Osteochondritis dissecans (OCD) characterises a disease process predominately affecting male teenagers and young adults which, as its end-product, yields the separation of osteochondral fragments from the joint surface. Although OCD may occur in many joints, the knee appears to be by far the most commonly involved. Hence painful locking in a teenage knee should always raise the suspicion of OCD. The cause of the disease remains unclear and many theories have been developed, none of which has received unequivocal agreement. A multifactorial aetiology including elements of repetitive trauma and vascular insufficiency is henceforth considered most likely.

The fundamental principle in the treatment of OCD is the timely recognition of the condition as lesions in the early disease process are more likely to be treated successfully. Proper initial assessment and grading of the lesion will allow for the right treatment protocol to be chosen. MRI may help in the staging process and in determining the lesions healing potential. Most cases fair well with conservative measures. In more advanced stages of the disease, surgical intervention ranging from subchondral drilling to fragment refixation may be required. The primary aim of any intervention is to achieve healing of the subchondral bone, and preservation of the articular cartilage as fragment separation may pave the way for subsequent development of osteoarthritis.

Introduction

Osteochondritis dissecans or OCD, is a misnomer in terms of phrase, as it wrongly implies an inflammatory aetiology. It describes an intra-articular pathology affecting subchondral bone and surface cartilage in diarthrodial joints, which most commonly occurs in children and adolescents of male gender. It may result in the partial or total separation of the osteochondral fragment, thus creating a loose body. If untreated, this may ultimately jeopardise the integrity of the joint and lead to the development of osteoarthritis. The disease may in principle affect any joint and is known to occur in the knee, elbow, ankle and hip. The knee, however, appears to be the site of predilection, with reported figures of 65–75%.

Historical review

The first description of loose bodies in the knee joint dates back to the 17th century and is credited to Ambroise Paré,
who also appears to be the first surgeon who successfully attempted their surgical removal.\textsuperscript{1,2} Monroe, in 1726, discovered a pea size loose body originating from the lateral femoral condyle during a cadaver examination, which he believed was caused by trauma.\textsuperscript{3} Another important historical description dates back to 1817, when the French surgeon Laennec described intra-articular loose bodies as joint mice or arthrophytes.\textsuperscript{4} By expressing his belief that loose bodies arise from the proliferation of cartilage of the periarticular synovial tissue he may have inadvertently described a condition later known as synovial chondromatosis, rather than OCD. Broca, in 1854, opposed both Monroe’s and Laennec’s opinion by promoting the theory that loose bodies are caused by ‘spontaneous necrosis’ of part of the articulating cartilage.\textsuperscript{4}

Sir James Paget is credited with the classic account of OCD, which he described as ‘quiet necrosis’.\textsuperscript{5} In 1870 he reported on two children, both of whom suffered loose bodies in their knee. One, a little girl, was known to break thick pieces of wood over her knee whilst the other was an athletic school boy ‘with many blows and strains to his knee from sports’. Paget wrote: ‘How can such pieces of articular cartilage be detached from living bone? They cannot be chipped off—no force can do this. These bodies are sequestra exfoliated after necrosis of injured portions of cartilage without inflammation’. He differentiated OCD from an acute osteochondral fracture and noted that the process involved an avascular sequestrum without evidence of inflammatory change.

The phrase ‘Osteochondritis dissecans’ however, was not coined until 1887. Franz König, Professor of Surgery in Göttingen, described a series of young and otherwise healthy adults suffering loose cartilaginous fragments in their knee, ankle or elbow.\textsuperscript{6} Through the lack of trauma in all but one case and the absence of any disease he became intrigued about the aetiology of this condition, which he thought might be caused by a spontaneous process. In König’s opinion, even severe trauma was unlikely to create osteochondral fragments in the common locations observed without creating significant damage to other structures. However, he did not rule out that in a minority of patients a localised joint surface contusion might induce a process of subchondral necrosis, followed by an inflammatory response and subsequent fragment dissecation. König deliberately used the suffix ‘-itis’, as he was under the impression that these ‘corpora mobile’ were caused primarily by an inflammatory response to subchondral necrosis, which he based on the finding of giant cells in one of the retrieved specimens. König was, however, unable to explain what initiated the process of osteochondritis, and hence concluded his article by saying that the true aetiology of OCD remained unknown.

Although it is widely thought that the lesion should be more accurately termed ‘osteochondrosis’ or ‘osteochondrolysis’, the term OCD has prevailed.\textsuperscript{7,8}

It is of interest to note that some confusion arose after König had published his original account on OCD in the late 1880s, through substitution of the word dissecans with desiccans. ‘One author has brought his facts into line with the error in nomenclature by finding that the loose bodies he described were desiccated’, as quoted by Timbrell Fisher in his excellent article on the pathology and aetiology of loose bodies.\textsuperscript{9}

### Aetiology

A review of the literature reveals the lack of agreement among investigators concerning the aetiology of OCD. The most commonly quoted theories include trauma, followed by ischaemia and abnormal ossification through accessory centres of ossification within the epiphysis. There also appears to be some support for a genetic predisposition. Riedel, in 1896, at the 25th Congress of the German Surgical Society, spoke on the subject ‘Contribution to the theory of arthrophytes’ by saying ‘Foreign bodies arise in the joints not only through trauma or as a consequence of arthritis deformans, but also through König’s OCD. Until now, only the end-products of this disease in the form of completely detached bone-cartilage pieces are known’.\textsuperscript{10} In many respects little has changed since Riedel’s account some 110 years ago. Although the frequent occurrence of OCD in patients involved in sporting activities would support a repetitive trauma aetiology, on the basis of the currently available knowledge a multifactorial aetiology of OCD appears more likely.

### Trauma

Most theories on OCD aetiology centre on trauma, described either as an initial macro-trauma followed by repetitive micro-trauma or persistent repetitive micro-trauma alone. Both Paget and König are often falsely associated as being proponents of the trauma theory, which could not be further from the truth. On reading their original accounts it would appear that both men believed that trauma, when it occurs, should be considered incidental rather than causative in the aetiology of OCD.\textsuperscript{5,6}

Many authors, however, have considered trauma as the predisposing factor in the aetiology of OCD. Their theories are based on anatomical and biomechanical peculiarities of the knee joint. Fick, in 1904, demonstrated the importance of the tibial spines and the inner aspects of the femoral condyles for absorbing compressive forces and for providing guidance under varus, valgus and rotational stress,\textsuperscript{11} an observation later confirmed by Goodfellow and O’Connor.\textsuperscript{12} Owing to the central position of the eminence and its antero-posterior course, guidance is most effective in mid flexion and thus at a point where a high degree of rotational freedom is combined with a high axial load.

Ludloff, in 1908, was the first to express the view that low-grade trauma slightly above physiological levels may be causative for OCD.\textsuperscript{13} In 1922, Roesner published a landmark article with the title ‘The development mechanics of OCD of the knee’.\textsuperscript{14} Based on Adolf Fick’s biomechanical principles of knee motion, Roesner believed that the mechanism of trauma was due to the repetitive impingement of the intercondylar eminence against the lateral aspect of the medial femoral condyle (MFC) during external tibial rotation—a movement pattern known as the ‘screw-home-mechanism’. In his cadaver experiments he observed surface cartilage damage in common OCD locations after repeated leg extensions with the tibia held in fixed external rotation. He believed that through this mechanism a ‘dry fracture’ of the subchondral bone is created. He used the term ‘dry fracture’, as he thought that continuing external pressure
through ongoing impingement prevented a subchondral haematoma, leading to a non-union and subsequent fragment dissecation.

Fairbanks, in 1933, published his version of an impingement theory which, contrary to Roesner’s ‘screw home mechanism’, was based on a ‘violent rotation inwards of the tibia, driving the anterior tibial spine against the inner condyle’ 15 a notion later supported by Wilson16 and Smillie.17 Fairbanks’ theory of anterior tibial spine impingement through internal tibial rotation, however, failed to explain lesions in the most common locations of the posterolateral aspect of the medial femoral condyle.

Whilst the impingement theses may explain the development of OCD around the lateral aspect of the medial femoral condyle, they do not account for lesions occurring at other sites such as the lateral femoral condyle and patella. Furthermore, tibial eminence impingement is not seen during normal walking or running and evidence of abnormal tibial eminence morphology or abnormalities in the relationship between tibial eminence to the MFC in OCD cases is missing. Green reported on a number of cases with incomplete separation of the osteo-articular fragment, all of which presented intact surface cartilage in the area closest to the tibial eminence, henceforth questioning the validity of Fairbanks’s and Roesner’s theory of impingement.18

Other theories consider pressure exerted from the patella onto the lateral aspect of the MFC between 90° and 130° of knee flexion as a potential pathomechanism,19 Bandi based his theory on the observation that during mid-flexion the area of OCD predilection on the MFC becomes trapped between patella and tibia.20 He believed that both tibia and patella work like a pair of pliers, exerting pressure onto the femoral condyle. Through the creation of a perpendicular pressure gradient, which he described as ‘strain waves’, subchondral bone bulges forward and eventually demarcates, thus explaining the often observed chondral prominence of OCD lesions (Fig. 1).

A number of experimental studies have given support to the trauma hypothesis. Rehbein performed an experimental study on dogs in which he reproduced OCD-like lesions by exposing the anterior aspect of their knees to repetitive micro-trauma. He observed a fibrous demarcation surrounding the surface cartilage and subchondral bone, which was histologically indistinguishable from OCD lesions seen in humans.21 Langenskiold produced lesions, which histologically and radiologically resembled OCD, by cutting a segment of articular cartilage in young rabbits, leaving the cartilage attached to the synovium and replacing the fragment in its bed. He concluded that OCD might be caused by a cartilage fracture in childhood.22 Aichroth conducted a similar study using adult New Zealand White rabbits.23 He was able to show that undisplaced but stable osteochondral fractures resembled OCD in humans both radiologically and histologically. Lesions that were stabilised healed but those with tenuous stability developed into avascular unstable lesions.

Clinical and observational studies have also confirmed the association between OCD and trauma. Rosenberg examined osteochondral fractures involving the lateral femoral condyle, which had been caused by endogenous trauma.23 He demonstrated that un-united osteochondral fractures become indistinguishable from OCD both radiologically and microscopically. Kennedy et al.24 found two clinical groups in their study of osteochondral fractures of the femoral condyles. They distinguished between those patients who sustained ‘exogenous’ fractures caused by direct trauma (e.g. direct blow), to patients with ‘endogenous’ fractures caused by rotatory and compressive forces. They were able to reproduce some of these lesions in cadaver knees, although the variety of locations observed in OCD has not been reproduced experimentally. A history of previous knee trauma, usually of moderate degree, is reported in approximately 50% of all patients suffering OCD.7,18,25-29

In light of the clinical and experimental evidence it would appear that direct and indirect trauma, usually related to sports injuries, are implicated in the pathogenesis of OCD, although it is widely agreed that acute macro-trauma when it occurs appears to be incidental rather than causative.

Ischaemia

It has been suggested that the interruption of blood-flow to end arteries of the femoral condyle may precipitate ischaemic necrosis with subsequent sequestration of the subchondral bone and articular cartilage.20 Koch, in 1879, performed a series of experiments on bone necrosis and noted that subchondral bone infarction with loose body formation was the result of obstruction of the entire capillary bed of the area concerned.31 Rieger suggested that fat emboli were responsible for blocking the end arteries.32 Watson-Jones, however, believed in a systemic abnormality causing thrombosis or embolism of end arteries, which also helped to explain the large number of cases with multiple site involvement in the absence of antecedent trauma.33

The anatomical microvascularity of the distal femur was first investigated by Rogers and Gladstone in the late 1940s.34 They performed injection studies confirming a rich blood supply with numerous anastomoses in the subchondral bone. The authors concluded that ischaemia would be an unlikely factor in the aetiology of OCD. Enneking, a firm proponent of the ischaemic theory, compared the blood

Figure 1  Diagram demonstrating Bandi’s theory of compression. (Reproduced with kind permission and copyright © of the Hans Huber Verlag, Bern.20)
supply of the subchondral bone to that of the bowel mesentery with its end arterial arcades.\[sup]\textsuperscript{30}\] He found that the terminal branches within the subchondral bone poorly anastomose with their neighbours, hence infarction would result in necrosis of wedge-shaped areas immediately beneath the articular cartilage. Further support to the ischaemia hypothesis was provided by Ficat et al.\[sup]\textsuperscript{35}\] who performed marrow pressure studies on patients suffering OCD and osteonecrosis. All of their patients had abnormal haemodynamics, with increased marrow pressure and circulatory obstruction owing to stasis.

Löh\[sup]\textsuperscript{36}\] and Lang\[sup]\textsuperscript{37}\] both believed that irregularities in epiphyseal perfusion through changes from capsular to diaphyseal blood supply during epiphyseal closure are a key issue in the development of OCD. Their theory would appear plausible but fails to account for the occurrence of OCD in patients with a wide open physis.

**Accessory centres of ossification**

The development of separate ossific nuclei in the distal femoral epiphysis and their ossification process during maturation has led some researchers to conclude that OCD may simply be a variant of normal growth. Seidenstein\[sup]\textsuperscript{18}\], who considered this process to be a localised disturbance of condylar growth, observed spontaneous healing without active treatment. Ribbing\[sup]\textsuperscript{39}\] reviewed knee radiographs of 291 children for fragmentation of the ossific nucleus and compared those evidencing detached osseous islets within the articular cartilage outside the epiphysis to those with OCD. He found the locations similar in both groups and concluded that the ossification centre was a ‘locus minoris resistentiae’. He henceforth proposed the aetiology of OCD to be based on the separation of an accessory bone nucleus, which at least partially re-attaches during maturation, but which may completely separate if exposed to trauma.

**Genetic**

This notion gained some popularity through a number of studies reporting a familial incidence of OCD.\[sup]\textsuperscript{40–45}\] Petrie, however, detected only one OCD case amongst first degree relatives of 34 patients suffering OCD, indicating the rarity of a potential hereditary influence.\[sup]\textsuperscript{46}\] Multiple epiphyseal dysplasia, with its autosomal dominant and recessive hereditary patterns, should always be considered in patients with OCD. Hence, patients who are thought to have a familial form of OCD may, in fact, be suffering a variable expression of multiple epiphyseal dysplasia. Some authors have described an association of OCD with dwarfism, tibia vara and Perthes disease, indicating a possible genetic influence in isolated cases.\[sup]\textsuperscript{43,47–49}\] It would, however, appear that heredity has little if any relationship with OCD other than a possible rare familial form that may exist.

**Pathophysiology**

Precipitating insults at a vulnerable site causes a stress reaction, with locally impaired subchondral bone homoeostasis, which may progress into a stress fracture. If repetitive loading continues, the healing ability of bone may be impaired leading to avascularity and subsequent bone necrosis. Under ideal circumstances (early detection and treatment), resorption of the avascular bone is initiated by ingrowth of vascular buds and mesenchymal cells, creating a zone of granulation tissue between the viable and necrotic bone. This process of dead bone resorption and its replacement with new bone characterises the repair mechanism known as ‘creeping substitution’, which may, especially in juvenile patients, successfully re-establish the subchondral cancellous bone architecture.\[sup]\textsuperscript{14}\]

In most cases, however, the repair is inadequate. The formed callus remains un-calciﬁed, showing an exuberance of cartilaginous elements, which may shield-off the lesion preventing active repair. Expansion of this interstitial callus layer, which Roesner\[sup]\textsuperscript{14}\] described as ‘callus luxurians’, may elevate the osteo-articular fragment beyond the joint level increasing its mechanical vulnerability. This phenomenon is often observed during arthroscopy, when OCD lesions appear slightly protuberant despite intact surface cartilage. The lesion is biomechanically ill-equipped to withstand mechanical forces applied, especially if it is situated in a weight bearing area. Although the necrotic bone is held in place by the macroscopically intact overlying surface cartilage, it is assumed that this process also influences the basilar growth of the articular cartilage and its stress resilience. Once a subchondral non-union is established, the support for the articular cartilage is lost and degenerative changes are likely to occur. Under these circumstances the articular cartilage may fracture, leading to synovial fluid intrusion and inhibition of a potential healing response. Continuing hostile mechanical forces will further compromise fragment stability, eventually creating a loose osteochondral fragment.

The fragment may remain in its crater, and through continuing joint movement be eroded. Once discarded into the joint cavity, a loose body is liable to impinge between tibia and femur, leading to third-body wear. The articular cartilage of the detached osteochondral fragment usually remains viable as it receives its nutrition from the synovial

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**Figure 2** Anatomical OCD classification. (Reproduced with kind permission and copyright © of the British Editorial Society of Bone and Joint Surgery, London.)
fluid. The subchondral bone, however, will undergo necrosis and complete resorption.

Epidemiology

OCD has been classified according to the maturation status of the distal femoral physis into three groups: juvenile OCD in patients with wide open physis, adolescent OCD in patients with closing phys and adult OCD with fully closed phys. Juvenile OCD is by far the most common form observed. It has a peak prevalence between the age of 10 and 13 and is rarely seen in patients younger than 10 years of age. Boys are more commonly affected than girls, with a ratio of 5:3. Bilateral cases of OCD have been reported in up to 25% of affected individuals, with lesions usually being different in terms of size and symptoms.

Anatomic location

Aichroth developed a classification system based on a survey of 200 patients, defining the various sites of OCD within the knee joint (Fig. 2). The lesion most commonly involves the lateral posterior portion of the MFC, where it can be found in about 70% of cases. This is therefore described as the ‘classical’ OCD location. A more wide-spread involvement of trochlea and central areas of the MFC is seen in about 15% of cases and is classified as the so-called ‘extended classical’ locations. The lateral femoral condyle is affected in about 10% of cases, where lesions are commonly seen around the central weight-bearing portion, whilst lesions close to the intercondyalar notch are rather unusual. Isolated involvement of the femoral trochlea and the patella is seen in less than 5% of cases, with almost all patellar lesions being located in the infero-medial quadrant (Fig. 5).

Natural history

The natural history of OCD is dependant on a multitude of circumstances, with the degree of patient maturity being by far the most important predictive factor. Patients in the juvenile group usually have a high healing potential ranging from 65% to 75%. In principle, the older the individual the less predictable the outcome and the higher the complication rate. In adolescent patients only about 50% achieve complete resolution. In the adult group the chances of healing are low and hence the likelihood of developing premature degenerative joint disease are high. The natural history of OCD in adults has been well documented by Linden, who reviewed 40 patients with an average age of 29.4 at diagnosis. All patients were treated conservatively and followed-up for 32.5 years on average. After 20 years, 79% of patients had already developed symptoms of degenerative arthritis, which he estimated to be at least 10 years earlier when compared to the occurrence of primary knee arthritis in an otherwise normal patient group.

Other important factors influencing healing and long-term prognosis include the duration of the disorder, size of the lesion and its location. The latter factor is of particular importance as lesions close to or within the weight-bearing surface are at a higher risk of developing non-union and of detaching. Additional prognostic factors are fragment stability, the status of the articular cartilage, and the mechanical environment, especially in view of a potential deviation of the mechanical axis.

Clinical evaluation

The symptoms associated with OCD are often vague and poorly localised. Some patients, however, may be aware of more circumscribed discomfort antero-medial to the patella, owing to the location of the classic OCD lesion on the medial femoral condyle. In the latter patient group a potential differential diagnosis of a medial plica syndrome, a medial meniscal tear or patello-femoral maltracking should be borne in mind. The degree of pain described by patients suffering OCD varies greatly, but is usually dull in character. Stiffness and swelling are infrequent and not a general feature in patients with OCD. In more advanced cases, especially those where joint incongruity develops, a sensation of catching, locking or giving-way may be reported. Symptoms are usually intermittent and often related to exertion. Once the lesion detaches and a loose body forms, symptoms become more specific and, more often than not, are unrelated to activities. In these patients a knee effusion and synovitis are sometimes observed. Quadriceps atrophy and weakness may also develop and reflect the chronicity of the condition. A positive Axhausen’s sign describes local tenderness of the involved femoral condyle on direct palpation of the flexed knee. Once the osteoarticular fragment has dissecated, the loose body or the defect may be palpable. Movements may be restricted and a mild flexion deformity is often observed (Fig. 3).

When examining a child with suspected OCD it is advisable to watch the patient walk, as they may display an antalgic gait. Some patients may walk with an out-toeing gait which, according to Fairbanks and Wilson, is based on the thesis that impingement of the medial tibial eminence on the
lesion causes patients to walk with the leg externally rotated to avoid impingement. The ‘Wilson’s sign’ describes a test in which the knee is flexed to 90°, the tibia held in internal rotation and the knee slowly extended. The sign is considered positive if the patient experiences pain at around 30° of knee flexion. External rotation of the tibia immediately relieves this pain.

Investigations

Plain radiographs are usually diagnostic. An antero-posterior tunnel view in 45–60° of knee flexion will place the femoral condyles in greater profile than a standard antero-posterior view and is therefore preferred. Good quality films will reveal a well circumscribed area of subchondral bone demarcated from the surrounding femoral condyle by a crescent-shaped radiolucent line (Fig. 4). On sagittal views the lesion is classically located posterior to the midline. The affected subchondral bone may present with slightly increased density when compared to the parent, a finding that is particularly noticeable in sub-acute and chronic cases. Milgram showed that the radiodensity is owing to either secondary calcification in degenerating articular cartilage, new bone formation following revascularisation or calcification in new surface layers of cartilage and bone. Subchondral sclerosis alongside the margin of the lesion provides an indication of the chronicity of the disease process and is considered by some authors to be a sign of progressive non-union (Fig. 5).

Before the advent of magnetic resonance imaging (MRI), radio-isotope bone scans (Tc99) were considered to be a more sensitive diagnostic investigation for monitoring the clinical course of OCD than radiographs (Table 1). Although technetium bone scans are thought to provide information about the biological healing capacity of OCD, they have failed to gain widespread acceptance, most likely owing to the length of the test and the perceived risks associated with the radiotracer. Furthermore, scintigraphy has a poor predictive index, which in part is due to the persistence of nucleotide activity long after lesion resolution.

MRI is by far the most sensitive and sophisticated imaging modality allowing for early diagnosis of OCD of the knee (Fig. 4). It may also be helpful in the distinction from osteonecrosis, which often presents a more widespread involvement of the condyle, especially in the adult. Increased signal from fluid at the interface is indicative of delayed union. MRI appears to be the investigation of choice in monitoring the healing process and revascularisation of OCD lesions, by providing the added benefit of assessing the articular cartilage status (Table 2). Hefti et al. described a popular classification system for OCD based on MRI findings, which is in wide usage. Most imaging methods however, carry the disadvantage of failing to provide conclusive evidence on the subchondral fragment stability unless the osteochondral fragment has dissecated.

Arthroscopy has become an invaluable tool in the diagnosis and management of OCD in selected cases. Direct visualisation and probing of the lesion allows the surgeon to confirm the diagnosis, accurately stage the lesion, assess fragment stability and to execute treatment. Arthroscopy should, however, be used sparingly and reserved for such cases where surgical intervention is required, especially if a good quality MRI is readily available. Guhl developed a classification system based on the arthroscopic assessment of 44 patients with OCD, which has found widespread acceptance (Table 3). It distinguishes four different OCD
Conservative treatment

The treatment of OCD is dependent upon the patient’s age, the anatomical location of the lesion and the degree of involvement. Non-operative management still remains the treatment of choice for skeletally immature children, based on numerous observations of successful healing in this patient group.\textsuperscript{67-69} Most children with grade I and II lesions can be treated by activity adjustment and observation alone, but symptoms might dictate the need for a temporary regimen of reduced weight bearing.\textsuperscript{70} Impact and sporting activities, especially those with elements of twisting and turning, should be avoided. It is also important to appraise patients and parents of the 12–18 months time frame for progressive healing.

The question of whether or not to immobilise the knee has been the subject of some controversy. Prolonged immobilisation in a position evading contact between the OCD lesion and the tibia has been advocated by many authors, who believe that movement jeopardises healing of subchondral bone. Others have argued that immobilisation has a detrimental effect on cartilage nutrition and emphasised the need for passive mobilisation to promote cartilage health.\textsuperscript{17,26,71} The failure of the articular cartilage in OCD, however, appears to be secondary to the failure of the subchondral bone. It is therefore plausible to implement a period of rest, if not immobilisation, at least during the acute stages of OCD. Cylinder cast or hinged braces are applied in slight knee flexion and implemented for periods of up to 6 weeks. Braces have the advantage of allowing for passive knee flexion but should only be considered in reliable patients. The use of hinged off-loader braces appears to be an attractive treatment alternative but their efficacy has not yet been evaluated in clinical practice.

Flynn et al.\textsuperscript{72} have recommended a three-phase approach to the non-operative management of OCD. In phase I (week 0–6), the knee is immobilised and patients required to partial weight bear. Patients usually progress to phase II at around 6 weeks, when the acute pain has subsided, and radiographs provide evidence of healing. At this stage, full weight bearing without immobilisation is allowed and a physical therapy programme commenced to improve knee motion and muscle strength. A patient who remains pain-free after 12 weeks and whose radiographs continue to show healing progresses to phase III, in which low level sporting activities are introduced. Activities involving high impact and shear stresses are to be avoided until the child has been pain-free for several months and the lesion shows radiological evidence of resolution. Repeat immobilisation might have to be considered if radiographs show progressive non-union or if symptoms recurr.

Treatment compliance poses a problem in this young and active patient group, challenging the physician to emphasise to both parents and patients potential risks and long-term consequences if the protocol is not followed.

Operative treatment

Surgical management is dependent on certain characteristics of the lesion but usually reserved for patients who fail to improve with conservative measures or those with lesions of questionable stability and abnormal articular and subchondral status. The main goal is to preserve the
fragment and to maintain joint congruity. Once a lesion has reached Guhl stage II or III, surgical intervention to avoid potential non-union and subsequent detachment of the lesion should be considered. In order to promote healing and successful union between the osteochondral fragment and the subchondral bone, revascularisation of both areas has to be established. In situ drilling is recommended for stable lesions with intact articular cartilage (Fig. 6). The concept, which was first described by Wildey for the treatment of un-united fractures in two naval officers, is to create channels for vascular ingress stimulating local revascularisation. Either a trans-articular (antegrade) or a retrograde approach may be used. Retrograde drilling using an ACL-guide is technically more demanding but avoids violation of the articular surface and may be particularly useful for inaccessible areas on the posterior condyles. The procedure is performed using arthroscopic and fluoroscopic control and is aimed to create 3–5 channels by placing a smooth Kirschner wire just below the subchondral articular margin. Post-operatively, patients are encouraged to maintain a full range of motion whilst weight bearing is restricted for 4–6 weeks. Non-weight bearing, however, is to be avoided as it increases articular compressive forces through quadriceps contraction and continuing knee flexion. Results with both techniques have generally been excellent particularly in the immature patient, with healing rates of up to 90%. In skeletally mature patients, the outcome is less predictable with reported healing rates around 50%. In patients with closed, unstable or partially detached lesions (Guhl III), in situ fixation is usually the treatment of choice, with the post-operative rehabilitation regimen being essentially the same as described earlier and based on a prolonged period of limited weight bearing. A variety of bio-absorbable and non-absorbable fixation devices have been promoted which again may be used in antegrade or retrograde fashion. In order to provide rotational stability, two such devices are required, which should be placed in divergent directions. Traditionally, Kirschner wires, Herbert screws, and cannulated AO screws have been used successfully, but may require a second procedure for hardware removal. The use of cannulated screws provides better compression and superior stability than wires or bio-absorbable pins and should be considered for the fixation of very large fragments. Screw heads need to be countersunk to avoid damage to opposing surfaces unless the retrograde technique is utilised. Alternatively, bio-absorbable pins and nails made of polylactide or polyglycolide, which degrade within 6–18 months may be used. Some have surface barbs to improve fixation and to prevent the device from backing-out. Drilling of the bone and the introduction of the device is facilitated through a cannula, allowing for a truly arthroscopic application by causing minimal surface cartilage damage. Some clinical studies have provided encouraging results even in the skeletally mature patient. Despite the reported success clinicians should be aware of the potential morbidity associated with intra-articular fragment fixation, as a number of complications have been reported. Metallic devices have shown wire migration or screw head prominence, with subsequent damage to adjacent articular surfaces. Bio-absorbable pins and nails may break or loosen and through degradation of the material may create synovitis, foreign body reaction or lytic lesions. Such local reactions are primarily associated with polylactide implants with reported figures of up to 40%, and are thought to be due to the speed of material degradation. Polylactide implants, which degrade far more slowly, present local reaction in less than 5% of cases (Fig. 7).

Sclerotic margins between the opposing surfaces of subchondral bone sometimes develop in partially detached lesions. These features are well demonstrated on MRI scan or by direct visualisation during arthroscopy. Such lesions require superficial debridement using a burr or a Volkmann spoon, to remove the sclerosis and to expose bleeding bone.

Figure 6 Diagram showing the technique of ‘trans-lesional’ Pridie drilling. (Reproduced with kind permission and copyright © of the Thieme Verlag, Stuttgart, New York.)

Figure 7 Diagram showing the use of biodegradable pins in the fixation of potentially unstable OCD lesions. Introduction of pins in a cross fashion facilitates compression and rotatory stability. (Diagram reproduced with kind permission of ArthroCare Europe, Stockholm.)
before stabilisation is attempted, as otherwise healing is less likely to be achieved (Fig. 8). Application of additional bone graft is occasionally necessary. This procedure often necessitates an open exposure of the lesion, but in experienced hands an attempt can be made to perform the debridement arthroscopically.

Once the lesion has reached Guhl stage IV, arthroscopic assessment is urgently required either to retrieve the loose fragment, to prevent impingement or to attempt fragment re-fixation. The viability and suitability for re-fixation depends on the condition of the surface cartilage and the amount of remaining subchondral bone. If the cartilage appears crenellated or hypertrophic and the subchondral bone stock inadequate to facilitate secure fixation, the fragment should be discarded. The subchondral defect is then debrided of fibrous tissue to evoke a vascular response, which will recruit mesenchymal stem cells that differentiate into fibrocartilage. Alternatively, subchondral bleeding can also be facilitated with marrow stimulation techniques such as microfracture and Pridie drilling. The patient is then required to touch-toe weight-bear for 5 weeks followed by partial weight-bearing for a further 3 weeks. Some surgeons have recommended the use of CPM for 4–6 h daily during the first 4 weeks of treatment. Although this treatment approach is suitable for patients with defects of less than 10 mm in depth, fibrocartilage is less resilient to mechanical stress, especially shear, compared to hyaline cartilage and deterioration of the repair has been observed over time. If the defect is deeper than 10 mm and located in a weight-bearing area, grafting procedures may need to be considered. Osteochondral autografts (OATS, Mosaicplasty) have been used with variable success. The technique involves the harvest of cylindrical grafts of at least 10 mm in depth from a non-weight bearing aspect of the joint, usually around the trochlea, and its placement in the defect. Graft positioning with matching of surface convexity and graft stability is critical in establishing a normal load bearing surface. Potential disadvantages of this technique are donor site morbidity and graft loosening, especially in uncontained defects and those requiring multiple cylindrical grafts. Successful secondary reconstruction with osteochondral block allograft has been described in patients with significant surface defects, but no long-term results in paediatric patients are yet available.

Autologous chondrocyte implantation (ACI) combined with autologous bone grafting, the so-called ‘sandwich technique’, is currently under evaluation and has provided some encouraging preliminary results (Figs. 9 and 10). Bone graft can be obtained from the proximal tibia, therefore reducing donor site morbidity usually associated with iliac crest graft harvest. The lesion is debrided and freed of fibrous tissue until bleeding subchondral bone is exposed. Loose or damaged cartilage has to be removed and...
a firm cartilage rim established. The bone graft is then impacted into the lesion and covered with a periosteal flap or type I collagen membrane. If performed as a two-stage procedure, the patient is then rehabilitated until the bone graft has consolidated, which may take 8–16 weeks. At the second stage a further membrane is placed onto the defect, sutured against the surrounding cartilage rim and sealed with fibrin glue. A suspension of autologous cartilage cells, which are derived from cartilage harvested at least 6 weeks earlier, is then injected under the membrane.

Especially in uncontained lesions close to the intercondy- lar notch, watertight fixation of the type I collagen membrane or periosteal flap is often impossible. In this situation, matrix autologous chondrocyte implantation (MACI) represents an attractive alternative. The membrane, which is impregnated with chondrocytes, is attached to the bone graft using fibrin glue. Normal lower extremity alignment is critical for success in ACI, and the clinician should therefore consider performing a corrective osteotomy in mature patients with mechanical axis deviation. Post-operative protocol includes limited weight bearing for 6–8 weeks, avoidance of impact and shear forces, with restrictions being placed on sporting activities for up to 18 months. The patient’s commitment to the post-operative rehabilitation programme is of the utmost importance to safeguard success. Although biopsies to-date have not shown true hyaline cartilage regeneration, the durability of these transplants has been well documented in the medium term. In this difficult group of patients, 84% presented good to excellent results, at an average of 3.9 years of follow-up.

**Summary**

Although OCD has been a known entity for over a century, a definitive cause has yet to be established. The primary aims of treatment are healing of the subchondral bone and preservation of the articular cartilage, with the ultimate goal of preventing arthritis. Surgical intervention has been able to alter the natural history in the more severely affected cases. However, long-term outcome studies to reveal if such interventions will allow the knee to stand up to the various stresses imposed over a lifetime, are still awaited. Timely recognition of the disease is essential as lesions in the early disease process are more likely to be treated successfully. Proper initial assessment and grading of the lesion will allow for the right treatment protocol to be chosen. MRI may help in the staging process and in determining a lesion’s healing potential. The fundamental principle in the treatment of stable lesions is based on short-term immobilisation, the cessation of repetitive impact loading and a gradual return to normal activity. For stable lesions that fail to improve within 6 months, arthroscopic drilling should be considered. Unstable and partially detached lesions require fixation. If the fragment is unsalvageable and the lesion is small, simple debridement may achieve success through fibrocartilage ingrowth. In larger defects, however, complex reconstructive procedures may be necessary. Patients and parents need to receive realistic appraisal of the long-term prospects and the potential risks and complications, especially if treatment advice is not followed.
References

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