Osteochondritis Dissecans

History, Pathophysiology and Current Treatment Concepts

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Osteochondritis dissecans, a lesion of bone and articular cartilage which alters the smooth integration of motion and force transmission in a joint, has been the subject of much research.[60,70,75,93,97] However, the etiology remains unclear, and treatment still provokes controversy in the orthopedic community.

Osteochondritis dissecans occurs in the knee joint in two groups of patients: (1) in young patients as a result of trauma and irregular ossification centers;[68,70,75] treatment can often be expected to produce good results;[31,70] (2) in adults as a result of vascular phenomenon; treatment is more aggressive and the results are less satisfactory.[21,48,70]

HISTORY

Ambrose Pare was credited with removing loose bodies from joints as early as 1558.[60] The origin was described as "quiet necrosis" by Paget in 1870.[63] The term "osteochondritis dissecans" was coined in 1888 by Konig,[41] based on his hypothesis that these "corpora mobile" were caused by spontaneous necrosis resulting from trauma. Since then, multiple etiologies have been proposed.[4,14,18,21,60,65,70,77]

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Received: October 2, 1981.

ETIOLOGY

A review of the literature underlines the lack of agreement among investigators concerning the etiology of osteochondritis dissecans. The most accepted theories include trauma,[2,10,18,22,27,41,52,59,63] ischemia,[4,14,21,31,69,90] abnormal ossification within the epiphysis,[12,44,68] or a combination of these.[44] A few series have shown a genetic predisposition.[7,78,89]

TRAUMA

Trauma was suggested as a cause quite early by both Paget[63] and Konig.[41] In 1933, Fairbanks[22] first implicated indirect trauma, suggesting impingement of the tibial spine on the lateral aspect of the medial femoral condyle during internal rotation of the tibia, a theory later advocated by Smillie.[77] Makin[56] reported osteochondral fractures of the lateral femoral condyle produced by patellar dislocation and emphasized that loose bodies caused by this mechanism must be differentiated from osteochondritis dissecans. Rosenberg[72] reviewed 15 cases of endogenous, *i.e.*, not caused by direct external trauma, osteochondral fractures involving the lateral femoral condyle. In one case, he demonstrated a roentgenographic correlation between nonunion of an undisplaced osteochondral fracture and osteochondritis dissecans. He concluded that osteochondral fractures that do not unite become roentgenographically and microscopically indis-

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tinguishable from osteochondritis dissecans. Green,[30] however, documented several cases of incomplete separation of the fragment, noting intact articular cartilage on the side of the fragment *closest* to the tibial spine. This finding suggests that the tibial spine may *not* play a role in osteochondritis dissecans.

Direct trauma has also been advocated. The medial articular facet of the patella has been shown to contact the classic site of osteochondritis dissecans when the knee is fully flexed.[1] Rehbein,[67] in an experimental study in dogs, produced lesions resembling osteochondritis dissecans histologically and radiographically by repeated minor trauma to the anterior aspect of the knee. A fibrous demarcation in the cartilage was produced, but no loose bodies. There has been no follow-up study, however, to confirm or refute these findings.

O'Donoghue,[62] in a detailed discussion of chondral and osteochondral fractures, pointed out that three types of trauma can cause osteochondral fractures: (1) *compaction*, a direct force applied vertical to the joint surface; (2) *shearing*, a tangential blow to the joint surface; and (3) *avulsion*, a separation of a cartilage fracture with a shell of bone.

Matthewson and Dandy[58] also studied fractures of the lateral femoral condyle produced by indirect violence. Similarly, Kennedy *et al.*[39] found two clinical groups in their study of osteochondral fractures of the femoral condyles: patients who had "exogenous" fractures from direct trauma, and those who had "endogenous" fractures from rotatory and compression forces. They produced these lesions in cadaver knees, although the variety of locations observed with osteochondritis dissecans has not been reproduced experimentally. Buchner and Rieger[11] used mathematical models and force estimates to discount direct trauma as an etiologic mechanism. They found that the force necessary to produce the fracture in cadavers was greater than the force estimated to occur clinically.

Langenskiold[43] produced lesions which radiographically and histologically resembled osteochondritis dissecans by cutting a segment of articular cartilage in four to seven-day-old rabbits, leaving the cartilage attached to synovium in the intercondylar notch and then replacing the fragment in its bed. This method was confirmed by Tallqvist.[84]

About 40% of the patients who have osteochondritis dissecans give a history of prior knee trauma, usually of a minimal to moderate degree.[2,15,30,47,52,74,97] Aichroth[2] elicited a history of significant knee trauma in 46 of 100 patients who had osteochondritis dissecans. Twenty-three of 40 patients in Green's[30] series had some history of injury to the knee, while Linden[48] reported 50 of 95 joints involved with osteochondritis dissecans to have a history of injury (usually minor). Twenty-nine of 81 patients presented by Scott and Stevenson[74] gave a history of knee injury when evaluated for osteochondritis dissecans. Zeman and Nielsen[97] reported a history of trauma in seven of nine patients. Lindholm[52] noted 47 of 108 patients who had some history of trauma prior to diagnosis of osteochondritis dissecans. However, Carroll and Mubarak[15] reviewed 75 patients who had osteochondritis dissecans and found no relationship with trauma, patellar dislocation, or tall tibial spines.

The unsettled nature for the role of trauma in osteochondritis dissecans was reflected in an excellent review article by Nagura.[60] In light of the accumulated clinical and experimental evidence, direct and indirect trauma are apparently involved in producing the lesions of osteochondritis dissecans.

Ischemia

The obstruction of end arteries to the femoral condyle at the site of involvement has been suggested to precipitate cartilage

and bone separation.[21] Axhausen[4] suggested that tubercle bacilli blocked the end arteries, while Rieger[69] implicated that the blockage was caused by fat emboli. Watson-Jones[90] suggested a systemic abnormality as causing thrombosis or embolism of the end-arteries to explain the large number of cases with multiple site involvement and no history of antecedent trauma.

Enneking[21] has been one of the major recent proponents of the ischemic theory. He compared the blood supply of the subchondral bone to that of the bowel mesentery with its end arterial arcade and found that that terminal branches anastomose poorly with their neighbors. Therefore, infarction results in necrosis of wedge-shaped bone pieces immediately beneath the articular cartilage. Resorption of the necrotic bone is initiated by ingrowth of vascular buds and mesenchymal cells. This produces a zone of granulation tissue between the viable bone and the necrotic wedge. The wedge is held in place by the intact overlying articular cartilage. Additional trauma causes the articular cartilage to fracture, leading to loosening and detachment of the wedge. Once a wedge has separated into the joint as a loose body, the articular cartilage remains viable because it receives its nutrition from synovial fluid (Fig. 1A). The subchondral bone undergoes necrosis due to loss of nutrition (Fig. 1B).

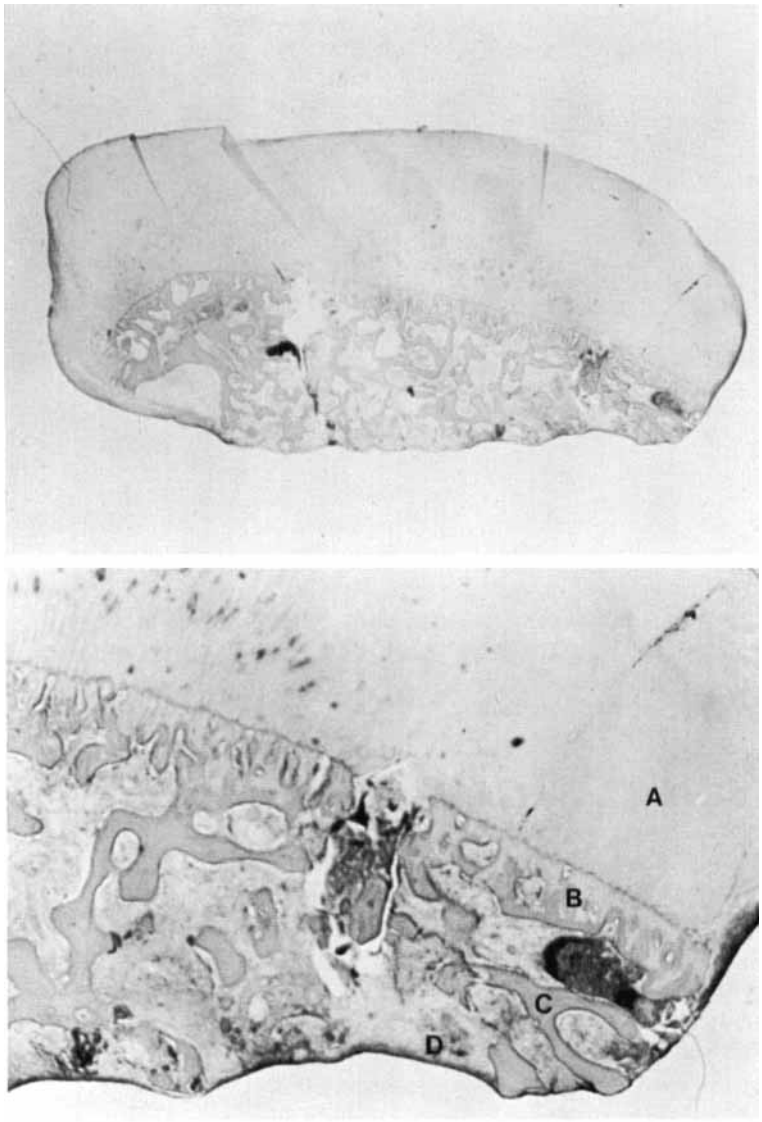
Rogers and Gladstone,[71] however, studied the blood supply to the distal femur anatomically and with injection techniques. They concluded that the subchondral bone had a luxurient blood supply and that ischemia was unlikely.

Intraosseous circulatory disorders can occur nevertheless. Ficat *et al.*[23] contrasted 11 cases of osteochondritis with eight cases of osteonecrosis of the knee, all of whom had hemodynamic disorders with increased marrow pressure and circulatory obstruction owing to stasis. Patients who had osteonecrosis were generally older than 60 years of age. Bone scans were recommended to differentiate these patients from the younger patients who have osteochondritis dissecans. Hence, some early reports of osteochondritis dissecans may actually have included cases of osteonecrosis, *e.g.*, the report by Wolbach and Allison[94] of a diabetic at autopsy.

Accessory Centers of Ossification

The difference in the clinical picture between the skeletally mature patient and the child with open epiphyses has led some to conclude that osteochondritis dissecans in young patients may simply be a variant of normal growth. Sontag and Pyle[79] were the first to note roughening or complete loss of the regular epiphyseal outline as normal in the distal femoral epiphyses of children. They noted this after reviewing roengenographs from 220 children and were able to correlate the changes with periods of rapid growth.

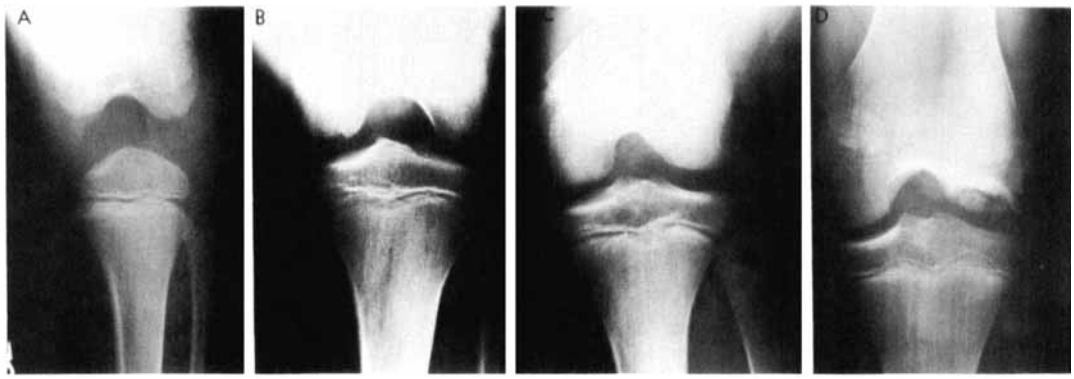
Caffey *et al.*[12] clarified the work of Sontag and Pyle,[79] reviewing knee roentgenographs of children referred to radiology for roentgenography other than the knee. They found marginal irregularities to be common and subsequently divided the patients into three groups by roentgenographic appearance. Group I patients had varying degrees of roughening of the margins and occasional small foci of calcification beyond the roughened margin (Fig. 2A). Group II patients had larger marginal irregularities in the form of indentations (Figs. 2B and 2C). Group III patients resembled Group II, with the addition of an independent island of bone in the marginal crater (Fig. 2D). They noted involvement of both condyles in 44% of cases, the lateral condyle only in 44%, and the medial only in 12%. While the changes were often present in both knees, they were not necessarily bilaterally symmetrical. They postulated that the changes they observed radiographically in Groups II and III patients were similar to osteochondritis dissecans. Unfortunately, they were able to obtain follow-up roentgenographs in only two patients; one showed partial filling of the



FIGS. 1A AND 1B. (A, top) Low power photomicrograph of an osteochondritic dissecans loose body (original magnification ×10) (B, bottom) High power photomicrograph of same lesion (original magnification ×35): (A) articular cartilage; (B) subchondral bone; (C) necrotic trabeculea; (D) fibrous granulation tissue.

defect at 18 months, and the other had a normal appearing condyle at 27 months. The latter case may have indeed been a circumstance of spontaneous healing of childhood osteochondritis dissecans, inasmuch as a history of mild trauma was reported with discomfort in the knee. The authors excluded three other cases of Group III defects, because they were referred for knee roentgenographs as a result of trauma and a history of knee symptoms. All three patients had bilateral involvement with symptoms in only one knee and spontaneous disappearance of their defects (Figs. 2B and 2C). The type of treatment was not mentioned.

Ribbing[68] reviewed roentgenographs of knee joints in 291 children and compared those evidencing detached osseous islets within the articular cartilage outside the epiphysis to those evidencing osteochondritis dissecans. He found the locations similar and concluded that the ossification center was a



FIGS. 2A–2D. Caffey classifications: (A) Type I: irregular ossification; (B) Type II: initial roentgenograph; (C) Type II: roentgenograph after healing; (D) Type III.

"locus minoris resistentiae." His proposal for the etiologic mechanism of osteochondritis dissecans was that an accessory bone nucleus separates in childhood, with subsequent partial reattachment. Further trauma could result in complete separation.

GENETIC

Numerous studies have mentioned a familial incidence of osteochondritis dissecans.[7,26,28,35,61,66,80,81,86,91] However, an excellent review article by Petrie in 1977,[65] showed no definite genetic etiology associated with the condition. He studied the first-degree relatives of 34 patients who had osteochondritis dissecans and found only one who had osteochondritis dissecans, indicating the rarity of a hereditary influence.

Multiple epiphyseal dysplasia (MED) must always be considered in patients who have osteochondritis dissecans. Because MED has autosomal dominant and recessive hereditary patterns, patients thought to have a familial form of osteochondritis dissecans may actually have had variable expression of MED. Heredity apparently has little if any relationship with osteochondritis dissecans other than a possible rare familial form that may exist.

An association of osteochondritis dissecans with dwarfism,[91] tibia vara,[92] and Legg-Calvé-Perthes disease,[96] has also been mentioned in the literature. Many of the prior studies have commented on a relationship between dwarfism and osteochondritis dissecans, although this was not confirmed by Petrie.[65]

Hence, a review of the literature finds that the etiology of osteochondritis dissecans remains questionable. The obvious alternative is a multifactorial etiology.

MATERIALS AND METHODS

Osteochondritis dissecans is commonly encountered in the young male athlete and most often (75% of cases) occurs in the knee. Other sites include the patella,[20,82] the capitellum of the distal humerus,[40,77,85,95] the talar dome,[77] and the femoral head.[53,77,96] Males predominate (about 2:1 or 3:1), and the incidence of bilaterality is about 30%. The condition is rare in patients younger than ten years of age or older than 50.[47] The authors' experience and that found in the literature pose two different clinical subsets: osteochondritis dissecans occurring in (1) the child or young adolescent five to 15 years of age who has open physes; (2) in the older adolescent or adult 15 to 50 years of age. These cases are less frequently bilateral and the onset is more acute.[70]

HISTORY

The symptoms with osteochondritis dissecans are often vague and poorly localized. Pain is of varying degrees, with stiffness and, infrequently, knee swelling. As joint incongruity progresses, a

sensation of catching, locking, or giving way develops. Symptoms are usually intermittent and related to exertion, and as the fragment separates and loose bodies form, symptoms become more specific.

Physical Examination

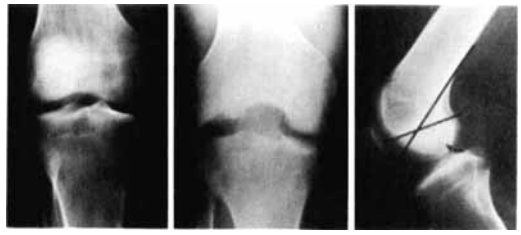
At physical examination, evidence of quadriceps atrophy and weakness with an occasional effusion may be found. The involved femoral condyle may be tender to palpation when the knee is flexed. Rarely, the loose body or the defect may be palpated. A child will often walk with the tibia externally rotated to avoid impinging the tibial eminence with the lateral aspect of the medial femoral condyle. Wilson's sign, tested by flexing the knee to 90°, internally rotating the tibia and extending the knee slowly, may be positive (pain at 30° flexion indicates a positive sign). The pain is often relieved by external tibial rotation.[93]

Roentgenographic Examination

Roentgenographic examination is diagnostic, revealing a well circumscribed area of subchondral bone separated from the remaining femoral condyle by a crescent-shaped radiolucent line. The classic location is the posterolateral aspect of the medial femoral condyle (Fig. 3B); hence, a standard anteroposterior roentgenograph of the knee may miss the lesion (Fig. 3A). An intercondylar notch or tunnel view is often more useful (Fig. 3B). The lateral roentgenograph of the knee will usually show the defect between the line formed by the intercondylar notch and the line extended from the posterior femoral cortex, as reported by Harding[36] (Fig. 3C). In 75%–85% of cases, the medial femoral condyle is involved. Approximately 20% of these will include a significant portion of the weight-bearing condylar surface. Rarely, osteochondritis dissecans involves the articular suface of the patella.

Milgram[59] showed that the radiodensity present in specimens of osteochondritis dissecans is owing to (1) subchondral bone with articular cartilage; (2) secondary calcification in degenerating articular cartilage; (3) new bone formation following revascularization; (4) calcification in new surface layers of cartilage and bone.

Occasionally, arthrograms are of value in determining an irregular joint surface or a separated fragment. Almgard and Wikstad[3] recommended routine use of the arthrogram and based their treatment protocol on arthrographic findings.



FIGS. 3A–3C. (A, left) Anteroposterior roentgenograph of medial condylar osteochondritic lesion. (B, center) Tunnel view of same lesion. (C, right) Lateral view showing typical location of osteochondritis dissecans.

Arthroscopy

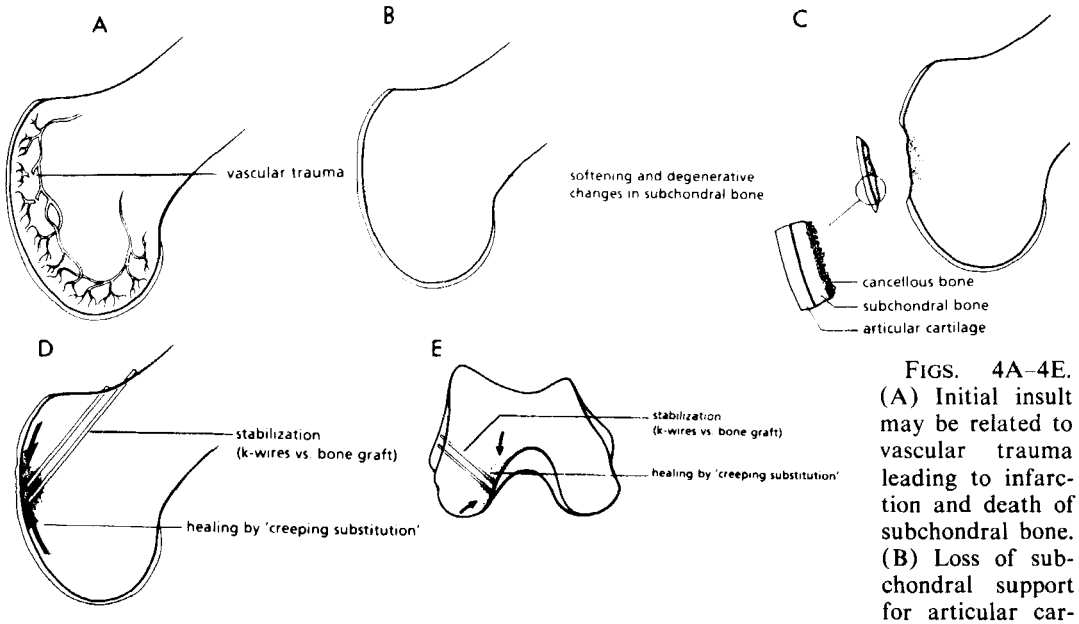
Arthroscopy has become an invaluable tool in the treatment of osteochondritis dissecans.[9,34] Arthroscopy allows direct visualization of the involved area and provides, in conjunction with probing, a method for determining the degree of articular cartilage separation. The base of an osteochondritic defect can be drilled under arthroscopic control, and a partially separated fragment can be stabilized with Kirschner wires or bone pegs.[34] The arthroscope also provides an atraumatic means for removing loose bodies. Finally, in some cases, follow-up arthroscopy can be used to confirm reconstitution of the articular surface prior to returning the patient to full activity.

Pathology

Green and Banks[31] suggested that the pathologic course was related to death of subchondral bone which had subsequently resorbed (Fig. 4A). A support for the articular cartilage is lost, leading to cartilage softening and degenerative changes (Fig. 4B), and when unprotected, additional trauma can cause fragment separation (Fig. 4C). With protection, they suggested that the cartilage would remain intact and healing would ensue in the dead bone layer by "creeping substitution" (Figs. 4D and 4E). They demonstrated good results in children who were treated conservatively.

King[40] reported a series of patients who had osteochondritis dissecans in whom he noted an inflammatory reaction and foreign body giant cell formation around a partially necrotic bony nucleus. The patients' ages were not given.

Landells[42] proposed that injured articular cartilage separated at the junction of calcified and uncalcified cartilage, but this is rare in specimens of osteochondritis dissecans examined microscopically (Figs. 1A and 1B).



FIGS. 4A–4E. (A) Initial insult may be related to vascular trauma leading to infarction and death of subchondral bone. (B) Loss of subchondral support for articular cartilage leads to softening and degenerative cartilaginous changes. (C) Separation of the osteochondritic fragment occurs with additional trauma. (D) Protection by stabilization with Kirschner wires or bone pegs allows healing by "creeping substitution." (E) Anterior view of same lesion.

In an experimental study, Convery *et al.*[17] created defects in the articular surface of the medial femoral condyles of Shetland ponies. Three millimeter lesions healed within nine months. Some loss of function was noted in those with 9 and 15 mm defects, while gross lameness was common in those with 21 mm defects. They concluded that the subchondral bone supplied the tissue (fibrous tissue, fibrocartilage, or bone) that filled the defect. The degree of repair was directly proportional to the size of the defect.

Chiroff and Cooke[16] studied histologically and microradiographically the osteochondritis lesion in six patients ranging in age from 13 to 29 years. No patient had separated lesions, and the cartilage was viable in all cases. The underlying bone was viable and appeared actively engaged in a reparative-type process. The bed was composed of fibrocartilage undergoing enchondral ossification. On the basis of these findings, Chiroff and Cooke recommended nonoperative treatment unless the lesion separates. However, their findings were not confirmed in a similar study by Linden and Telhag.[49]

Once the lesion separates, a loose body results. The etiology and pathology of loose bodies have been studied as a separate entity since Pare's report in 1840.[64] An excellent review of this history was published by Fisher in 1921.[25]

TREATMENT

Treatment of osteochondritis dissecans is dependent on the patient's age and degree of involvement (Figs. 5 and 6). Most children can be treated by observation or immobilization as symptoms dictate, and good results may be expected. Lofgren[55] and Van Demark[87] mentioned spontaneous healing of osteochondritic defects in children, which was reiterated by Edelstein.[19] Of the six cases he described, one had normal roentgenographic findings within two months and the other five required nine to 27 months. Care must be taken not to view all cases that heal by immobilization as cases of spontaneous healing; some may represent irregular ossification, as in a four-year-old reported by Strange.[83]

Smillie[76] suggested a period of immobilization of up to 16 weeks for nondisplaced lesions in patients who are younger than 15 years of age. Open drilling and possibly, pinning, were reserved for partially or completely separated fragments. Smillie pro-

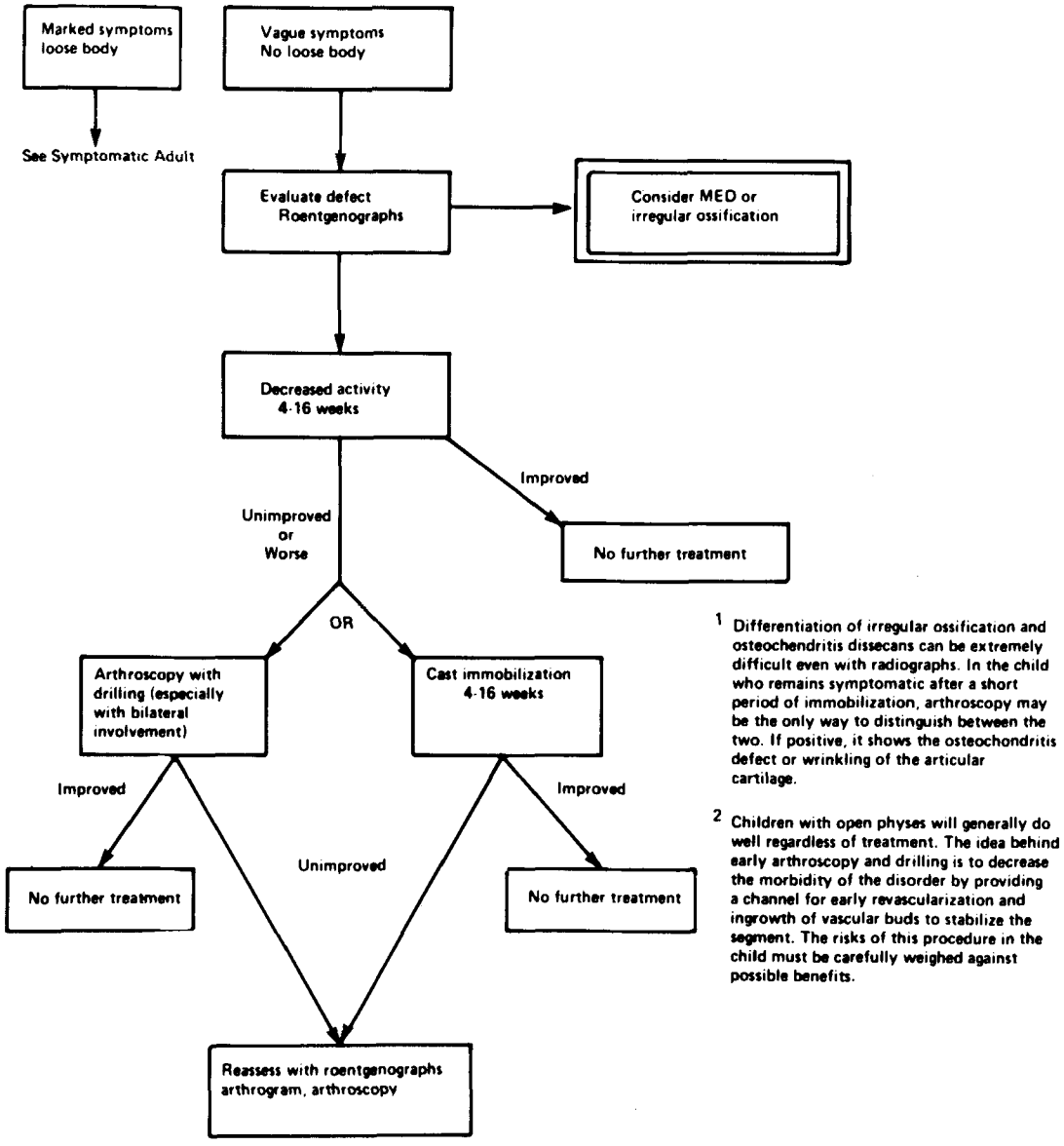
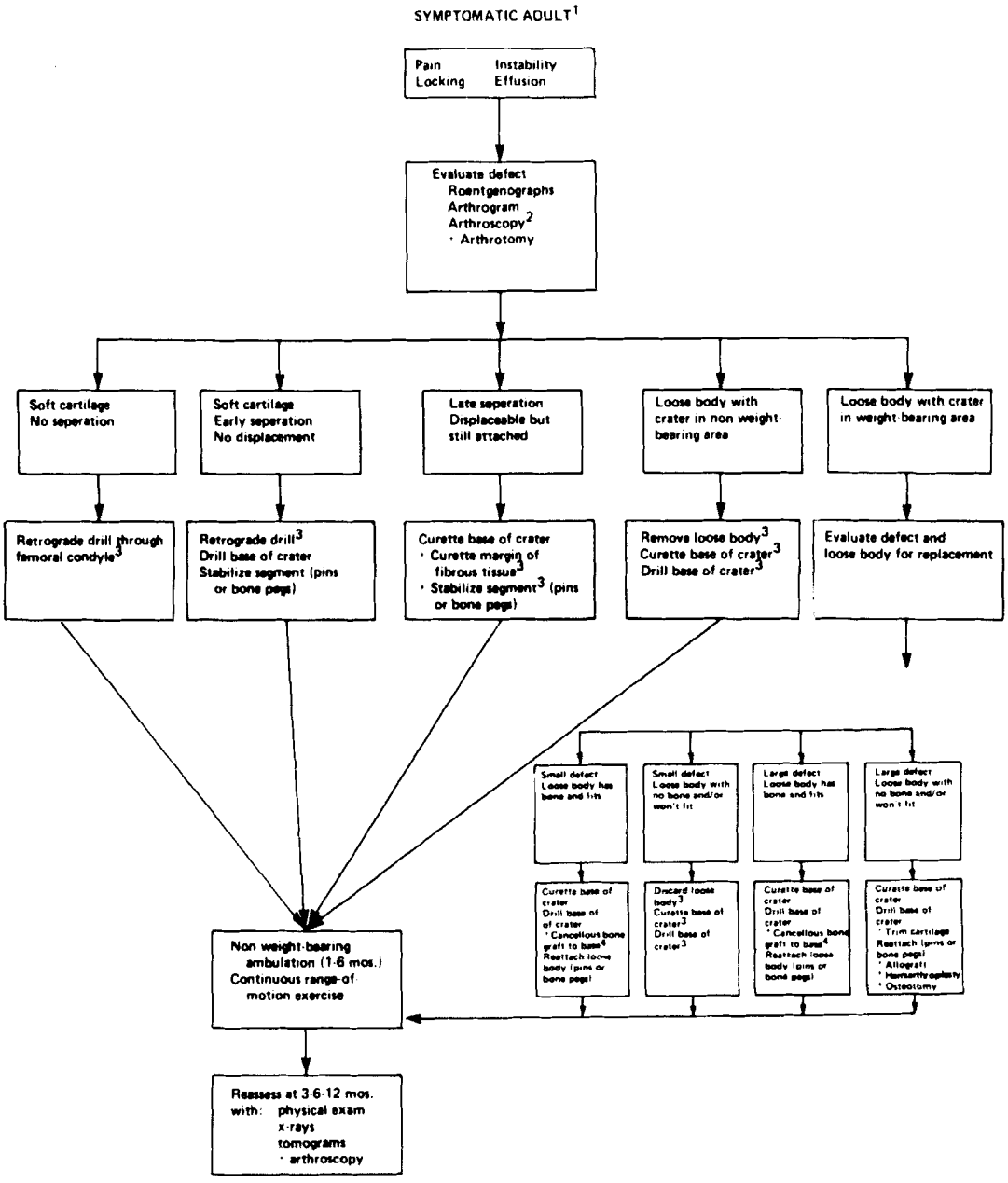


FIG. 5.

posed that a cleavage plane in the subchondral bone progressed to delayed or nonunion without immobilization. He advocated excising any fibrous tissues within the crater, drilling the base and fixing the loose fragment with a screw. This procedure required a second arthrotomy to remove the screw. The patients were immobilized initially for 12 to 16 weeks, with nonweight-bearing ambulation.[77]

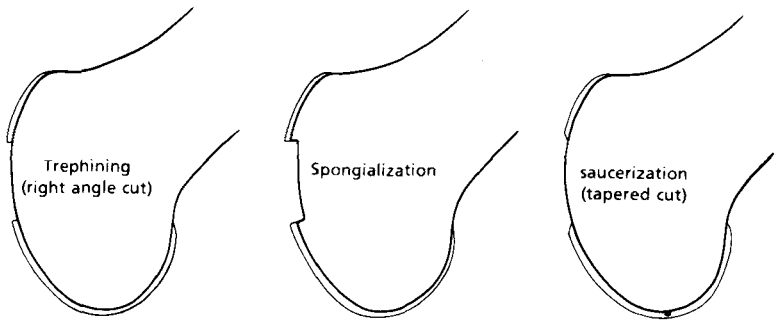
The arthroscope is a valuable tool in treating children, allowing drilling of unseparated bilaterally symptomatic knee lesions without requiring prolonged cast immobilization. The difference between treating osteochondritis dissecans in a child as compared to an



[1] The nonoperative approach is not recommended because of the degenerative joint changes which will unquestionably result
[2] Arthroscopy has minimal morbidity and allows excellent evaluation of the defect with the opportunity for some forms of treatment
[3] These are procedures which can often be done through the arthroscope.
[4] Cancellous bone grafting can be used to provide support to a loose segment and to elevate it to restore normal joint congruity

FIG. 6.

adult is extremely important. The function of drilling in the child who does not respond quickly to immobilization is merely to speed the revascularization process and to reduce the period of morbidity; most will do well regardless of the treatment protocol.



FIGS. 7A–7C. Methods of dealing with articular cartilage defects: (A, left) trephining; (B, center) spongialization; (C, right) saucerization.

In the older group, the prognosis is less satisfactory regardless of treatment protocol, as emphasized by Linden[48] in a study of the long-term results in osteochondritis dissecans. He evaluated 67 joints in 58 patients, with an average follow-up of 33 years. No loose fragments were replaced, nor was any internal fixation used. He concluded that children did well with no secondary degenerative change or complications. Adults, on the other hand, often had pain, instability, decreased range of motion, angular deformity, or other disease complications. The signs and symptoms of degenerative arthritis eventually appeared in 100% of patients who developed osteochondritis dissecans when adults. It should be noted that 28 of 40 adult joints in this series also had evidence of chondrocalcinosis, which is known to be a factor in the development of arthritis. As a result, caution must be taken before accepting Linden's estimate that at least 4% of cases of gonarthrosis in men are caused by osteochondritis dissecans.[47]

Careful assessment of the patient and the lesion is mandatory. Correlation of the size and location of the lesion with the patient's age and clinical presentation is important.[75] Determination of the surface topography by arthrography or arthroscopy is essential. The latter technique is useful not only in establishing the diagnosis, but a lesion may be drilled, curetted or pinned through the arthroscope and loose bodies removed with minimum morbidity.

In selecting the appropriate individualized treatment, O'Donoghue[62] suggested not replacing free fragments, encouraging active function, and trephining the lesion rather than saucerizing it. Trephining entails cutting the edges of the defect at right angles to the articular surface rather than tapering (Fig. 7). Theoretically, this will allow more congruous ingrowth by replacement fibrocartilage and less tampering with the normal articular surface, and it avoids creating any further joint instability. Ficat *et al.*[24] proposed another method for treating cartilage defects which they termed spongialization, removal of subchondral bone to bleeding cancellous bone (Figs. 7A–7C).

In 1975, Larson[45] reviewed the location of lesions and discussed treatment. He basically condemned the conservative approach, particularly when signs of locking, a loose body, or an acute condylar defect were present. Furthermore, he pointed out that delayed surgery could complicate repositioning the free fragment, emphasized trephining rather than tapering cartilage edges, and advised burying pins in the articular cartilage to prevent synovial irritation, pannus formation, and joint stiffness.

Reattaching a separated fragment with the use of a peg bone graft was first mentioned in the English literature in 1955, in the discussion by Osborne of a paper presented by Smillie.[75] Bandi and Allgower in 1959,[5] advocated drilling and bone pegging. Scott and Stevenson[74] also used internal fixation with autogenous bone pegs and fine threaded Kirschner wires. The fashioning of

cortical matchstick bone peg grafts from the proximal tibia with a cancellous graft to the bed was first reported in five knees by Greville in 1964.[32] Lindholm and Pylkkanen[50] reported eight patients, all of whom united at 2.5 to 10.0 months postoperatively and had good to excellent results at an average ten-years follow-up. The technique of bone pegging was also mentioned by Bigelow in 1975,[8] for use in cases that are unresponsive to conservative treatment. Johnson and McLeod[38] reported several advantages of bone pegging as compared to pin fixation from experience in two patients. These include no need for a second arthrotomy to remove the pins, easy access to the bone pegs, and stimulated revascularization of the subchondral bone. Lindholm and associates[51] also reported the use of bone pegging in a series of 20 patients, with 73% good to excellent results at an average five-year follow-up. They also reported two cases in the hip treated by a similar technique.[51] Gillespie and Day[29] used bone pegs to fix 18 defects in 17 patients, with union achieved in all cases and good results in 16.

Van Der Weyer[88] treated cases of osteochondritis by undermining an *in situ* defect by way of the intercondylar notch and removing the bony focus without disturbing the overlying articular cartilage. The defect was then packed with cancellous bone. Loose fragments were treated similarly; the cartilaginous cap was preserved and sutured in place. If the cap could not be used, a piece of tibial cortex and periosteum were used to recreate the joint surface.

In 1978, Lipscomb *et al.*[54] reported the use of Kirschner wires to reattach loose osteochondral fragments in eight knees of seven patients, with successful union in seven. They emphasized the need to remove fibrous tissue from the crater, freshen dense cortical bone, and restore joint congruity with a cancellous bone graft in the bed before replacing the lesion. Pins were removed within three to 16 weeks, but they currently advocate removing pins within three to six weeks, with mobilization at removal. Weight-bearing was begun when there was roentgenographic evidence of early union of the fragment to underlying bone. Cameron *et al.*[13] reported the experimental use of porous surfaced Vitallium pins, which bond to subchondral bone and thereby prevent migration and obviate the need for pin removal.

Guhl[34] recently reported the advantages of arthroscopy in treating osteochondritis dissecans. These include immediate evaluation of the surface topography, treatment with a decrease in total rehabilitation time, avoidance of open surgery and associated risk of infection, decreased morbidity, avoidance of the physical as well as psychological scars with knee surgery, and decreased length and hence, cost of hospitalization. He classifies lesions by location, per cent of weight-bearing surface and degree of separation, and then correlates these with treatment. His indications for operative treatment are a symptomatic knee in a patient who is skeletally older than 12 years, a lesion larger than 1 cm, and involvement of the weight-bearing surface. Lesions with intact articular cartilage are simply drilled. Those separated early are drilled and occasionally pinned. Because partial detachment leads to a break in the cartilaginous border and protusion of fibrous tissue, he removes the fibrous tissue to bleeding bone and pins the fragment. Craters are trephined and drilled and fresh loose bodies are replaced and pinned. Cancellous grafts are used in selected cases to restore surface congruity. He reported a series of 42 patients with osteochondritis dessicans, ten of whom had bilateral knee involvement. Forty-three of these were treated entirely via the arthroscope; some were drilled, others drilled and pinned, and others curretted and/or grafted. Of these, 34 healed, eight showed evidence of healing, and nine were too early to evaluate. There was one definite failure. Guhl uses a 22.5 cm Kirschner wire, which was 7.5 cm of raised threads at one end, and cuts the pin off under the skin. The pins remain in place for four to 12 weeks. Postoperative therapy is individualized, but those who have

intact lesions may weight-bear immediately, while those who have detached fragments weight-bear when the lesions are stable. Pins are removed when there is laminographic evidence of early union. Guhl points out that motion is far more important than weight-bearing, as supported by Salter's[73] work on the effect of motion in rabbit knee joints.

Those cases of osteochondritis dissecans that involve a significant portion of the weight-bearing surface of the femoral condyle and in which the segment has completely separated and cannot be replaced pose a serious therapeutic dilemma, especially in the younger patient. Patients older than 60 years of age are candidates for total knee arthroplasty and can anticipate a satisfactory prognosis, whereas the alternatives for the younger patient are not as satisfying.

Much work has been done with fresh osteochondral allografts, and this technique shows promise. Gross[33] reported some excellent results with allografts in patients who had osteoarthritis, osteonecrosis, and posttraumatic arthritis. In patients who have severe varus or valgus deformities, Gross advocates osteotomy followed nine months later by allografting. Osteotomy may, in fact, be a useful alternative in these patients who have significant osteochondritis defects, allowing transfer of stress to the uninvolved condyle. Hemiarthroplasty has been advocated for use in patients who have unicompartmental arthritis[37,46,57] and might play some role in the treatment of osteochondritis dissecans. The authors agree with Laskin[46] and Insall and Aglietti[37] in limiting use of this procedure to the lateral compartment and would use it only when there were no other satisfactory treatment modalities. The use of the allograft, osteotomy, or hemiarthroplasty should be reserved for those severe cases when the only other course is arthrodesis.

After reviewing the literature the authors synthesized the following treatment scheme: It is essential to differentiate between the childhood and adult forms. Treatment varies slightly on an individual basis and according to the physician's experience. The symptomatic child is initially treated by decreasing activity (Fig. 5). After multiple epiphyseal dysplasia and irregular ossification have been ruled out, arthroscopy permits direct visualization of the lesion and drilling of soft but intact articular cartilage. Drilling provides a channel for early revascularization and stabilizes the segment by the ingrowth of vascular buds. Flap fragments are reattached with pins after the base is curetted under arthroscopic control. Detached fragments in the child are treated as in the adult (Fig. 6).

The adult form has greater morbidity. Therefore, treatment is more aggressive in the symptomatic adult and, to a large degree, based on the roentgenographic and arthroscopic appearance of the lesion (Fig. 6). Soft cartilage without separation is drilled retrograde while observing through the arthroscope. Arthroscopy also allows drilling, curettage, and pin or bone peg stabilization of a separated but undisplaced segment. Once separation occurs, a loose body in a nonweight-bearing area is removed during arthroscopy. The crater base is spongialized. Larger loose bodies composed of articular cartilage and bone are reattached, particularly when a weight-bearing section of the femoral condyle is involved. This usually requires an arthrotomy. Cancellous bone graft is used in the base of the crater when needed to support a loose segment and to elevate it for restoring normal joint congruity. When a large, weight-bearing defect cannot be restored, minimal treatment includes spongialization of the crater base. If the defect remains after nonweight-bearing ambulation and continuous range of motion exercises, osteotomy or the use of an allograft is considered. This treatment protocol has been applied in more than 50 cases of osteochondritis dissecans.

SUMMARY

The past and current status of osteochondritis dissecans suggests that there is still no

clear cut etiology. The etiologic mechanism is generally assumed to be multifactorial and related to minor trauma occurring at a susceptible location. The existence of two clinical patterns is important. Conservative treatment should be emphasized in the young patient who has open physes and a more aggressive approach in the older symptomatic patient. Drilling has a use in the loose unseparated fragment. Free fragments should be replaced when possible if they involve a portion of the weight-bearing articular surface. When replacement is impossible, treatment must be individualized, either by trephining or spongialization followed by joint ranging exercises with nonweight-bearing, or in cases which involve a large portion of the weight-bearing surface of the femoral condyle, a more radical treatment, including osteotomy, hemiarthroplasty, or allograft.

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