



## Osteochondral lesions of the talus

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### History

The term “osteochondritis dissecans” (OCD) is attributed to Konig [1] who described the loose body formation that was associated with articular cartilage and subchondral bone fracture. Kappis [2] was the first to describe a similar process that affected ankle joints. Kappis [2] and Konig [1] attributed the formation of OCD lesions to ischemic necrosis of the underlying subchondral bone with eventual separation of the fragment and its overlying articular cartilage.

In 1959, Berndt and Harty [3] published a review of all the literature pertaining to “transchondral fractures of the talus” from 1856 through 1956. They designed a classification system that has become the gold standard of radiographic staging systems. This classic report also summarized their data from reproducing transchondral fractures in 15 cadaveric specimens. These laboratory data are often cited as support for the posttraumatic cause of osteochondral lesions.

Inflammation is not an important factor in the cause of this disorder, and, thus, many investigators avoid the use of “osteochondritis dissecans.” Assenmacher et al [4] used the term “osteochondral lesions of the talus” (OLT) to describe these lesions. Although trauma is not documented in all cases of OLT, a traumatic cause probably plays an important role in the development of many cases.

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## **Anatomy**

Osteochondral lesions of the talus are rare joint disorders. The talus is the third most common location of this disorder, following the knee and elbow joints [5]; OLT represent 4% of all osteochondral lesions in the body.

The dome of the talus is covered by the trochlear articular surface, which supports the weight of the body. The talar dome is trapezoidal in shape and its anterior surface is an average of 2.5 mm wider than the posterior surface. The talus has medial and lateral articular facets that articulate with the medial and lateral malleoli. The articular surface of these facets is contiguous with the superior articular surface of the talar dome.

The talus has no muscular or tendinous attachments and 60% of the surface is covered by articular cartilage. Most of the blood supply of the talus enters through the neck by way of the sinus tarsi. The dorsalis pedis artery supplies the head and neck of the talus. The artery of the sinus tarsi is formed from branches of the peroneal and dorsalis pedis arteries. The artery of the tarsal canal is a branch from the posterior tibial artery. These two arteries join to form an anastomotic sling inferior to the talus, from which branches enter the talar neck.

## **Cause and mechanism of injury, and natural history**

Most anterolateral OLT, but fewer posteromedial lesions, are likely caused by trauma. A collection of studies by Parisien [6], Baker et al [7], Pettine and Morrey [8], Van Buecken et al [9], and Anderson et al [10] reported a history of a traumatic event in more than 85% of patients. Canale and Belding [11] found that all of the lateral talar dome lesions in their patients were associated with trauma, but only 64% of the medial lesions had a traumatic history. Alexander and Lichtman [5] confirmed these results by analyzing 25 patients. They concluded that all lateral lesions were associated with a traumatic event, although 18% of the patients with a medial lesion denied a history of trauma [5]. Flick and Gould [12] reviewed reports of more than 500 patients with OLT and found that 98% of the lateral lesions and 70% of the medial lesions were associated with a traumatic event. Pritsch et al [13] found that in a group of 24 patients, 75% of medial and lateral lesions were preceded by a traumatic event [13].

Lateral lesions on the anterolateral aspect of the talar dome are created by inversion and dorsiflexion force which causes the anterolateral aspect of the talar dome to impact against the fibula. Lateral lesions are usually more shallow and wafer-shaped than medial lesions, possibly because of a more tangential force vector that results in shearing-type forces [14].

Posttraumatic medial lesions are created by a combination of inversion, plantar flexion, and external rotation forces. The posteromedial talar dome impacts the tibial articular surface which creates a relatively more perpendicular force vector that causes a deep, cup-shaped lesion.

Trauma is implicated in a substantially smaller proportion of the medial lesions. Some investigators associate nontraumatic OLT with a primary ischemic event. Other findings that support a nontraumatic causation include reports of lesions that run in families, multiple lesions in the same patient, and identical medial talar lesions that occur in identical twins [15]. The exact cause of the atraumatic variety of OLT remains unknown.

The biomechanical properties of the ankle joint may provide further insight into the pattern of osteochondral injuries. Bruns et al [14] evaluated contact pressures on the talus with varying degrees of lateral ligament transection and ankle positioning. The results showed that the medial rim of the talus was subjected to high pressures, even without ligamentous transection. In 1995, Athanasiou et al [16] published results that corresponded to a cartilage stiffness disparity between the tibia and talus. The tibial cartilage was found to be 18% to 37% stiffer than the anatomically corresponding sites on the talus. Treppo et al [17] and Shepherd and Seedhom [18] also published results on cartilage stiffness and thickness in the lower limb. Their studies suggested that there is an inverse relationship between the mean cartilage thickness and the mean compressive modulus. These four studies may explain the clinically-observed cause of OLT, with a repetitive overuse syndrome cause for medial lesions and an acute traumatic event cause for lateral lesions.

The biomechanical literature suggested that the size of the OLT may alter the contact stresses that are observed in the ankle. Christensen et al [19] found statistically significant changes in contact characteristics for lesions that were larger than 7.5 mm × 15 mm [19]. Therefore, determining the size of a lesion may be a factor in predicting the long-term outcome.

Despite the detailed anatomical descriptions and causal relationships described above, little has been published on the natural history of osteochondritis of the ankle joint. McCullough and Venugopal [20] explored the natural history of OLT and how it relates to the development of osteoarthrosis of the ankle joint. They reported the results of 10 patients who were treated with various methods with an average follow-up of 15 years. The results showed that conservative treatment often failed to achieve radiographic union; however, osteoarthrosis seemed to be an uncommon sequela, unless the fragment became completely separated. The investigators concluded that acute fractures should be reduced and internally fixed immediately to avoid the development of interposing fibrocartilage and possible complete separation. If a fragment is nondisplaced, then conservative treatment can be used. Surgical treatment should follow if displacement occurs or if mechanical symptoms recur after 2 months of immobilization.

Bauer et al [21] reported the 20-year natural history of 30 patients with osteochondritis dissecans of the ankle. Their findings were that most patients will not develop clinically or radiographically significant osteoarthrosis of the ankle joint. This study also showed that OLT in children (skeletally immature) have a high likelihood of healing and completely disappearing with conservative treatment only.

## Patient evaluation

Typically, patients present with chronic ankle pain, particularly after an inversion injury to the lateral ligamentous complex. Patients describe pain in the ankle, usually on the side of the lesion that is accompanied by intermittent episodes of swelling. Mechanical symptoms, such as catching and popping, are common, although true joint locking is unusual. Recurrent ankle swelling, weakness, and stiffness can be present. Patients often describe instability symptoms (giving way and frequent inversion injuries), but objective joint laxity is less common. Joint laxity should be assessed with an anterior drawer test and comparison with the contralateral side.

Physical examination findings include tenderness over the lesions. Posteromedial lesions may demonstrate tenderness when palpating behind the medial malleolus with the ankle in a dorsiflexed position. Similarly, anterolateral lesions may reveal tenderness when palpating the anterolateral part of the ankle joint in maximal plantarflexion.

Stroud and Marks [22] developed an evaluation protocol based on their comprehensive review of the available radiologic literature. All patients with an acute ankle injury that is accompanied by hemarthrosis or substantial tenderness are examined first by weight-bearing plain radiography (anteroposterior, lateral, and mortise views). Radiographs in varying degrees of plantarflexion and dorsiflexion may help diagnose posteromedial and anterolateral lesions, respectively. Stone [23] recommended plain radiographs of the opposite ankle because of 10% to 25% occurrence rate of a contralateral lesion [23].

In general, conservative management of OLT should always be initiated. Symptomatic patients with negative plain films should be treated with an initial period of immobilization, followed by a trial of physical therapy. Two studies suggested that a trial of nonoperative therapy does not adversely affect the results of later surgical treatment [5,12]. Pettine and Morrey [8] concluded that even higher stage lesions have a chance of healing with nonoperative therapy. Patients with radiographically evident OLT or patients who remain symptomatic at the 6-week follow-up visit should be evaluated further with an MRI.

MRI studies can identify occult injuries of subchondral bone and cartilage that escape detection by routine radiographic analysis [24]. Classic MRI findings in patients with OLT include areas of low signal intensity on T1-weighted images and high signal rims that surround osteochondral fragments on T2-weighted images [25]. The low signal area on T1 images suggests sclerosis of the bed of the talus and indicates a chronic lesion [26]. The signal rim on the T2 image represents instability of the osteochondral fragment [25,27]. MRI can also denote the changes in the lesions before and after treatment. Higashiyama et al [25] reported on 22 ankles that were scanned before and after treatment of talar lesions. The low signal intensity on T1 images and the rim signals on T2 images were reduced or disappeared completely after arthroscopic drilling.

Magnetic resonance arthrography that using the intra-articular injection of gadolinium can be advantageous in the analysis of articular cartilage, the assess-

ment of stability, and the detection of intra-articular bodies [24]. A physiologic arthrogram in the presence of a significant ankle effusion may also be helpful.

If an OLT is diagnosed, the lesion should be staged. An OLT that is seen on plain films should be evaluated by MRI, to enable the clinician to evaluate the quality of the overlying cartilage and assess the stability of the lesion.

## Staging

In their 1959 work, Berndt and Harty suggested a staging system for OLT [3]. This staging system appeared as a footnote to a table in which their patient data are presented. They did not state clearly whether this system was based on radiographs or surgical appearance. Nevertheless, their system remains the most widely-used radiographic staging system (Fig. 1). Recently, Loomer et al [28] described a modification of this system that included the existence of subchondral cysts (Fig. 2). Ferkel [29] developed a classification scheme based on computed tomography. This system corresponds to stages described by Berndt and Harty [3] but also considers fragment separation, the presence of subchondral cysts, and extent of osteonecrosis. High definition three-dimensional reconstructions have also proven useful in diagnosis and treatment (Fig. 3).

Magnetic resonance imaging also has been used to stage OLT and is sensitive in detecting bone signal changes. Diapola et al [30] developed a prospective MRI classification scheme in 1991. As the techniques have evolved, so have the MRI classification schemes. Hepple et al [31] revised the MRI classification in 1999. This staging system resembles Berndt and Harty's original classification [3] with the added descriptors provided by modern imaging techniques. Stage 1 represents articular cartilage damage only. Stage 2a is cartilage injury with underlying fracture and edema. Stage 2b is the same as stage 2a without the edema. In Stage 3, the fragment is detached (rim signal) but not displaced. In Stage 4, the fragment is displaced and Stage 5 shows subchondral cyst formation (Figs. 1, 4).

Pritsch et al [13] developed one of the first arthroscopic grading systems but only included three classifications. Grade I lesions were intact, firm, shiny

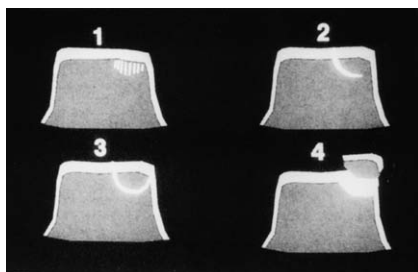


Fig. 1. Berndt and Harty staging system for OLT. (From Taranow WS, Bisignani GA, Towers JD, et al. Retrograde drilling of osteochondral lesions of the medial talar dome. *Foot Ankle Int* 1999;20(8): 474–80; with permission.)

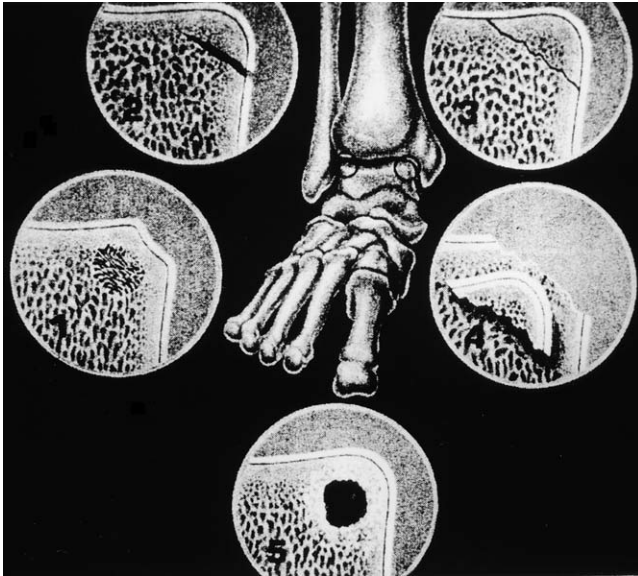


Fig. 2. Loomer et al modified staging system for OLT. The subchondral cyst (stage 5) was the new classification stage proposed by Loomer. (From Shearer C, Loomer R, Clement D. Nonoperatively managed Stage 5 osteochondral talar lesions. *Foot Ankle Int* 2002;23(7):651–4; with permission.)

cartilage. Grade II lesions were intact, but soft cartilage and Grade III was frayed cartilage. Cheng et al [32] developed a more comprehensive arthroscopic classification system after an independent, blind review of 100 arthroscopic videotapes (Table 1).

Despite the abundance of literature that describes systems for classification and staging, there is little confirmation of prognostic value of these schemes. Pettine and Morrey [8] reviewed 71 ankles with an average follow-up of 7.5 years. Using the Berndt and Harty classification system, they found 82% good to fair outcomes in patients with Stage 1 and 2 lesions and only 48% good to fair outcomes for patients with Stage 3 and 4 lesions. Furthermore, if surgical intervention occurred after 1 year for a Stage 3 or 4 lesion, then the results were poor.

### Surgical treatment

Because of variable characteristics of patients (eg, activity level, age, presence of degenerative changes) and lesions (location, size, chronicity), several factors influence surgical treatment. Principles of surgical treatment fall into one of three categories:

- Loose body removal with or without stimulation of fibrocartilage growth (microfracture, curettage, abrasion, or transarticular drilling)

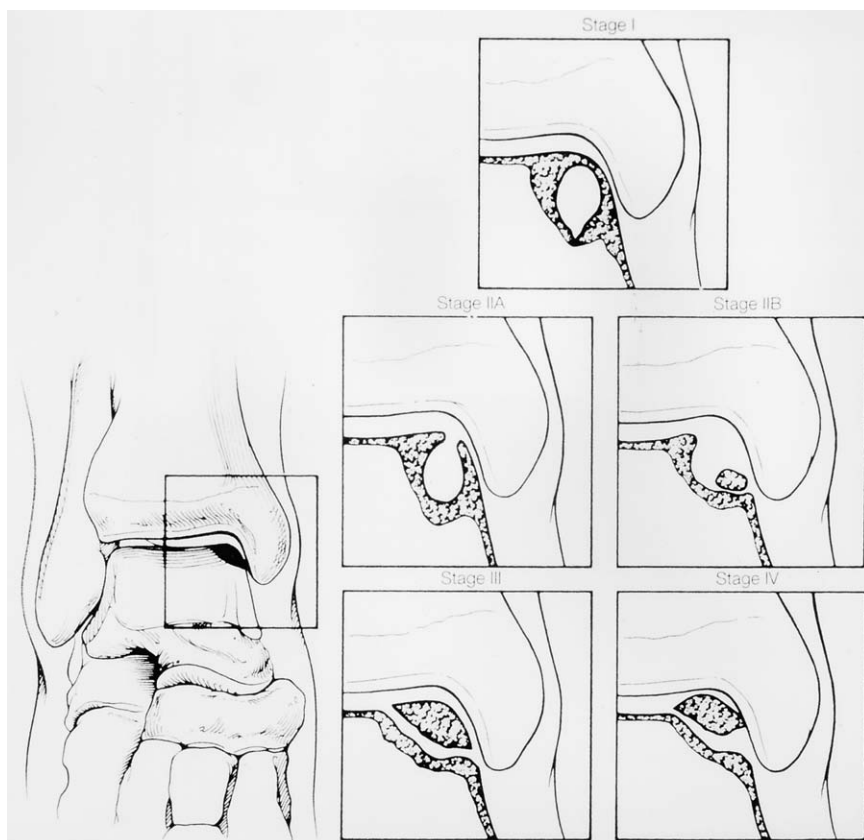


Fig. 3. Classification system for OLT based on computed tomography. (From Ferrel RD. Arthroscopic surgery: the foot and ankle. Philadelphia: Lippincott-Raven, 1996; with permission.)

- Securing OLT to talar dome through retrograde drilling, bone grafting, or internal fixation
- Stimulating development of hyaline cartilage through osteochondral autografts (OATS, mosaicplasty), allografts, or cell culture (Carticel, Genzyme Biosurgery, Cambridge, MA).

A propensity toward surgical treatment dates back to the Berndt and Harty study [3]; they reported poor results in a high percentage of patients who were treated nonoperatively and good results in 84% of patients who were treated surgically. Tol et al [33] confirmed these results recently in a large meta-analysis of OLT treatment strategies that were used from 1966 to 1998. The success rate for nonoperative treatment averaged only 45%. Shearer et al [34] recently published the results of 34 patients with Stage 5 lesions who were treated nonoperatively [34]. This study confirmed the findings of Tol et al [33]; approximately one half of the patients who were treated nonoperatively for OCD of the talus will have a good

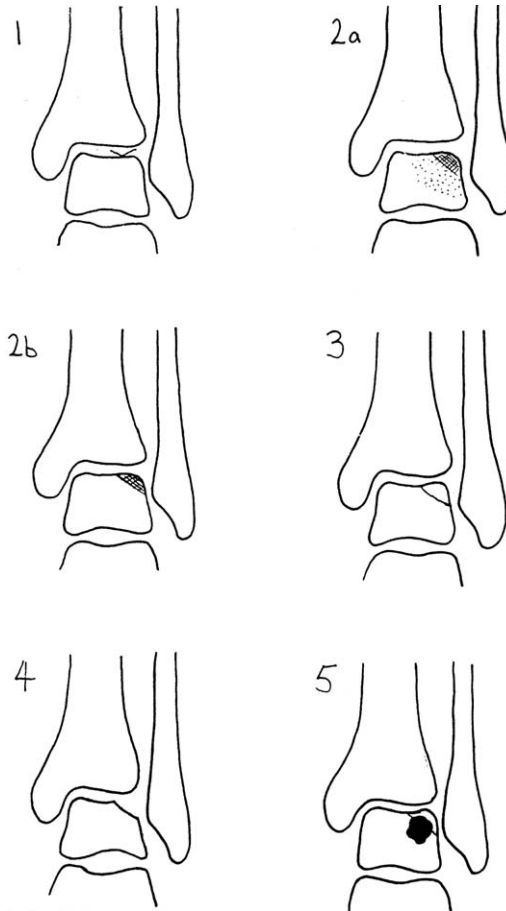


Fig. 4. Classification of OLT using MRI. Grading of lesions similar to those proposed by Berndt and Harty but updated for modern imaging techniques. (From Hepple S, Winston IG, Glew D. Osteochondral lesions of the talus: a revised classification. *Foot Ankle Int* 1999;20(12):789–93; with permission.)

to excellent outcome. This study also confirmed the previously stated finding that osteoarthritis is a rare sequela of OCD of the talus.

The choice of surgical exposure includes open and arthroscopic techniques and depends largely on the expertise of the surgeon. Open surgical exposure of anterolateral lesions is accomplished using an anterolateral approach to the ankle joint. Plantar flexion facilitates exposure of the lesion. This approach requires caution to avoid damaging the branches of the superficial peroneal nerve.

Posteromedial lesions pose an additional challenge with open approaches. Often, a medial malleolar osteotomy is used for additional exposure. Care must be taken to ensure correct placement of the osteotomy. A cut that enters the joint too far laterally endangers the weight-bearing plafond. A cut that is made too far

Table 1  
Cheng-Ferkel staging system for OLT

Stage	Findings	Stability
A	Smooth, intact, but soft or ballottable	Stable
B	Rough surface	Stable
C	Fibrillation/fissuring	Stable
D	Flap present or bone exposed	Unstable
E	Loose, undisplaced fragment	Unstable
F	Displaced fragment	Unstable

distally on the medial malleolus limits exposure. Screw holes must be predrilled before osteotomy. Care must be taken to protect the saphenous nerve and vein, the anterior tibial tendon, posterior tibial tendon, flexor digitorum longus (FDL), posterior tibial artery, and tibial nerve. A variety of bone cuts has been described. An apex proximal chevron cut that was described by Anderson et al [10] allows for anatomic repair with excellent visualization. Cohn et al [35] reviewed their results of chevron medial malleolar osteotomy in 19 patients. At an average of 13 months of follow-up, there were no nonunions or malunions. The most common complaint was prominent hardware, which was treated with screw removal in 16% of the patients. The average time to radiographic union was 7 weeks.

Deland and Young [36] recently described their results with using a combined exposure (anterior and posterior arthrotomy) for treatment of posterior medial OLT [36]. They could access 80% of the talar dome with this combined approach and, thus, avoided the medial malleolar osteotomy in most cases.

Arthroscopic treatment of talar dome osteochondral lesions continues to evolve. Arthroscopic treatment is often performed on an outpatient basis and carries the theoretical benefits of limited surgical morbidity, decreased stiffness, less muscle atrophy, shorter rehabilitation times, and improved functional outcomes. Wide-angle, 2.7 mm arthroscopes offer similar resolution, but more maneuverability, than traditional 4 mm and 5 mm arthroscopes. Another important technical innovation is the development of efficient, noninvasive joint distraction techniques, that result in easier visualization of the entire talar dome.

Despite the advances in arthroscopic visualization, posterior lesions continue to challenge the surgeon. Puri et al [37] and Sitler et al [38] recently reviewed the safety and efficacy of posterior portal ankle arthroscopy in the prone position. These studies suggested that arthroscopic equipment may be introduced into the posterior aspect of the ankle without gross injury to the posterior neurovascular structures.

Schimmer et al [39] performed 413 ankle arthroscopies within 6 years. They believe that ankle arthroscopy should be performed in all patients with OLT. Apart from enabling the various minimally invasive surgical treatment options, they concluded that arthroscopy helps to define treatment strategy and avoids unnecessary surgery on stable lesions.

## **Debridement and microfracture**

When a completely detached lesion is diagnosed and believed to be inappropriate for internal fixation, removal of the loose body and debridement of the bony bed are indicated. The base of the bed should be debrided back to bleeding bone and the edges should be trimmed back to viable cartilage. A variety of specialized surgical instruments should be available, including blunt-tipped probes, pituitary graspers, ring curettes, Kirschner wires, gouges, awls, full radius shavers, and high-speed burrs. After thorough debridement, punctate bleeding should be visualized when inflow pressure is decreased.

Penetration of the subchondral bone stimulates the formation of a fibrin clot and fibrocartilaginous repair tissue often forms in the defect in an effort to protect the bone from loading. This tissue consists mainly of type I collagen with properties that are inferior to those of hyaline cartilage and theoretically predisposes the joint to early degeneration. Angerman and Jensen [40] performed curettage of medial OLTs. They described a deterioration of their results with time with the early onset of pain and degenerative changes. Schuman et al [41] treated 38 patients with OLT with arthroscopic curettage and drilling [41]; 16 of the 38 patients (42%) had failed previous surgery. At an average follow-up of 4.8 years (range, 2 to 10 years), 86% of the patients who had their first surgery and 75% of the patients who had revision surgery reported good or excellent results, as assessed by the Ogilvie-Harris score. This report suggested that arthroscopic curettage and drilling are appropriate in primary and revision cases.

The meta-analysis by Tol et al [33] suggested that simple excision and nonoperative treatment yield uniformly poor results. Excision, curettage, and drilling (ECD) had the highest success rate at 85%, followed by excision and curettage (78%), and excision alone (38%). Although the investigators failed to include results of more current surgical treatments, they demonstrated a high success rate for ECD and concluded that nonoperative therapy and excision alone are not recommended for OLT. A current, prospective study by Thermann et al [42] has shown thus far in early follow-up that microfracture technique is successful in improving function and in restoration of cartilage (as seen on MRI).

## **Drilling of intact lesions**

Recent scientific data are beginning to support drilling of the subchondral bone; this has been practiced by many surgeons to treat intact OLT. Theoretically, these holes provide vascular access channels and promote revascularization of the avascular fragment. Drill holes may be placed through an open or arthroscopic approach. Multiple techniques exist, including the use of existing arthroscopic portals, use of a curved meniscus-repair needle guide [43], transmalleolar drill holes [23], and sinus tarsi approaches to posteromedial lesions. The last, also

known as retrograde drilling or transtalar drilling, has the advantage of not disrupting the articular surface.

Retrograde drilling is gaining popularity and recently has been coupled with bone grafting. The same theory applies; the drill path supplies a route for revascularization, whereas the bone graft or bone graft substitute provides osteoinduction and osteoconduction. This technique is ideal for large subchondral cystic lesions with intact articular cartilage. A new cannulated system, COLT (Interpore Cross, Irvine, CA) facilitates easy and accurate positioning of the drill hole and provides a delivery cannula for bone graft substitute or autograft.

Transmalleolar/transarticular and retrograde/transtalar methods have shown promising results in recent publications. Kumai et al [44] published a study that showed good clinical results and improvement radiographically when patients with OLT were treated with transarticular/transmalleolar drilling. Taranow et al [45], in a similar study, also showed good clinical results with retrograde/transtalar drilling. The latter study had the advantage of not disrupting the articular surface; 16 patients were treated with retrograde drilling and bone

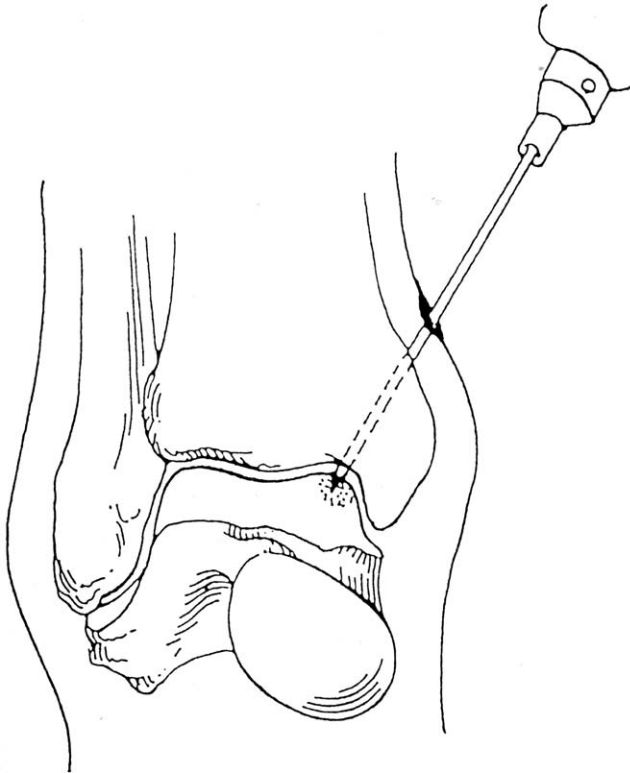


Fig. 5. Illustration of percutaneous transmalleolar drilling. (From Kumai T, Takakura Y, Higashiyama I, et al. Arthroscopic drilling for the treatment of osteochondral lesions of the talus. *J Bone Joint Surg* 1991; 81A:1229–35; with permission.)

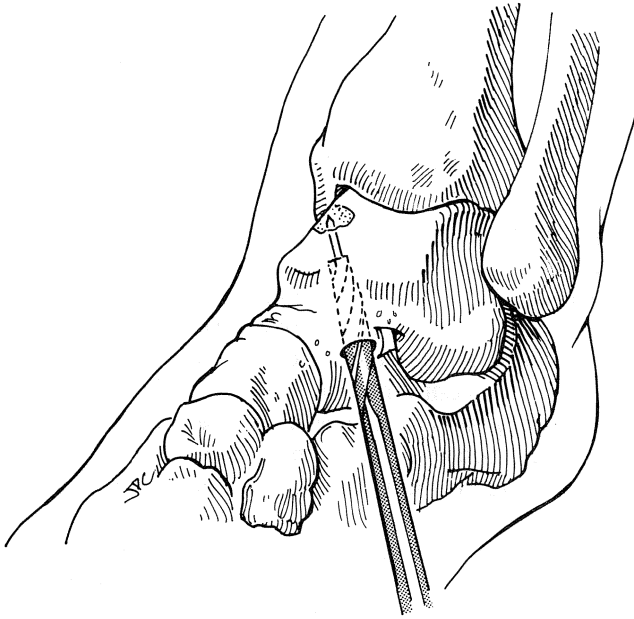


Fig. 6. Cannulated drill placed over guidewire. (From Taranow WS, Bisignani GA, Towers JD, et al. Retrograde drilling of osteochondral lesions of the medial talar dome. *Foot Ankle Int* 1999;20(8): 474–80; with permission.)

grafting. There were no surgical complications and there was a mean increase of 25 points in the American Orthopedic Foot and Ankle Society (AOAFAS) Ankle Hindfoot score (Figs. 5, 6).

### Internal fixation of osteochondral lesions

The ideal candidate for internal fixation of an osteochondral lesion is a young patient with an acute lesion of sufficient size to accept internal fixation. Unfortunately, there is a lack of scientific evidence to define which lesions would benefit from internal fixation.

Internal fixation with traditional bone screws that are passed in an antegrade fashion causes irreparable damage to the intact articular cartilage. Because of the difficult exposure for posteromedial lesions, this technique is typically used only for anterolateral lesions. Most surgeons have been deterred from using traditional screws to internally secure osteochondral lesions.

Although Kirschner wires are unable to provide compression they have the added benefit of retrograde insertion through a nonarticular portion of the talus. The pins are advanced immediately below the articular surface then cut off at the skin for later removal. The use of bioabsorbable pins eliminates the need for future removal.

## **Autograft and allograft**

The role of bone grafting in patients with talar osteochondral lesions is not well defined. There are increasing reports of coupling bone grafting with retrograde drilling of a lesion with a congruent articular surface. Many adult patients present with a displaced lesion and simple bone grafting is not appropriate treatment. Most children will respond to a less invasive method of treatment.

Berlet et al [46] reported the MRI findings at 6 and 9 months following autogenous osteochondral grafting of an OCD of the knee (mosaicplasty). Their case report shows that biological fixation to an OCD is a viable treatment with the advantage of osteoinduction/osteoconduction to complement the stabilization.

Kumai et al [47] treated 27 ankles with large lesions (none was smaller than 8 mm × 8 mm) by using a cortical bone peg technique. These pegs, which were 2 mm to 3 mm wide and 15 mm to 20 mm long, were taken from the distal tibia and passed through the articular surface. Despite the penetration of the articular surface, the investigators retrospectively reported a good (symptom-free or minor symptoms) clinical result in 89% of their patients after a mean follow-up of 7 years.

Gross et al [48] reported an average 11-year (range, 4 to 19 years) follow-up of nine patients who required fresh osteochondral allograft transplantation. Of these nine grafts, six (66%) remained in situ and three cases required ankle fusion because of resorption and fragmentation of the graft. This report discouraged the use of allografts in the treatment of OLT. Brage et al [49] published the results of a series of 16 patients in whom they performed tibiotalar osteochondral allografting. They reported a high number of complications which again emphasizes the difficulty in performing this procedure.

## **Autologous osteochondral grafting (OATS, mosaicplasty)**

These techniques transplant viable osteochondral plugs from the femoral trochlea or condyle into the talar dome. Several proprietary systems exist that differ in one fundamental way: single-plug systems use a single, large plug to match the size of the OLT. Proponents of these systems point to the reduction in fibrocartilage ingrowth when a single osteochondral plug is used; however, critics point to the donor site morbidity of the larger plug harvests. In multiple-plug systems, several osteochondral plugs are used to cover the defect completely. Proponents state that this provides the best match of the talar dome contour and surface area of the OLT. Critics state that because of the small size of the plugs, 20% to 40% of the defect is filled in with fibrocartilage [50]. Gautier et al [51] retrospectively reviewed 11 patients who had been treated surgically by mosaicplasty using Sulzermedica's "Soft Delivery System" (Sulzer Orthopedics, Austin, TX) for open autologous osteochondral grafting. All patients received either a medial malleolar or fibular and Chaput's tubercle osteotomy to improve surgical exposure. The lesions averaged 18 mm × 10 mm. Good to excellent results were obtained for the ankle without adverse effects on the knee at an average follow-up

of 24 months. The investigators stated that there are no reported absolute limits for the size of the defect in the ankle that is suitable for autologous osteochondral grafting. A lower size limit of 10 mm was suggested in previous studies [52].

Hangody et al [52] provided a 2 to 7-year follow-up of “large or unstable” talar osteochondral lesions in 36 patients who were treated with mosaicplasty [52]. Twenty-nine of the 36 patients had failed previous surgical treatment. All of the lesions were greater than 10 mm in diameter, an average of three plugs was used, and the size of plug ranged from 3.5 mm to 6.5 mm. Using the Hannover scoring system, 94% of patients showed good to excellent results with no long-term, ipsilateral knee donor site morbidity.

Assenmacher et al [4] performed arthroscopically-assisted, Arthrex OATS harvest procedures in nine patients and followed them for an average of 9.3 months. Posttransplantation MRI revealed stable graft osteointegration by DeSmet criteria [27] in all patients. Postoperative visual analog pain scales showed significant improvement and AOFAS Ankle Hindfoot scores averaged 80.2. The investigators concluded that this technique is a viable alternative for treating unstable osteochondral defects in the talus that are refractive to more commonly used surgical techniques.

Recently, Al-Shaikh et al [50] retrospectively reviewed the results of the Arthrex single-plug OATS technique in 19 patients. The lesions averaged 12 mm × 10 mm before autografting, and 68% of the patients had failed previous excision, curettage, or drilling of the lesion. At an average follow-up time of 16 months, the AOFAS ankle scores averaged 88 and the Lysholm knee scores averaged 97 (47% of the patients were in litigation or receiving workers’ compensation). There was no evidence of graft subsidence and all grafts and malleolar osteotomies (73% of cases) healed. Eighty-nine percent of the patients stated they would have the procedure again. Al-Shaikh et al [50] concluded that this is an effective salvage procedure after failed previous procedures and for patients with long-standing symptoms. This study used much larger plug sizes (5 mm to 10 mm) than the Hangody et al study [52].

An alternative to the donation of osteochondral plugs from the knee is to harvest the plugs from the ipsilateral talus. Sammarco and Makwana [53] reported the results of the technique in 12 patients [53]. The grafts were harvested from the ipsilateral medial or lateral articular facet of the talus. They report a significant AOFAS score improvement and no structural failures with the donor site or graft site.

### **Autologous chondrocyte transplantation**

Recently, Koulalis et al [54] reported good and excellent results at an average of 17.6 months (range, 8 to 26 months) in eight patients who underwent autologous chondrocyte transplantation. The average lesion size was 14.4 mm × 12.8 mm and six of the eight patients had failed previous surgery. All patients underwent initial diagnostic arthroscopy, cartilage biopsy, chondrocyte extraction, and culture. An arthrotomy, malleolar osteotomy, bone debridement, and chondrocyte

transplantation were performed at an average of 2.5 weeks postoperatively. Patients remained nonweight bearing for 6 to 7 weeks. Routine arthroscopic re-examination at 6 months showed the existence of “cartilage-like tissue” with complete coverage of the chondral defect. The histologic examination of one biopsy failed to show the existence of hyaline cartilage. This early report of a small group of patients provided an exciting treatment alternative. Giannini et al [55] reported similar results in 2001, which showed that hyaline cartilage can be transplanted in the ankle joint and a good functional outcome can be expected.

Autologous chondrocyte implantation (ACI) research has been studied further in the knee. Browne et al [56] recently presented results of a 5-year, prospective, 38-center study. Autologous chondrocyte implantation (or Carticel) is a proprietary cartilage repair process that involves growing the patient’s own cartilage cells and reimplanting them at the cartilage defect site. Seventy-nine percent of patients showed improvement at 5 years. Thirty-eight percent of the patients were involved in workers’ compensation cases and 78% had undergone previous surgery. There were 13 failures. Patients who underwent ACI had greater improvement and reached higher levels of functioning at follow-up compared with a control group of patients who were treated with marrow stimulation techniques, such as drilling, abrasionplasty, or microfracture (MST).

### **Osteochondral lesions associated with ligamentous instability**

Controversy surrounds the timing of treatment of coexisting ligamentous instability and a talar osteochondral lesion. In acute ankle ligament injuries with a large, unstable fragment, many surgeons treat the talar lesions surgically and allow the ligaments to heal postoperatively.

Options for treating chronic talar lesions coupled with chronic ankle ligament instability are less clear. Unfortunately, the postoperative rehabilitation goals of the two procedures are completely different. The early motion that is needed for treatment of osteochondral lesions is detrimental to the reconstructed ankle ligaments. One solution to this dilemma is to perform the surgery for the osteochondral lesion first and treat the ankle instability as a second-stage procedure, if indicated.

Perhaps the postoperative protocols in the treatment of OLT may be more forgiving with regard to motion. The knee tends to be much more dependent on early aggressive motion, in contrast with the ankle. Therefore, if the ankle will tolerate a longer period of immobilization, one could consider treating OLT and instability at the same operative time. Newer stabilization techniques, such as thermal shrinkage, may prove beneficial in this setting [57].

### **Postoperative rehabilitation**

Attaining full ankle range of motion should be the primary initial goal of therapy. Patients who undergo more extensive procedures, such as drilling or

internal fixation, may require a longer period of nonweight bearing (up to 6 weeks or until healing is demonstrated) before beginning a rehabilitation program. A registered physical therapist can tailor a rehabilitation program for each individual patient, which will include an active and passive range of motion home program, edema control modalities, along with strength and proprioceptive training. For patients who wish to return to a high level of physical activity, a plyometric exercise program that involves the optimization of muscle power will be beneficial.

## References

- [1] Konig F. Uber freie korper in den gelenken. *Dtsch Z Chir* 1888;27:90–109.
- [2] Kappis M. Weitere beitrage zur traumatisch-mechanischen Entstehung der “spontanen” Knorpelabiosungen. *Dtsch Z Chir* 1922;171:13–29.
- [3] Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg* 1959;41A:988–1020.
- [4] Assenmacher JA, Kelikian AS, Gottlob C, et al. Arthroscopically assisted autologous osteochondral transplantation for osteochondral lesions of the talar dome: an MRI and clinical follow-up study. *Foot Ankle Int* 2001;22(7):544–51.
- [5] Alexander AH, Lichtman DM. Surgical treatment of transchondral talar-dome fractures (osteochondritis dissecans): long-term follow-up. *J Bone Joint Surg* 1980;62A:646–52.
- [6] Parisien JS. Arthroscopic treatment of osteochondral lesions of the talus. *Am J Sports Med* 1986;14:211–7.
- [7] Baker CL, Andrews JR, Ryan JB. Arthroscopic treatment of transchondral talar dome fractures. *Arthroscopy* 1986;2:82–7.
- [8] Pettine KA, Morrey BF. Osteochondral fractures of the talus: a long-term follow-up. *J Bone Joint Surg* 1987;69B:89–92.
- [9] Van Buecken K, Barrack RL, Alexander AH, et al. Arthroscopic treatment of transchondral talar dome fractures. *Am J Sports Med* 1989;17:350–6.
- [10] Anderson IF, Crichton KJ, Grattan-Smith T, et al. Osteochondral fractures of the dome of the talus. *J Bone Joint Surg* 1989;71A:1143–52.
- [11] Canale ST, Belding RH. Osteochondral lesions of the talus. *J Bone Joint Surg* 1980;62A:97–102.
- [12] Flick AB, Gould N. Osteochondritis dissecans of the talus (transchondral fractures of the talus): Review of the literature and new surgical approach for medial dome lesions. *Foot Ankle* 1985;5:165–85.
- [13] Pritsch M, Horoshovski H, Farine I. Arthroscopic treatment of osteochondral lesions of the talus. *J Bone Joint Surg* 1986;68A:862–5.
- [14] Bruns J, Rosenbach B, Kahrs J. Etiopathogenetic aspects of medial osteochondrosis dissecans tali. *Sportverletz Sportschaden* 1992;6:43–9.
- [15] Woods K, Harris I. Osteochondritis dissecans of the talus in identical twins. *J Bone Joint Surg* 1995;77B:331.
- [16] Athanasiou K, Niederauer G, Shenck R. Biomechanical topography of human ankle cartilage. *Ann Biomech Eng* 1995;23(5):697–704.
- [17] Treppo S, Koepf H, Quan EC, et al. Comparison of biomechanical and biochemical properties of cartilage from the human knee and ankle pairs. *J Orthop Res* 2000;18(5):739–48.
- [18] Shepherd D, Seedhom B. Thickness of human articular cartilage in joints of the lower limb. *Ann Rheum Dis* 1999;58(1):27–34.
- [19] Christensen JC, Driscoll HL, Tencer AF. Contact characteristics of the ankle joint. Part 2. The effects of talar dome cartilage defects. *J Am Podiatr Med Assoc* 1994;84(11):537–47.
- [20] McCullough CJ, Venugopal V. Osteochondritis dissecans of the talus: the natural history. *Clin Orthop* 1979;144:264–8.

- [21] Bauer M, Jonsson K, Lindén B. Osteochondritis dissecans of the ankle. A 20-year follow-up study. *J Bone Joint Surg* 1967;69B:93–6.
- [22] Stroud CC, Marks RM. Imaging of osteochondral lesions of the talus. *Foot Ankle Clin* 2000; 5(1):119–33.
- [23] Stone JW. Osteochondral lesions of the talar dome. *J Am Acad Orthop Surg* 1996;4:63–73.
- [24] Loredó R, Sanders TG. Imaging of osteochondral injuries. *Clin Sports Med* 2001;20(2):249–78.
- [25] Higashiyama I, Kumai T, Takakura Y, et al. Follow-up study of MRI for osteochondral lesion of the talus. *Foot Ankle Int* 2000;21(2):127–33.
- [26] Mesgarzadeh M, Sapega AA, Bonakdarpour A, et al. Osteochondritis dissecans: analysis of mechanical stability with radiography, scintigraphy, and MR imaging. *Radiology* 1987;165: 775–80.
- [27] DeSmet AA, Fisher DR, Burnstein MI, et al. Value of MR imaging in staging osteochondral lesions of the talus (osteochondritis dissecans): results in 14 patients. *Am J Roentgenol* 1990; 154:555–8.
- [28] Loomer R, Fischer C, Lloyd-Schmidt R, et al. Osteochondral lesions of the talus. *Am J Sports Med* 1993;21:13–9.
- [29] Ferkel RD. *Arthroscopic surgery: the foot and ankle*. Philadelphia: Lippincott-Raven; 1996.
- [30] Diapola JD, Nelson DW, Colville MR. Characterizing osteochondral lesions by magnetic resonance imaging. *Arthroscopy* 1991;7:101.
- [31] Hepple S, Winson IG, Glew D. Osteochondral lesions of the talus: a revised classification. *Foot Ankle Int* 1999;20(12):789–93.
- [32] Cheng MS, Ferkel RD, Applegate GR. Osteochondral lesions of the talus: a radiologic and surgical comparison. Presented at the Annual Meeting of the American Academy of Orthopedic Surgeons. New Orleans, 1995.
- [33] Tol JL, Struijs PA, Bossuyt PM, et al. Treatment strategies in osteochondral defects of the talar dome: a systematic review. *Foot Ankle Int* 2000;21(2):119–26.
- [34] Shearer C, Loomer R, Clement D. Nonoperatively managed stage 5 osteochondral talar lesions. *Foot Ankle Int* 2002;23(7):651–4.
- [35] Cohn B, Anderson R, Davis WH. *Transmalleolar osteotomy — an approach to medial talar dome lesion*. San Francisco: American Academy of Orthopaedic Surgeons; 1997.
- [36] Deland JT, Young K. *Medial approaches to osteochondral lesions of the talus without medial malleolar osteotomy*. San Diego: The American Orthopaedic Foot and Ankle Society; 2001.
- [37] Puri RD, Berlet GC, Lee TH. Description of a new posteromedial portal in ankle arthroscopy. Presented at First Pan-American Congress on Medicine and Surgery of the Foot and Leg. Buenos Aires, Argentina: September 13–17, 2000.
- [38] Sittler DF, Amendola A, Bailey CS, et al. Posterior ankle arthroscopy: an anatomic study. *J Bone Joint Surg* 2002;84A(5):763–9.
- [39] Schimmer RC, Dick W, Hinterman B. The role of ankle arthroscopy in the treatment strategies of osteochondritis dissecans lesions of the talus. *Foot Ankle Int* 2001;22(11):895–900.
- [40] Angerman P, Jensen P. Osteochondritis dissecans of the talus: long-term results of surgical treatment. *Foot Ankle* 1989;10:161–3.
- [41] Schuman L, Struijs PA, van Dijk CN. Arthroscopic treatment for osteochondral defects of the talus. Results at follow-up at 2 to 11 years. *J Bone Joint Surg* 2002;84B(3):364–8.
- [42] Thermann H, Becher C, Mueller S, et al. *The microfracture technique for the treatment of osteochondral and degenerative chondral lesions of the talus*. San Diego: The American Foot and Ankle Society; 2001.
- [43] Bryant III DD, Siegel MG. Osteochondritis dissecans of the talus: a new technique for arthroscopic drilling. *Arthroscopy* 1993;9:238–41.
- [44] Kumai T, Takakura Y, Higashiyama I, et al. Arthroscopic drilling for the treatment of osteochondral lesions of the talus. *J Bone Joint Surg* 1999;81A:1229–35.
- [45] Taranow WS, Bisignani GA, Towers JD, et al. Retrograde drilling of osteochondral lesions of the medial talar dome. *Foot Ankle Int* 1999;20(8):474–80.
- [46] Berlet G, Mascia A, Miniaci A. Treatment of unstable osteochondritis dissecans lesions of

- the knee using autogenous osteochondral grafts (mosaicplasty). *Arthroscopy* 1999;15(3): 312–6.
- [47] Kumai T, Takakura Y, Kitada C, et al. Fixation of osteochondral lesions of the talus using cortical bone pegs. *J Bone Joint Surg* 2002;84B:369–74.
- [48] Gross AE, Agnidis Z, Hutchison CR. Osteochondral defects of the talus treated with fresh osteochondral allograft transplantation. *Foot Ankle Int* 2001;22(5):385–91.
- [49] Brage ME, Bugbee W, Tontz Jr. W. Intraoperative and postoperative complications of fresh tibiotalar allografting. Dallas: American Orthopaedic Foot and Ankle Society; 2002.
- [50] Al-Shaikh RA, Chou LB, Mann JA, et al. Autologous osteochondral grafting for talar cartilage defects. *Foot Ankle Int* 2002;23(5):381–9.
- [51] Gautier E, Kolker D, Jakob RP. Treatment of cartilage defects of the talus by autologous osteochondral grafts. *J Bone Joint Surg* 2002;84B(2):237–44.
- [52] Hangody L, Kish G, Modis L, et al. Mosaicplasty for the treatment of osteochondritis dissecans of the talus: two to seven year results in 36 patients. *Foot Ankle Int* 2001;22(7):552–8.
- [53] Sammarco GJ, Makwana NK. Treatment of talar osteochondral lesions using local osteochondral graft. *Foot Ankle Int* 2002;22(8):693–8.
- [54] Koulalis D, Schultz W, Heyden M. Autologous chondrocyte transplantation for osteochondritis dissecans of the talus. *Clin Orthop* 2002;395:186–92.
- [55] Giannini S, Buca R, Grigolo B, et al. Autologous chondrocyte transplantation in osteochondral lesions of the ankle joint. *Foot Ankle Int* 2001;22:513–7.
- [56] Browne JE, Anderson AF, Micheli LJ, et al. Five-year multicenter outcome of autologous chondrocyte implantation of the knee: results in the first 100 consecutive patients. Dallas: American Academy of Orthopaedic Surgeons; 2002.
- [57] Berlet GC, Saar W, Lee TH. Thermal assisted capsular modification for chronic ankle instability. *Techniques Foot Ankle Surg* 2002;1(2):145–50.