complex picture and of variable significance. It may result from direct trauma or from indirect forces which have produced patellar subluxation or dislocation. In such a case anterior knee pain may persist for three or four months as part of the post-traumatic symptoms. Very occasionally, a full-thickness fragment of overlying articular cartilage may be shed into the joint a month or two after the injury and this will be accompanied by a sudden deterioration in symptoms and an effusion in the knee.

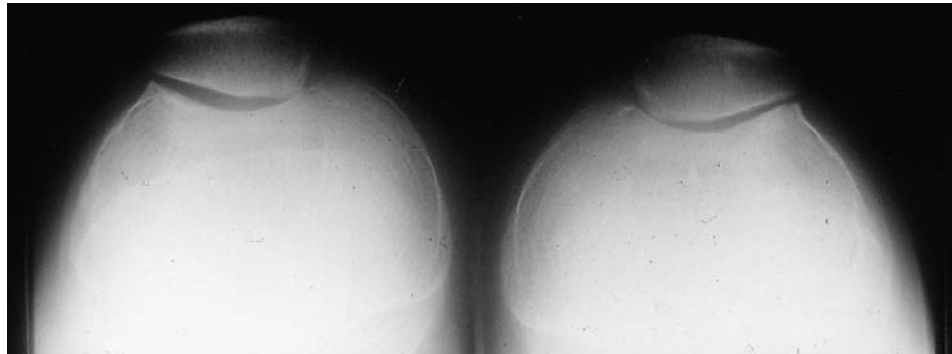


Fig. 6

Radiograph showing premature osteoarthritis in the dysmorphic knee of a patient born with the Stickler syndrome.

Post-fracture. Successful treatment of patellar fractures demands accurate reduction of the fracture, adequate fixation and restoration of a nearly normal joint surface. The state of the articular cartilage is often worse than plain radiographs suggest and the gliding surface must always be inspected by the operating surgeon. There is a tendency to try to preserve the patella at all costs, but if a reasonable joint surface cannot be reconstructed, patellectomy is preferable to the months of fruitless rehabilitation which follow a poor operation.

Syndromes and dysplasias. Dysplasia of the patella and condylar surfaces can lead to incongruity and premature degenerative change as well as maltracking and a tendency to dislocation. Anterior knee pain due to incongruity is seen in the Stickler syndrome (arthro-ophthalmoplegia) (Fig. 6). When patellar dislocation occurs in childhood there is often a positive family history, and associated dysplasia may be associated with a neuromuscular problem or a condition such as the nail-patella, Down or Marfan syndromes.

Tumours. Primary, metastatic and even metabolic brown tumours have all been reported within the patella (Fig. 7). The usual presentation is non-specific anterior knee pain but, occasionally, a lytic lesion is the site of a pathological fracture. It should not be forgotten that osteomyelitis of the patella can resemble a bone tumour. The dorsal defect of the patella is an oddity and presents as a lytic lesion at the superolateral aspect of the patella in the same region as in a type-3 bipartite patella. It may or may not be symptomatic and the surface of the joint is not involved. Very occasionally, such a lesion is so large that it may require curettage and grafting. Very rarely, soft-tissue tumours may occur within the fat pad and synovial lesions such as pigmented villonodular synovitis can cause anterior knee pain, clicking and catching.

Miscellaneous

Plicae. These are found at four sites within the knee. They are infolded shelves or partitions of synovial lining and contain a core of fibrous tissue. They are inconstant, vary in size and can be regarded as normal anatomical variants; only rarely do they cause symptoms.

The medial plica in particular has become a much over-rated focus of attention and many innocent plicae have been removed for no good reason. If there is local tenderness at the correct site, a synovial snap between 30° and 60° of flexion, a fibrotic plica and an adjacent erosion on the condyle then the diagnosis should be considered to be correct. Surgery in such cases will be rewarding.¹⁹ To incriminate the plica in the absence of these symptoms and signs is a result of ignorance. A complete suprapatellar plica which separates the suprapatellar pouch from the joint proper may occasionally be a cause of symptoms. On occasion, MRI has shown an effusion in the upper compartment which does not communicate with the joint below. The infrapatellar plica or ligamentum mucosum sometimes forms a complete curtain between the two halves of the



Fig. 7

Radiograph showing chondroblastoma of the patella.

knee. Apart from obstructing the arthroscope, this plica has little clinical relevance nor does the somewhat thicker lateral plica, which is encountered in the lateral gutter.

Hoffer's syndrome. There is no doubt that some patients have an outsized infrapatellar fat pad which bulges out on either side of the patellar tendon when the knee is extended. Arthroscopically when viewed from above, it may seem to protrude more than normal into the distended knee. There may be areas of fibrosis and inflammation in the fat pad suggesting that it has been trapped or pinched from time to time, but there is uncertainty as to whether it has ever been truly pathological.

Bursae. These are common and well recognised and should be treated conservatively in the first instance. They may contain blood or synovial fluid. The prepatellar bursa is responsible for housemaid's knee and the infrapatellar bursa for the clergyman's variety. There is a third bursa between the patellar tendon and the upper tibia which is not easily seen and symptoms from this area may be confused with those of Hoffer's syndrome.

Rupture of the posterior cruciate ligament. Chronic deficiency of the posterior cruciate ligament is associated with patellofemoral arthritis, presumably because the posterior displacement of the tibia increases the resultant force on the patellofemoral joint.

Ossification of the patellar tendon. This may be related to injury and can be a cause of continuing minor disability (Fig. 8). Matsumoto et al²⁰ reported two cases and it seems that over the years as the ossification matures, pain, stiffness and disability will increase. In their series resection of the calcified tendon and reconstruction with a Leeds-Keio ligament was recommended.

Iatrogenic

Hauser procedure. Some adverse effects may follow operations such as the Hauser procedure for recurrent dislocation of the patella, in which the tibial tubercle is transferred medially and distally. While the distal displacement should never be more than is necessary to correct patella alta, the more harmful component of this operation is the medial shift. This inevitably recesses the insertion of the patellar tendon posteriorly and runs directly contrary to the Maquet principle. An increased incidence of late-onset osteoarthritis has been attributed to this procedure.²¹

Infrapatellar contracture syndrome. This form of patella infera may follow injury or surgery and is made worse by prolonged immobilisation. The knee is stiff and painful with restricted mobility of the patella and swelling is obvious around its tendon and the fat pad. Early recognition and treatment by vigorous physiotherapy and with anti-inflammatory drugs are important, but if the condition becomes established surgery is best delayed until the inflammatory phase dies down. This may take two or three months. The surgery involves extensive medial and lateral retinacular releases with the division of intra-articular adhesions and excision of the fibrous tissue which has replaced the fat pad. Sometimes it is necessary to slide the tibial tubercle proxi-



Fig. 8

Radiograph showing ossification of the patellar tendon associated with anterior knee pain in a 20-year-old woman.

mally to re-establish the correct patellar height.²² Patella infera may follow epiphyseal leg lengthening (Fig. 9).

Post-traumatic neuroma. Neuromas may complicate scars in front of the knee just as at other sites. In particular, care must be taken not to damage the saphenous nerve when harvesting the hamstring tendons.

Obscure causes of anterior knee pain

How should one approach patients in whom the diagnosis is not obvious? The problem can be studied clinically, by imaging and at arthroscopy and it is helpful to try to place patients into one of the categories suggested in Table I.

Idiopathic knee pain. In some patients the precise cause of pain will never be established. They are often young with high expectations and place great demands on their knees. Clearly, overuse is part of the problem. On the other hand inert and overweight patients can present with similar symptoms.

Fairbank et al²³ found that clinical examination was of little help in assessing patients with idiopathic knee pain. Joint mobility, the 'Q' angle, genu valgum and anteversion of the femoral neck were not found more commonly in patients with anterior knee pain than in a control group.



Fig. 9

Radiograph showing iatrogenic patella infera after epiphyseal lengthening of the tibia.

Tight hamstrings and the “hamstrung knee”, as described by Hughston,²⁴ do seem to be real entities which will respond to physiotherapy and stretching. These patients need to be reassured as to the benign nature of their pain. Protection from overuse, a review of sports and training activities, stretches when necessary and a programme of isometric and inner-image exercises on the quadriceps are beneficial. Patellar mobilisation with the aim of stretching the lateral retinaculum, which arises largely from the ilio-tibial band, is also helpful. Knee braces and supports should be avoided.

A reasonable outcome can be expected after six months of conservative treatment. Kannus et al²⁵ reported a seven-year follow-up of patients with chronic patellofemoral pain syndrome; 75% of their patients had recovered by six months and the results were little changed when seen at seven years. Interestingly, one of their subgroups was treated with intra-articular glycosaminoglycan polysulphate with no beneficial effect. Leslie and Bentley²⁶ showed that nearly 60% of their patients with anterior knee pain and a normal articular surface at arthroscopy were asymptomatic at one year. It is therefore clear that conservative treatment for six months will solve most problems, but a small hard core of patients will remain a therapeutic challenge.

In this recalcitrant group MRI or arthroscopy will probably show some degree of chondromalacia. It is wise to check again that there is no subtle element of maltracking which is contributing to this. In the absence of maltracking the choice of surgical treatment lies between chondral shaving, curettage and drilling, resurfacing the articular surface and, ultimately, patellectomy. A reasonable outcome from

patellectomy was reported by Bentley²⁷ who found good or excellent results in 77% of 60 patellectomies performed for severe chondromalacia at a mean follow-up of seven years. **Chondromalacia.** The term describes a pathological lesion of articular cartilage which, when mild, may be reversible but when severe can lead ultimately to osteoarthritis. Chondromalacia patellae may be idiopathic or secondary to maltracking, but since it is most often seen on the medial facet of the patella with osteoarthritis on the lateral facet, it can be concluded that the one does not necessarily lead to the other.²⁸ As with osteoarthritis the symptoms do not always reflect the macroscopic appearance. Standard radiographs are often normal when the malacic changes are relatively advanced, hence the importance of MRI and arthroscopy. When inspecting the articular surfaces of the patella it is important to try to distinguish between pathological abnormality and chondral changes that are part of the normal ageing process. A fine hairy appearance of the articular cartilage, in the absence of softening or fissuring, is most likely to be due to age-related wear on the surface. Ultimately, this may lead to osteoarthritis but in its early stages it is probably asymptomatic.²⁹

True chondromalacia has a separate clinical appearance. The four stages as defined by Outerbridge²⁹ can be modified to suit the arthroscopist:

Grade 1. Closed disease:

(a) At first sight the surface looks normal, but the probe reveals a soft spongy feel or pitting oedema. The surface is intact and the softening probably reversible.

(b) A blister or raised portion of the articular surface is seen but examination with a probe shows this to be boggy. The surface is still intact.

Grade 2. Open disease. The probe will now reveal fissures which may or may not be obvious initially.

Grade 3. Severe exuberant fibrillation or ‘crabmeat’ appearance.

Grade 4. The fibrillation is full-thickness and the erosive changes extend down to bone which may be exposed. This is in effect osteoarthritis. The size of the lesion also needs to be recorded.

There is conflicting evidence as to the true pathological nature of chondromalacia. Goodfellow, Hungerford and Woods³⁰ described primary changes in the deep and intermediate layers of the articular cartilage and referred to the process as “basal degeneration”. This seems to fit well with the appearance at arthroscopy in grade-1 disease. Bentley,³¹ however, has biopsied malacic areas on the medial facet and provided evidence of surface fibrillation and loss of superficial matrix. He believes that this marks the commencement of chondromalacic change.

How does chondromalacia develop? Cartilage can be regarded as a compressed matrix. The type-II collagen fibres tightly bind together the proteoglycans which carry a very high negative charge and are strongly hydrophilic. If the collagen breaks down the unconstrained proteoglycans will inevitably draw more water into the hydrated matrix causing

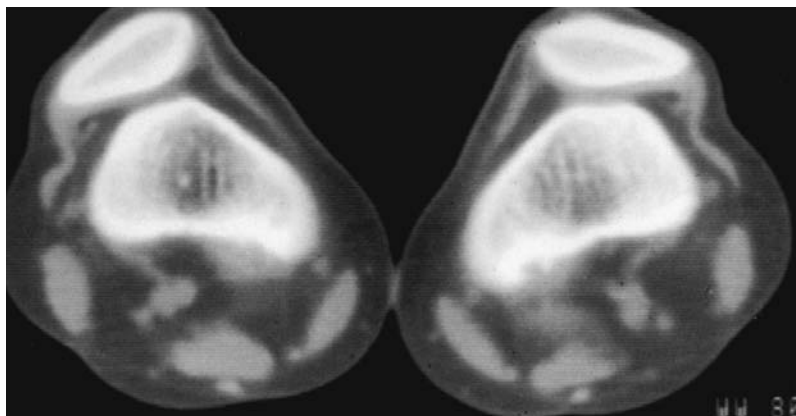


Fig. 10

Lateral subluxation of the patella in extension. The lateral retinaculum is lax and plays no part in causing the subluxation.

boggy swelling.³² Fissuring is the next step in this destructive process, followed by fragmentation and ulceration.

Maltracking of the patella. This may lead to excess overload with shear forces being applied to the articular cartilage in some areas while underloading may occur in others. Both hyperpression and hypoppression may be detrimental to nutrition of the cartilage.³³ The more extreme cases of maltracking will predispose to recurrent dislocation of the patella and this can be particularly damaging to the patellofemoral joint. The traditional tangential skyline views provide static images from which it is difficult to understand fully the dynamics of patellar tracking. Subtle disorders may be overlooked and it has been shown that as the normal knee moves from 90° flexion to full extension the patella shifts and tilts medially with respect to the tibial tubercle, with a slight clockwise rotation of the patella on the right and anticlockwise on the left.³⁴ These movements may not be clinically detectable but may be relevant. Some obvious signs such as the 'Q' angle, valgus deformity, recurvatum, triple deformity and patella alta may all be factors in producing maltracking, but when it comes to treatment it is the pattern of maltracking which matters most. This requires definition before recommending corrective surgery. It is most important to distinguish between lateral subluxation in extension and in flexion.

Lateral subluxation in extension is best demonstrated by clinical examination. The patient, sitting on the edge of the couch, extends the knee against gravity and as the patella disengages from a position of bony stability in the trochlear groove it kicks laterally. This physical sign is usually seen as the knee extends from 20° flexion up to full extension and may be associated with pain and crepitus. This pattern of maltracking is commonly associated with recurrent dislocation of the patella during childhood and adolescence but less degrees of maltracking can cause pain and a feeling of instability. Since the problem occurs in the last 20° of extension, inner-range exercises of the quadriceps which are freely prescribed for anterior knee pain are not appropriate for this group of patients. The principal cause is malalignment of the quadriceps, which produces a bowstring effect between its mean point of origin of the patellar tendon and

its insertion on to the tibial tubercle. The lateral retinaculum is not tight and surgical correction must include some type of distal realignment (Fig. 10). Most patients in this group whose symptoms are limited to pain and feelings of instability will respond to physiotherapy.^{35,36}

By contrast, maltracking in flexion is difficult to see clinically but can be defined radiologically on skyline radiographs taken between 20° and 45° of flexion. A standard technique is most important and the view of Merchant et al,^{37,38} in which the knee is flexed to 45° and the x-ray beam angled towards the foot, is probably the most popular. Fulkerson and Shea³⁹ have emphasised the importance of tilt and subluxation and have described three pathological patterns (Fig. 11). Tilt is produced by the posterior pull of the lateral retinaculum and can be recognised by drawing the line of Laurin et al⁴⁰ which connects the tips of the medial and lateral femoral condyles as a reference. The congruence angle is a method of measuring subluxation.

CT is used to study tilt/subluxation. Overlapping of images is avoided and reference points can be more precise. The knee can be studied as it approaches full extension and, the sophisticated scans described by Kawakubo, Fujikawa and Matsumoto⁴¹ have allowed the study of patellofemoral contact in the moving knee. These showed that in patients with recurrent dislocation of the patella the contact area moved laterally as the knee flexed. The contact area was smaller than in a normal control group, probably resulting in overloading. By superimposing the CT scans taken through the femoral sulcus and the tibial tubercle, the tibial tubercle/femoral sulcus distance can be measured. This is abnormal if greater than 20 mm and is then an indication for medial transfer of the tibial tubercle.

Arthroscopic assessment of patellar tracking is undertaken through an anterolateral or superolateral portal. Incremental patellar tracking can be studied at various angles between 0° and 90°. At 45° flexion and beyond, the patella should be centred and congruent in the trochlear groove even although the knee is distended.⁴² The arthroscopist can also observe overhang of the lateral facet.

Excessive lateral pressure syndrome. Ficat and Hungerford⁴³ first recognised a situation in which there was no

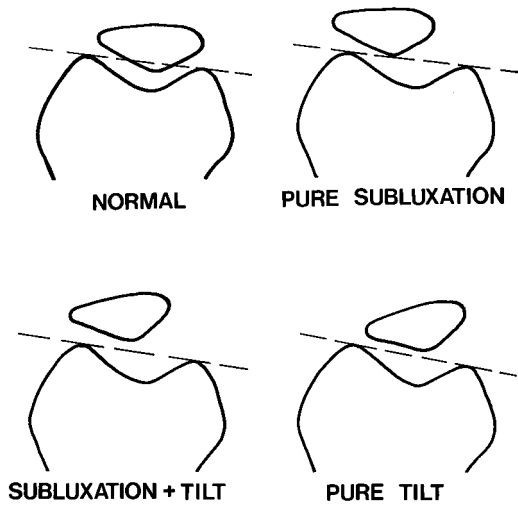


Fig. 11

Diagram showing the concept of tilt, subluxation and combined subluxation and tilt as different entities (reproduced with permission of Harcourt Publishers Ltd).

subluxation or maltracking but, nevertheless, a tight retinaculum placed the lateral patellar facet under increased load. Occasionally, the patella tilts without subluxation. This is most commonly seen when there is a predominantly “one facet patella”. The subchondral bone under the lateral facet is sclerotic while the medial third of the patella is osteoporotic. The skyline view may show lateral beaking or a marginal fracture which is taken as evidence of excessive traction on the lateral margin of the patella. At operation softening or fissuring of the articular cartilage of the lateral facet will probably be found and this is an indication for lateral release. As the joint becomes progressively more arthritic with loss of joint space, secondary subluxation and tilt will occur (Fig. 12).

Stiff painful knee/reflex sympathetic dystrophy. A typical patient in this group suffers from intractable knee pain of considerable duration and is severely disabled often walking with crutches. Time will have been lost from school or work and the patient is anxious and depressed. Admission to hospital for ‘the team’ approach is often the

best policy (Fig. 13). The knee must be fully investigated so that unlikely lesions, such as osteoid osteoma, are excluded. The usual concern is that there may be an element of reflex sympathetic dystrophy, lately renamed “complex regional pain syndrome”.⁴⁴ The patients whom we are considering are type 1 rather than type 2, which is the group associated with peripheral nerve injury.

The challenge in this group is early diagnosis and successful treatment. The diagnosis itself is not difficult once it has been considered. The patient presents with severe intractable pain which is out of proportion to the original injury. Occasionally, it may follow an operation, particularly when there has been difficulty with the immediate postoperative rehabilitation and mobilisation. In the leg the knee is commonly involved but, of course, the entire limb can be affected. Autonomic dysfunction is manifest by cyanosis or mottling, most easily seen when the patient is standing and there may be a noticeable difference in temperature between the affected area and the opposite side. In late cases there are trophic changes involving the skin as well as wasting of the quadriceps and stiffness of the joint. The inability to function and chronic pain feed the depression.

Dysfunction leads to radiological osteoporosis which may become apparent within a month of onset of symptoms. The periarticular medullary bone may have a moth-eaten appearance (Fig. 14).

A three-phase technetium bone scan can be helpful in diagnosis but less so in the younger patient.⁴⁵ The most useful diagnostic test is a trial of sympathetic block and the simplest of these is the phentolamine test. Al Duri and Aichroth,⁴⁶ in their excellent review of this difficult subject, agree with the consensus view to divide reflex sympathetic dystrophy into three stages as follows:

Stage I. 0 to 3 months; pain and hypersensitivity are dominant features.

Stage II. 3 to 6 months; diffuse radiating pain, stiffness, swelling, osteoporosis. Colour changes may appear.

Stage III. Over 6 months; the pain may begin to recede leaving osteoporosis, atrophy and stiffness.

Stiffness may be due to arthrofibrosis. Infrapatellar fibrosis may drag the patella downwards and in severe long-

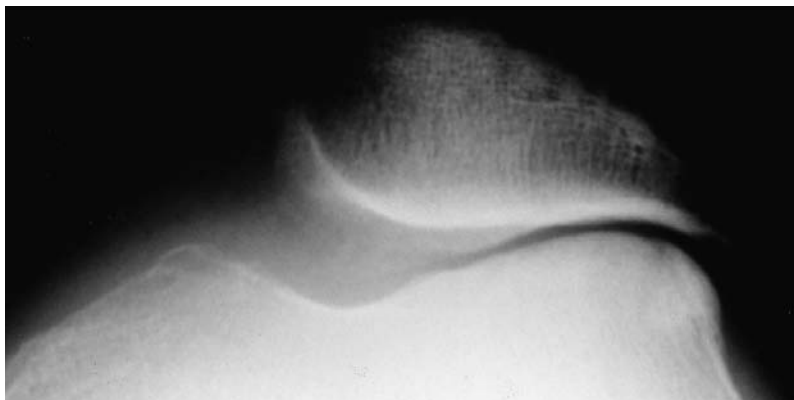


Fig. 12

Radiograph showing beaking of the lateral margin of the patella, sclerosis of the lateral facet and osteoporosis of the medial patella. It is suggested that the subluxation and tilt in this patient may be the result of loss of joint space and that the causative factor here is excessive lateral pressure syndrome.

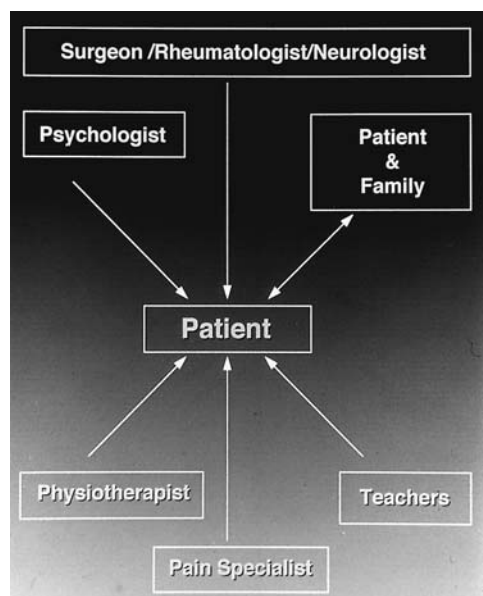


Fig. 13

Diagram showing those involved in the team approach.

standing cases there may be irreversible chondral damage. The first line of treatment includes intensive physiotherapy with a prolonged blockade, for example, epidurally with an indwelling catheter. If there is not an autonomic-mediated component to the pain the problem is likely to be psychogenic and should perhaps be regarded as a sort of orthopaedic version of 'anorexia' and equally mysterious.

The possibility of focal dystonia should not be forgotten.

Treatment of obscure knee pain

Surgery should not be performed in the absence of a demonstrable abnormality. When operation is indicated, the choice lies between the correction of maltracking if this has been demonstrated, a local procedure to repair or improve the articular surface or a biomechanical manoeuvre to reduce load on the patellofemoral joint. A combination of approaches may be appropriate. In some instances a trial of a minor operation such as lateral release may be worth pursuing before proceeding with a longer procedure, especially if the chondral changes are mild. When damage to the articular cartilage is severe and irreversible, patellectomy is the last resort.

Surgery for maltracking. Lateral subluxation of the patella in extension, if causing sufficient pain and crepitus, is an indication for distal realignment of the tubercle with or without proximal medial advancement of the patellofemoral ligament and the lower oblique fibres of vastus medialis. Lateral release alone will achieve nothing since the lateral retinaculum is not tight.⁴⁷

Pure subluxation in flexion is usually associated with mild chondral damage and the symptoms are pain and

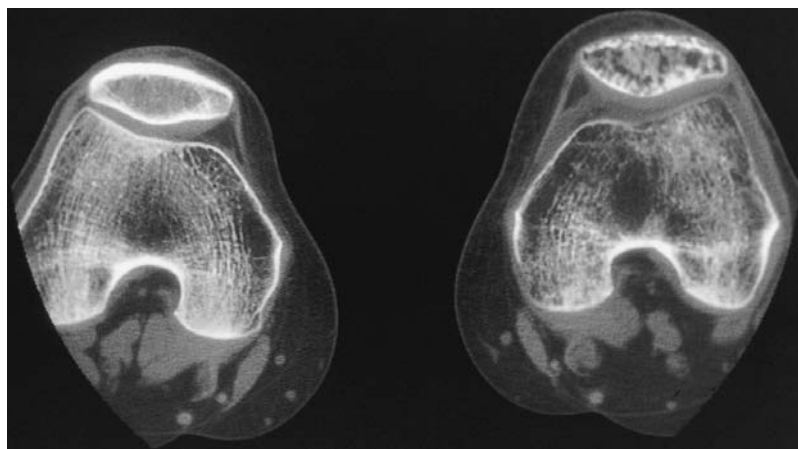


Fig. 14

The typical mottled appearance and osteoporosis seen in stage-II reflex sympathetic dystrophy.

instability. A simple lateral release often suffices but if this fails, or if chondral damage is severe, distal realignment in the form of an Elmslie-Trillat procedure or Fulkerson anteromedial transfer may be undertaken.⁴⁸ With combined subluxation and tilt the lateral facet is overloaded, the chondral damage is likely to be more severe, and the threshold for performing a distal realignment is lower. Pure tilt is probably the most sinister problem of maltracking. Movement is stable but it leads to excessive lateral pressure syndrome, retinacular pain and rapid progression to osteoarthritis. In some of these patients the process of wear can be relatively asymptomatic so that osteoarthritis of the lateral facet is severe at the time of presentation.

Open lateral release is best performed through an incision of 1 to 2 cm with the knee flexed so that the retinaculum is under tension at the moment of division. The release extends from the lower fibres of vastus lateralis down as far as the joint line. The superior lateral geniculate artery is secured and the wound infiltrated with local anaesthetic and adrenaline to diminish bleeding. Closed lateral release has become unpopular because of the high incidence of postoperative haemarthrosis, swelling and stiffness. This may change with the advent of newer electrosurgical instruments which vaporise rather than burn the tissue and which can be used in saline. The standard method is to place the arthroscope in the anteromedial portal and to use the cutting electrode in the anterolateral portal. The fibres of vastus lateralis must not be divided from the superolateral margin of the patella in case of producing iatrogenic medial subluxation. As the retinaculum is divided extravasated irrigation fluid will be seen subcutaneously in the line of the cut. If patellar shaving is contemplated it is best to divide the retinaculum first since

this improves access to the articular surface of the patella.

Chondral surgery. Attempts to improve or repair the articular surface of the patella are most appropriate when the area of damage is limited and the chondromalacia is idiopathic and not related to subluxation/tilt. Patellar shaving was first popularised by Wiles et al⁴⁷ who described an open method which has since been revisited by arthroscopists. It has not been found to be a rewarding procedure. Nevertheless, when there is severe fibrillation with effusion and small fragments of articular cartilage in the joint, it does seem reasonable to try to tidy up the articular surface. By reducing the amount of debris in the joint and the release of proteolytic enzymes, synovitis may be minimised and the onset of osteoarthritis delayed.⁴⁹

Excision of circumscribed malacic areas of chondromalacia and drilling of subchondral bone have met with some success. The defect will fill with repair fibrocartilage. This is viable but does not withstand large loads and shear forces because it lacks the properties of hyaline cartilage. In order to reinforce this fibrocartilage, carbon fibre pads have been used, with moderate success in the short term. Pongor et al⁵⁰ have reported pain relief in 67% of patients at three years and Muckle and Minns⁴⁹ describe the formation of a smooth low-friction surface with the abolition of crepitus in 70% of patients. However, Meister, Cobb and Bentley⁵¹ subsequently reported disappointing results in 27 patients in whom there was a poor outcome in 48%, with subsequent patellectomy in 30%.

Osteochondral grafting in the form of mosaicplasty can be moderately successful for defects on the femoral condyles. It has not been helpful on the patellar surface. The technique of autologous chondrocyte implantation is still under development and may improve as different scaffolds are found to carry the chondrocytes.

Biomechanical surgery. Maquet⁵² described how anterior displacement of the tibial tubercle reduces the forces acting across the patellofemoral joint. His initial operation was used in the treatment of osteoarthritis but its application soon spread to the management of anterior knee pain. It was thought that the larger the advancement the better the result and displacement of 2 to 3 cm was recommended as routine. There was a high cosmetic price to pay for improving the mechanics, since the resulting knees were ugly, and stretching of the skin over the tibial tubercle led to problems of skin necrosis and infection. Ferguson et al⁵³ subsequently pointed out that a large advancement of the tubercle reduced the vector force across the patellofemoral joint but also the area of contract stress between the patella and condyles because of rocking of the patella. A compromise had to be struck and advancement of 1.5 cm was recommended. Extreme advancement is no longer employed, but elements of the principle of Maquet are incorporated into the Fulkerson anteromedial realignment as well as the Elmslie-Trillat procedure which invariably leaves a slightly elevated tubercle.

Various tangential and sagittal patellar osteotomies have been described, but none of these operations has been received with much enthusiasm.

Ablative surgery. Patellectomy is indicated when the articular surface of the patella is severely damaged over a wide area and the rest of the knee looks normal. The repair of the quadriceps mechanism should be such as to allow rapid postoperative rehabilitation protected by a hinged knee brace. After patellectomy, patients can expect little problem in day-to-day living but sporting activities are likely to be curtailed. Even with vigorous rehabilitation some wasting of the quadriceps will remain and various authors have estimated that extensor power will never be better than 70% of normal. There is likely to be some loss of flexion and instability may become a problem if high demands are placed on the joint. The results are worse in the knee which has had multiple previous operations. The complications include rupture or persistent subluxation of the quadriceps mechanism and pain after the procedure. The Maquet procedure may be helpful in treating the latter problem.

The simplest method is enucleation of the patella through a minimal incision, otherwise known as the 'snatch patellectomy', but it may not be the best. Gunal et al⁵⁴ maintain that by advancing the oblique fibres of vastus medialis over the longitudinal repair they can overcome the problem of weakness of the quadriceps after patellectomy. Alternatively, if repair of the defect of the quadriceps seems tenuous it can be reinforced by turning down half the thickness of the rectus tendon which, incidentally, gives a better cosmetic result. When patellectomy is to be performed for an unstable patella the method of West and Soto-Hall⁵⁵ which combines realignment of the quadriceps with patellectomy can still be recommended.

The Genupath

No discussion of anterior knee pain is complete without alerting the reader to the existence of the genupath. The entire life and being of these unusual individuals centre around their knee symptoms which become chronic and are blamed for failure both at work and in their private lives.

With consummate skill and patience they can manipulate a succession of orthopaedic surgeons to perform a series of procedures, each one more drastic than the last. In some, self-mutilation may be suspected and in most, litigation is used at some stage to bankroll the lifestyle. The disruptive chain of events invariably commences as 'anterior knee pain of unknown cause' in adolescence and this progresses in stages which include patellectomy, knee replacement, arthrodesis and even above-knee amputation in just ten to 15 years. Orthopaedic surgeons must be on their guard since these patients will be deliberately misleading and they need psychiatric help, not surgery. With the exception of diagnostic arthroscopy, it is wise to make a rule never to operate on the basis of subjective symptoms alone and to adhere to it.

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