

# Osteochondritis Dissecans: A Historical Review and Its Treatment with Cannulated Screws

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**Summary:** The etiology of osteochondritis dissecans and the results of treating the early stages with arthroscopic fixation using cannulated screws is discussed. Arthroscopic surgery was performed on 14 patients with osteochondritis dissecans, and the osteochondral fragment was fixed with one or two screws. A second arthroscopic procedure was necessary to assess the lesion and remove the screws. Ambulation without weight bearing is allowed during the first 2 months postoperatively. Full range of motion is encouraged. The results indicate that all patients returned to their previous sport 3–11 months postsurgery. The authors conclude that fixation with cannulated screws is the ideal method of treating osteochondritis dissecans when the osteochondral fragment is still in its bed. **Key Words:** Osteochondritis dissecans—Cannulated screws—Osteochondral fragment fixation—Rehabilitation—Knee.

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Osteochondritis dissecans is a pathological process of obscure etiology in which an osteochondral fragment may partially or completely separate from the articular surface and the surrounding bone. Osteochondritis dissecans can affect any joint, but the most commonly affected is the knee. Usually the lesion affects only one joint but it may be bilateral.

Osteochondritis dissecans is the most frequent cause of “loose bodies” in the articular cavity of the knee in young people.

In the early stage of the process, the osteochondral fragment is in its bed, sometimes being asymptomatic. The diagnosis is made by radiological study when the patient is symptom free. However, when symptomatic in this stage, the principal symptom is pain.

Osteochondritis dissecans is most frequently seen in the age range of 13–21 years. Commonly the

patients do not remember any previous injury. The pain is usually diffuse, difficult to locate and define. Patients may experience locking or pseudo-locking of the joint, followed by effusion and wasting swelling of the quadriceps.

The aim of this study is to discuss the cause of osteochondritis dissecans and present the results of treating the early stages of the disease with arthroscopic fixation using cannulated screws.

## ETIOLOGY

### Literature review

Pare (1) in 1558 was the first to remove loose bodies from the interior of an articular cavity. Broca (2) in 1854 maintained that there was spontaneous necrosis with loss of fragments that were subsequently deposited on the knee.

Sir James Paget (3) in 1879 discovered the process he called “quiet necrosis” and published two cases. The first case involved a girl who was in the habit of breaking pieces of wood with her knee. The second involved a boy who participated in school sports and sustained repeated stress and trauma to his knees.

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However, it was König (4) in 1887 who gave the name osteochondritis dissecans to the entity that today is well defined radiologically and clinically, but whose cause is unclear. The most accepted causes relate to trauma, vascularity, accessory center of ossification, genetics, and heredity.

### TRAUMATIC CAUSES

#### Trauma alone

In 1738 Monro (5) found loose bodies in the articular cavity of the knee and thought them to be of traumatic origin, a theory that was shared by Reimer (6) in 1770 and Haller (6) in 1776.

Buchner and Rieger (7) in 1921 also talked of a traumatic etiology describing fractures of the lateral femoral condyle provoked by indirect traumas.

Rehbein (8) in 1950 conducted experimental studies in dogs in which he produced identical lesions and osteochondritis dissecans from a histological and radiological point of view. These lesions were provoked by repeated and small traumas on the anterior surface of the knee.

Experimental studies with rabbits to further the knowledge of the traumatic etiology of osteochondritis dissecans were conducted in 1955 by Langenskiöld (9) and in 1962 by Tallquist (10). Later, other investigators who also advocated this etiology were Nagura (11), Green (12), and Banks (13).

#### Trauma-induced inflammatory response

Rainey (6) between 1848 and 1850 published his theory that loose bodies were fragments of tissue that had broken away and were fed by absorption of the synovial fluid.

Kragelund (14) in 1887 said that traumas in the osteochondral area produced a separation of the fragments by a chronic inflammatory process.

In 1920–1921 Timbrell-Fisher (15) also observed that traumas in the epiphysis produced a certain degree of inflammation in the lower vitality surface that could be exfoliated gradually.

Sommer (16) in 1923 and later Mouchet et al. (17) in 1925 postulated that, after the traumas, there were other pathological processes such as “paralysis of local vessels.”

In 1937 Conway (18) suggested that the etiology of osteochondritis dissecans was not exclusively traumatic.

#### Trauma causing tangential and rotational forces

Kappis (19) in 1920 indicated that tangential and rotational forces could act on the convex surface of

the condyle and crack the distal articular portions totally or partially. He explained that the origin of a trauma, in the absence of large lesions, was due to predisposed factors and the lack of innovation of the cartilage.

Schmidt (20) in 1924 demonstrated through cadaveric studies that the cartilage of the femoral condyles were lesioned more easily with tangential forces than with vertical forces.

In 1928 Wolbach et al. (21) explained that osteochondritis dissecans was the effect of mechanical pressures on a portion of the articular cartilage that had cysts on the subchondral bone.

O'Donoghue (22) in 1966 reported on three different types of trauma that could produce osteochondral fractures: compaction, shearing, and avulsion. Also in 1966, Kennedy et al. (23) conducted experimental studies in which they attempted to reproduce osteochondritis dissecans in cadaveric specimens by subjecting them to direct traumas and rotational and endogenous compression forces, but they were unsuccessful.

#### Trauma of epiphysis

Barth (24) in 1898 believed that osteochondritis dissecans was due to intraarticular fractures.

#### Trauma due to patella impingement

Hellstrom (25) in 1922 reported that the patella damaged the medial femoral condyle when the knee was fully extended.

#### Trauma producing micro fractures

Phemister (26) in 1924 thought that osteochondritis dissecans in all cases was caused by fractures produced by micro traumas.

Aichroth (27) in 1970 concluded from studies of laboratory animals that possible causes of osteochondritis dissecans were osteochondral fractures that remained unattached. In 1971, Aichroth (28) reported that 46% of his patients with osteochondritis dissecans had previously sustained a significant trauma. These results were also obtained by Scott and Stevenson (29) in 1971, Lindholm (30) in 1974, Linden (31) in 1977, Carrol and Mubarak (32) in 1977, and Zeman and Nielson (33) in 1978, although they did have different percentages.

Milgram (34) in 1978 studied the possible traumatic etiology of osteochondritis dissecans and concluded that it was due to osteochondral fractures of the articular surface. The same year Matthewson et al. (35) described different types of frac-

tures of the lateral femoral condyle provoked by indirect traumas.

#### Trauma by medial tibial spine

The radiographer Richards (36) in 1928 reviewed a series of radiographs and found osteochondritis dissecans on knees with large medial tibial spines.

In 1933 Fairbank (37) suggested that the lesion was due to a violent internal rotation of the tibia steering the tibial spine against its own condyle.

In 1967 Wilson (38) advocated a traumatic etiology and described the sign that carries his name. Wilson's sign is detected by extending the knee until it reaches a flexion of 30°. If there is an internal rotation of the tibia, the patient feels a great deal of pain. This pain disappears if the tibia is rotated externally. This phenomenon occurs because the tibial spine hits the damaged surface of the lateral femoral condyle.

### VASCULAR

#### Vascular alone

In 1870 Paget (3) thought that loose bodies were exfoliated sequestrum after necrosis of the lesioned cartilage portions without acute inflammation and injury.

In 1912 Axhausen (39) defended the theory that a trauma from the articular surface would partially damage the vessels with or without partial fracture according to the degree of trauma. This would indicate necrosis of the irrigated bone as a result of the damaged vessels, gradual separation, and formation of loose bodies. Buchner and Rieger (7) in 1921 also defended the vascular etiology.

Ficat et al. (40) in 1975 studied cases of osteochondritis dissecans and osteonecrosis and observed hemodynamic processes in both.

Enneking (41) in 1977 was one of the biggest defenders of the ischemic theory, concluding that the bone necrosis was caused by loss of nutrition.

#### Middle geniculate artery

Ludloff (42) in 1908 defended the theory that the loose bodies originating in the lateral side of the medial femoral condyle were caused by trauma of the medial geniculate artery when it perforated the posterior capsule of the joint. This articular disorder indicated that bone necrosis was occurring and that the necrotic bone was separating gradually because of insufficient nutrition.

#### Vascular spasm

In 1854 Broca (2) reported that a process of drying sequestrum was the cause of loose bodies.

In 1864 Klein (43) advocated a spontaneous demarcation of a part of the intercondylar notch of the femur.

In 1885 Poulet and Vaillard (44) defended the theory that the osteochondral loose bodies could be seen after spontaneous necrosis.

#### Blood supply of the posterior cruciate ligament

Freiberg (45) in 1923 performed laboratory studies on the various degrees of impingement of the posterior cruciate ligament vessels against the tibial spine when the knee was flexed. Logically, the impingement was greater when the tibial spine was longer.

#### Hemarthrosis

In 1759 Hunter (46) theorized that the formation of intraarticular loose bodies was due to hemarthrosis.

#### Thromboembolism

In 1879 Koch (47) performed experiments on embolic necrosis and concluded that loose bodies were caused by the obstruction of the nutritious capillaries of the bone. This theory was shared in 1920 by Rieger (48), who thought that the lipid embolus was the cause of vascular blockage.

Watson-Jones (49) in 1952 suggested that systematic abnormality was the cause of the thrombosis or embolus of the arterial system.

### ACCESSORY CENTERS OF OSSIFICATION

Some investigators asserted that osteochondritis dissecans was caused by a variation of the normal growth in young people. In 1941 Sontag and Pyle (50) were the first to note partial or total loss of regularity of the epiphysis in childhood.

In 1958 Caffey et al. (32) demonstrated the great quantity of irregular ossifications in the femoral epiphysis of healthy children, not knowing this could produce erroneous diagnoses. After studying the knees of 147 healthy children, they classified the epiphyseal disorders into three groups: (a) those that had slightly altered edges and occasionally had small centers of ossification behind the principal edge; (b) those that had the largest localized irregularities in the form of indentations; and (c) those that had irregularities similar to those of the previous group and also had an osseous block in the marginal crater.

The same investigator observed that in the knees studied 66% of the men and 41% of the women had irregularities in the centers of ossification.

The explanation given by Sontag and Pyle (50) for these irregularities in the femoral epiphysis was the rapid growth of the cartilage proliferation nucleus and calcifications in this zone, which are deeper than those of slow growth. When the growth is extremely rapid, such as in the distal femoral epiphysis, the ordering process of the cartilage proliferation and provisional calcification can be altered (32).

Ribbing (51) in 1955 proposed that the cause of osteochondritis dissecans was the presence of the accessory bone nucleus, which separates and subsequently partially rejoins the adjacent bone.

Sontag and Pyle (50) in 1941 and Ribbing (51) in 1955 postulated that the irregularities in stage III were the precursors of osteochondritis dissecans.

In 1975, after studying six cases of osteochondritis dissecans, Chiroff et al. (52) proposed that the classic dissecans lesion was always a repairing process when the fragment remained "in situ."

#### GENETIC AND HEREDITARY CAUSES

There are numerous published studies that mention the familial incidence of osteochondritis dissecans, including those of Bernstein (53) in 1925, Wagoner et al. (54) in 1931, Novotny (55) in 1952, Pick (56) in 1955, Tobin (6) in 1957, and Smith (57) in 1960.

#### OTHER CAUSES

Axhausen (58) in 1922 stated that the vascular blockage was caused by tuberculosis bacteria.

In 1926, Knaggs (59) thought that the first lesion was a periostitis caused by a minor infection, probably as a result of *Staphylococcus* organism inoculation.

#### MATERIALS AND METHODS

The study was conducted from February 1988 to March 1991. Thirty-one patients who had osteochondritis dissecans in one or both knees underwent operative treatment. Arthroscopic fixation with cannulated screws was not performed on 17 of these patients because the osteochondral lesion was unattached. The remaining 14 patients composed the group studied, in which (a) the osteochondritis dissecans was not in the stage of loose bodies, that is, the osteochondral fragment was in its bed; and

(b) the cartilage of the lesion was sufficiently healthy to support the stress of fixation (Fig. 1).

There were 13 male patients and one female patient in the study group. The age range was 13–37 years. Eight patients had closure of their physis. All patients participated in sports. The study involved 13 right knees and two left knees. Both knees were affected in one patient.

All 14 patients experienced pain, effusion, and decreased range of motion of the affected knees for a period of 3 months to 1 year. All 15 knees showed osteochondritis dissecans on radiological studies. In 14 knees, the lesion was located in the medial side of the intercondylar notch. In one knee, it was located in the femoral trochlea (Fig. 2).

In 12 knees, the fragment was attached with two cannulated screws. In three knees, just one screw was used (Fig. 3). In one knee, the screws were inserted with washers because the size of the fragment was quite large (Fig. 4).

#### Arthroscopic technique

After the preoperative examination, arthroscopy was performed to determine the stage of the disease and whether there was associated pathology, and to determine the appropriate surgical technique. Fixation with cannulated screws is only used in stage I or II when the osteochondral fragment is still attached.

The arthroscopic procedure is performed without a tourniquet. Svedocain (0.5%) 2–3 cm<sup>3</sup> is injected before making the portals, which are located inferomedially, inferolaterally, superomedially, and at the optimum site of the anterior side of the knee (to

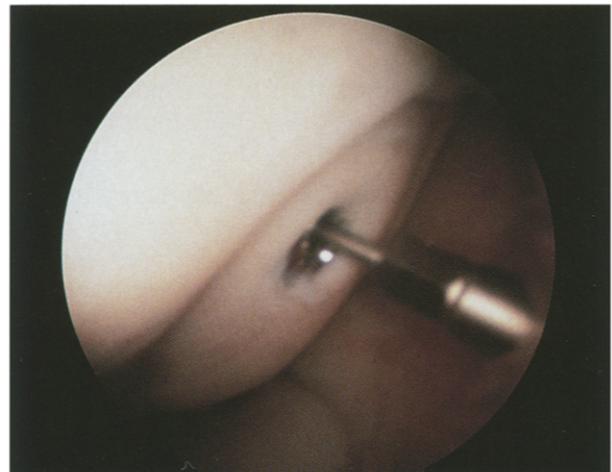


FIG. 1. Arthroscopic view of the case showing the criteria to carry out this technique.

2A,B

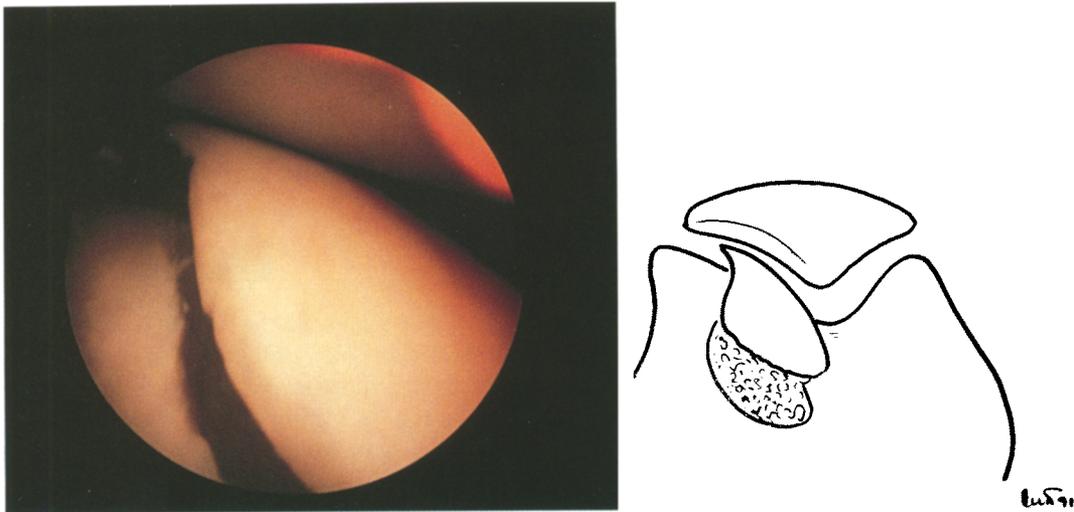


FIG. 2. A: Arthroscopic view of osteochondritis dissecans affecting the femoral trachlea. B: A diagram of this case.

approach the lesion perpendicularly). The inferomedial portal is made as central as possible and is 8–10 mm proximal to the superior side of the medial meniscus.

The knee is flexed at 70–90° depending on the location of the lesion because the screw must be inserted perpendicular to the damaged cartilage area (Fig. 5). The telescope and instruments are inserted through the portal giving the best view and working position. The inflow system to the joint is inserted through the superomedial portal using a 4.5- to 5-mm auxiliary sheath. A probe defines the edges of the lesion and establishes the quality of the cartilage. In young people with good cartilage, localization of the lesion is difficult. In these cases, it is more convenient to use radiographs. To facilitate access, the fat pad or synovium around the additional portal is removed with a pituitary grasper.

With a power drill, a Kirschner pin perpendicular to the lesion is inserted. Using a cannulated drill, the hole is made for the screw. The drill is with-

drawn, but the pin is left intact because it is used to guide the screw. It is necessary to ensure that the screw is sufficiently tightened to attach the fragment. The head of the screw should always be in line with the articular surface and should never be raised above it.

This procedure is the same whether one or two screws are used. However, when there is only one screw, it is placed centrally. When there are two screws, they are placed symmetrically. One screw is used when the lesion is small, and two screws are used when the lesion is more extensive.

When the fragment is fixed, the damaged area is

3A,B

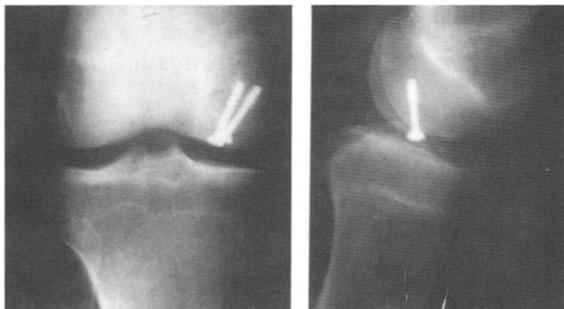


FIG. 3. A: Case fixed with two screws. B: Case fixed with one screw.

4A,B

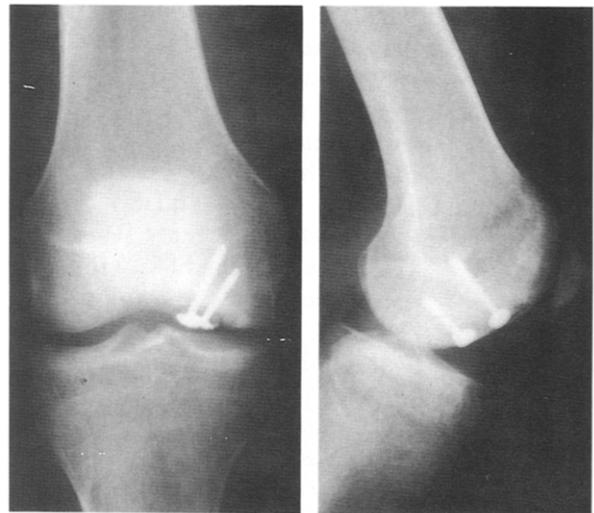


FIG. 4. A: Radiological image showing an anteroposterior view of the case fixed with two screws and two washers. B: Radiological lateral view of the same case.

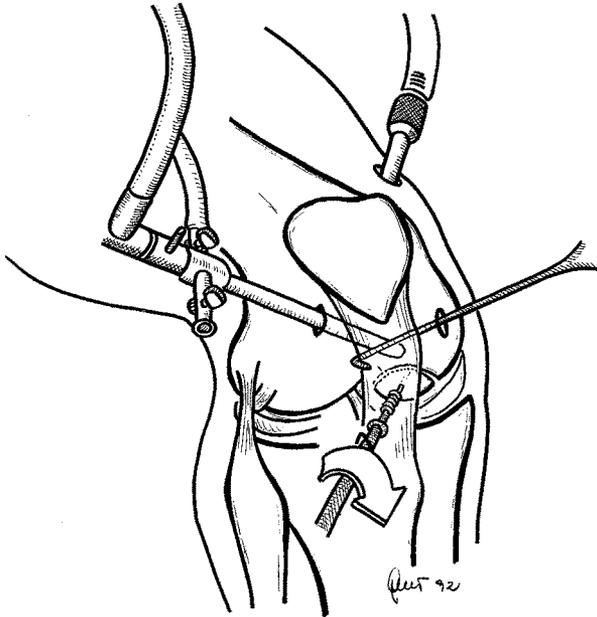


FIG. 5. Knee flexed at 70–90° with the screw being inserted perpendicular to the damaged cartilage area.

perforated with the Kirschner pin in a power drill. By doing this, the sclerotic barrier is penetrated and bleeding occurs from the adjacent bone tissue. This procedure is considered fundamental to the recovery of the patient.

Before removing the telescope, all the detritus is eliminated by washing the joint. The portals are sutured with a monofilament, nonabsorbable thread. The knee is dressed with an elastic bandage.

#### Postoperative procedure

Bed rest with the lower limbs extended is recommended for the first stage of the postoperative period. It is necessary to empty the hemarthrosis before initiating movement of the knee.

Walking is allowed without weight bearing 24 h after surgery. After 48 h, patients begin flexion–extension exercises. At 1 week, quadriceps and hamstring muscle strengthening exercises are begun. At 6–8 weeks, patients can walk with partial support. In this period, cycling and swimming are allowed. At 10–12 weeks, if there is no pain patients can increase weight bearing and no longer use crutches. Full range of motion is obtained in the first or second week.

When patients are able to walk without pain and radiological studies show that the lesion has responded to treatment (Fig. 6), a second arthroscopy is performed to reconfirm the consistency of the cartilage and the osteochondral fragment.

If the arthroscopic examination shows that the fragment is healthy, that is, the color and consistency are the same as the rest of the cartilage, the screws are removed. Bleeding is commonly observed after screw removal. A second examination is undertaken to confirm that the healing is complete (Fig. 7).

#### RESULTS

All the knees in the study were examined both subjectively with the Lysholm test (58) and objectively with radiography. The results were excellent or good in 93.33% of cases. Twelve were excellent, two were good, none were fair, and one was poor (Table 1).

Clinical symptoms disappeared during the postoperative period. The radiological images showed a progressive recovery of the lesion. At 43 months of follow-up, a faint image remained even though clinical symptoms had disappeared.

Results obtained from the second arthroscopy indicate that 14 knees at 3–6 months showed complete attachment of the fragment, allowing removal of the screws and completion of the rehabilitation program. One knee at 6 months showed an incomplete attachment of the fragment. It was necessary to tighten the screws and not remove them until 11 months, when a normal rehabilitation program was then followed.

Twelve patients returned to their previous level of sports participation, whereas two patients returned to a lower level of activity. There were no patients who returned only to a recreational level of sports activity.

#### DISCUSSION

Complications were observed in seven cases, but were only significant in three cases. In one case,

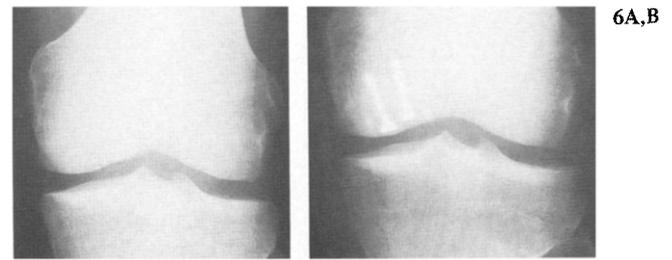


FIG. 6. A: Radiological anteroposterior view of a patient affected by osteochondritis dissecans before fixing the lesion with two cannulated screws. B: Another radiograph of the same patient 7 months later, just before removing the screws.

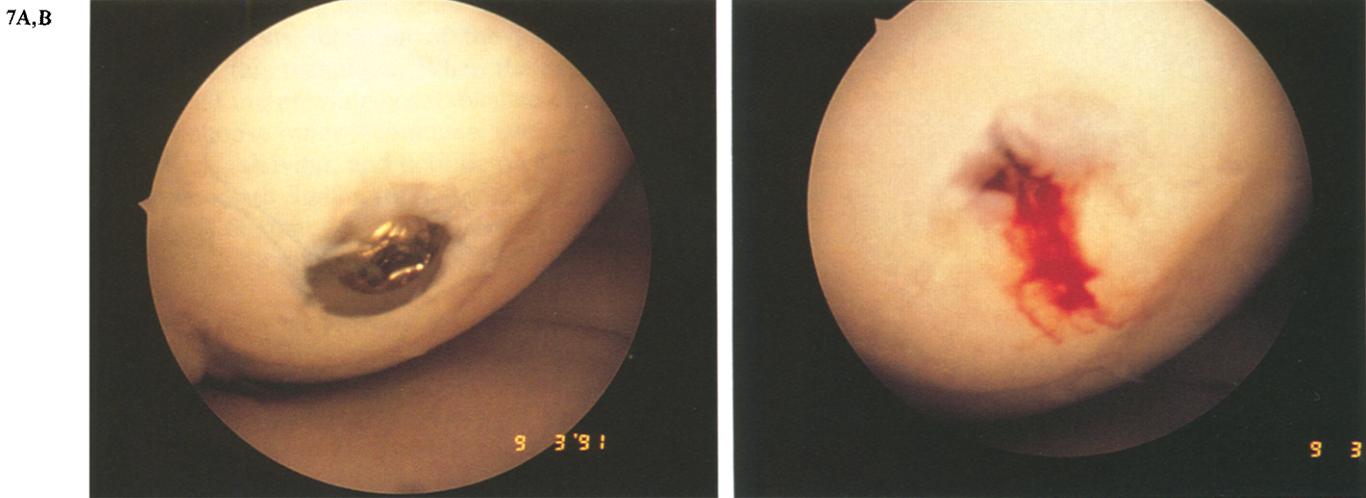


FIG. 7. A: Arthroscopic view of a patient reviewed in March 1991 to remove the screws. B: The bone bleeding on removing the screws.

radiographic control was fine at 6 months. However, at the second arthroscopy one screw was loosened. The screw was tightened and not removed until 11 months. However, this only delayed the recovery, and the result was satisfactory. The patient was satisfied and returned to a sport at the preinjury level (Fig. 8).

In the second case, in which the lesion was located in the femoral trochlea, a chondral lesion was found in the lateral articular surface of the patella on performing the second arthroscopy. This was due to friction between the articular surface and the head of the screw. Thus, it is important to place the screw heads at the same level as the articular cartilage surface and not above it (Fig. 9).

The last case involved a 17-year-old male football player. The second arthroscopy was performed in March 1990 to remove the screws (Fig. 10A and B). He returned to play football at the same level 2 months later. Sixteen months later, during a 24-h football tournament he sustained a knee injury (Fig. 10C). After clinical examination and radiology, an osteochondral loose body lying in the suprapatellar region was diagnosed. Further arthroscopy con-

cluded that the surgical procedure to fix the fragment had not been performed correctly. The loose body was removed, and the affected area was perforated. The patient recovered completely and returned to football at his preinjury activity level.

Minor complications were observed in four knees. In three cases, the Kirschner pin bent while drilling. It was removed and replaced. When the fat pad is extensive, we recommend removing the maximum amount possible to prevent loss of screws. In one case, a screw was lost inside the fat pad and radiographs were used to locate the screw.

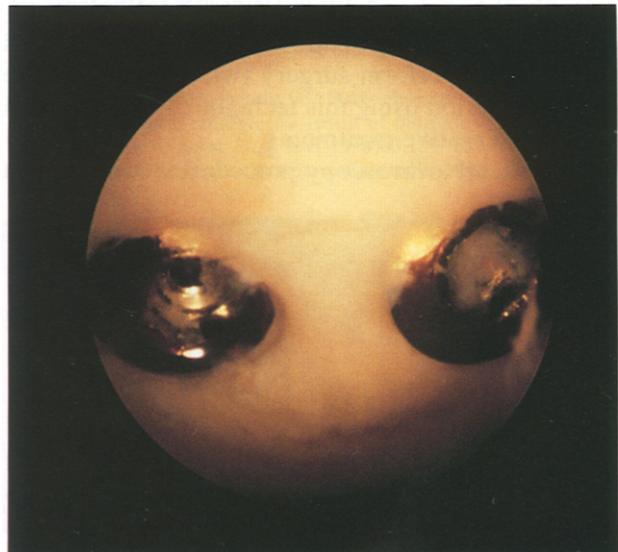


FIG. 8. Detail of the location of the screws in the first arthroscopy.

TABLE 1. Subjective results of the group of patients treated by fixation of the osteochondral fragment with cannulated screws at 1 year

Result	n
Excellent	12
Good	2
Fair	0
Poor	1

9A,B

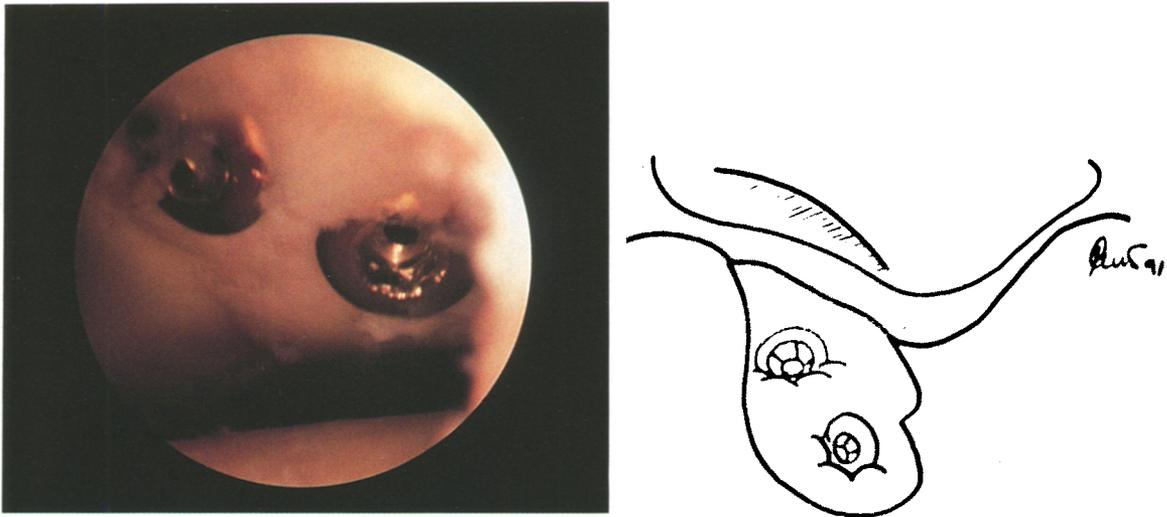


FIG. 9. A: Arthroscopic view of the patient with osteochondritis dissecans affecting the femoral trochlea when the screws were going to be removed. B: A diagram of this case showing the lesion provoked by the head of the screws.

There were no cases of infection, thrombophlebitis, thromboembolism, or ruptures of the osteochondral fragment during the surgical procedure.

The discrepancy between the remaining faint radiological images and the symptomatology and arthroscopic results occurred in each case. However, because all patients (except one who sustained another injury) were asymptomatic and there was complete attachment of the fragment, we were satisfied with the results.

We advised all patients to have an annual clinical and radiological examination. Both of these confirmed satisfactory results.

A disadvantage of this technique is that it is difficult to perform by arthroscopy. In addition, it is necessary to perform surgery twice. However, the recovery period using this technique is shorter than with conservative treatments.

As with all arthroscopy procedures, it requires a

short hospital stay and is less aggressive than conventional surgery. The procedure assures a precise fixation and compression of the fragment to the adjacent bone.

This procedure is very precise and delicate. Sudden movements and excessive forces while fixing or extracting screws can break the osteochondral fragment or the screws, as documented by Merchan et al. (60).

### CONCLUSION

During management of osteochondritis dissecans, it is important to remember that the radiological images may not coincide with those of arthroscopy or with the clinical symptoms. Thus, radiology alone is not sufficient to evaluate recovery of the lesion; clinical and arthroscopic examination are also required.

10A-C



FIG. 10. A: Radiological lateral view just after surgery, showing the fixation of the fragment with two cannulated screws. B: Radiological lateral view of the same case in May 1991 after the second arthroscopy, when the patient was able to return to sports. C: Radiological lateral view of the same case in July 1991 after the injury, showing a loose body in the subquadriceps space.

We think that the good results obtained were due to the meticulous selection of the patients to be treated with this technique. When the patient does not fulfill all the previously established criteria, we prefer to choose other techniques, such as multiple perforations, shaving, and debridement.

We agree with Smillie (61–63), Johnson (64), and Johnson (65,66) that walking without weight bearing is essential for a minimum period of 2 months post-operatively.

The minimum age to treat the osteochondritis dissecans by surgery is 13 years. Patients younger than this have been treated by conservative procedures, consisting of walking without weight bearing, but with full range of motion of the knee. All patients recovered within 1 year.

Of the patients studied, the younger patients had the most rapid recovery (3–5 months). The group as a whole had a recovery period of 3–11 months (average 6). All patients returned to their habitual sport without symptoms.

We conclude that fixation with cannulated screws is the ideal method to treat osteochondritis dissecans when the osteochondral fragment is still in its bed.

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