Arthroscopic Assessment of Occult Intra-articular Injury in Acute Ankle Fractures

Gregory J. Loren, M.D., and Richard D. Ferkel, M.D.

Purpose: To arthroscopically assess the pattern and extent of intra-articular damage associated with ankle fractures. **Type of Study:** Prospective case series. **Methods:** From 1989 to 1998, 48 consecutive patients with acute unstable ankle fractures underwent ankle arthroscopy followed by reduction and internal fixation. In addition, all injuries were categorized by specific osseous, ligamentous, and articular pathology, based on clinical and arthroscopic examination. Whenever possible, the fractures were classified according to Lauge-Hansen and Danis-Weber schemes. **Results:** Traumatic articular surface lesions (TASLs), including chondral defects and osteochondral lesions measuring greater than 5 mm in diameter, were identified in 30 of the 48 ankles (63%), with 11 lesions localized to the tibia and 19 noted on the talus. The tibial lesions were at the posterior syndesmotic ligament insertion in 6 cases, at the anterior capsule origin in 3 cases, and at the central articular surface in 2 cases. Of the 19 talar lesions, 15 involved the medial dome and 4 involved the lateral articulation. TASLs of the talus in this series were uniformly unstable or displaced and virtually devoid of subchondral bone, precluding satisfactory internal fixation. Of the 10 pronation-external rotation fractures, 7 had articular surface defects with 5 involving the medial and 2 the lateral dome. Ten of the 24 supination-external rotation or Danis-Weber B fractures were found to have TASLs of the talus, 9 medial and 1 lateral. Nine of 12 fractures with syndesmosis disruptions sustained full-thickness damage to the talar chondral surface ($P = .01$). **Conclusions:** Ankle fractures have a high incidence of concomitant intra-articular pathology with syndesmosis disruption portending a particularly high risk of articular surface injury to the talar dome. Arthroscopy is a valuable tool in identifying and treating intra-articular damage that would otherwise remain unrecognized and may provide prognostic information regarding the functional outcome of these injuries. **Key Words:** Ankle—Arthroscopy—Fractures—Osteochondral lesions—Open reduction—Percutaneous osteosynthesis.

The improved functional outcome following a fracture of the ankle in the latter half-century has emphasized anatomic restoration of the ankle joint and mortise.[1-3] Residual problems, including chronic pain, arthrofibrosis, recurrent swelling, and perceived instability, continue to occur unpredictably in the setting of significant ankle injury. The cause of such poor outcomes is unclear, yet may be related to occult articular trauma.[4-7] The clinical result, then, may reflect not only a suboptimal reduction, but also a concomitant chondral injury.[8] The pattern and extent of articular injury associated with acute ankle fractures have not previously been comprehensively evaluated because thorough inspection of the articular cartilage and intra-articular ligaments was not possible, even with classic open reduction and internal fixation.

With advanced video fiber optics, small-joint instrumentation, and improved joint distraction methods, ankle arthroscopy has become a safe and effective technique to address various pathologic conditions involving the tibiotalar joint. Ankle arthroscopy is useful as both a diagnostic and therapeutic procedure and, consequently, the clinical indications for operative ankle arthroscopy have expanded.[9] Ferkel and

From the San Dieguito Orthopaedic Medical Group, Inc., Encinitas (G.J.L.); the Southern California Orthopedic Institute, Van Nuys; and the Department of Orthopaedic Surgery, UCLA Center for the Health Sciences, Los Angeles (R.D.F.), California, U.S.A.

Address correspondence and reprint requests to Richard D. Ferkel, M.D., 6815 Noble Ave, Van Nuys, CA 91405, U.S.A. E-mail: ferkel@scoi.com

0749-8063/02/1804-2348$35.00/0
doi:10.1053/jars.2002.32317

Orwin[10,11] have documented the value of ankle arthroscopy in evaluating and treating ankle fractures. In acute injuries, ankle arthroscopy allows careful examination of the chondral surfaces of the talus, tibia, and lateral malleolus, as well as the capsular and intra-articular ligaments. Moreover, in the less displaced fracture, arthroscopy may facilitate reduction and internal fixation of the articular fragments.[10-14]

The purpose of this study was to document arthroscopically the prevalence of intra-articular pathology in patients with acute ankle fractures. The articular surface lesions (chondral and osteochondral defects) and ligamentous injuries were subsequently evaluated in consideration of the fracture morphology enabling us to propose suggestions for arthroscopic intervention following ankle fractures.

METHODS

Patient Demographics

From 1989 to 1998, 48 consecutive patients with acute unstable fractures of the ankle underwent ankle arthroscopy followed by open reduction and internal fixation. In no case did customary preoperative anteroposterior, lateral, and mortise radiographs of the injured ankle reveal an osteochondral fracture. There were 19 female patients and 29 male patients. The mean age at injury was 35 years (range, 12 to 77 years). The mechanism of fracture involved a fall in 34 patients, a motor vehicle collision in 3, and a sporting activity in 1 (6 in football and 1 each in surfing, skiing, wrestling, hockey, and dodgeball). The interval from injury to surgery averaged 7 days (range, 1 to 26 days), varying dependent on referral delays or excessive swelling. There were 23 right and 25 left ankle fractures; no bilateral injuries were encountered. None of the patients had previous ankle disability.

Fractures were classified radiographically by both the Lauge-Hansen[15,16] and Danis-Weber[17,18] schemes. Fracture configurations such as triplane, Tillaux, and isolated medial malleolus fractures that failed classification were anatomically described. Additionally, all injuries were categorized by specific osseous, ligamentous, and articular pathology based on clinical and arthroscopic examinations.

Surgical Technique

The operative procedure was performed under general, spinal, or epidural anesthesia. The patient was positioned supine with a padded bolster placed under the ipsilateral hemipelvis. A thigh holder was used to facilitate hip flexion of 50° to 60°. A proximal pneumatic tourniquet was applied in all cases, yet was not inflated unless excessive bleeding prohibited adequate visualization. A radiolucent table was used to permit fluoroscopic evaluation if indicated during the case. Ankle distraction was achieved by gravity alone or by manual traction on a clove-hitch strap.[9]

Following sterile preparation, surface anatomy was carefully outlined. In the swollen ankle, more care and time is necessary to delineate the important structures. Standard anteromedial, anterolateral, and posterolateral portals were made as previously described.[19] A 2.7 mm, 30° short arthroscope was used; motorized arthroscopic resectors and burrs, basket forceps, curettes, and Freer elevators were used as needed. After the joint had been entered, the hemarthrosis and fracture debris were evacuated by copious irrigation, careful shaving, and gentle curettage. Gravity inflow-outflow was used. The entire joint was inspected systematically using the 21-point examination, with visualization from both anterior and the posterior portals in all cases.[20] Articular surface and ligamentous lesions as well as loose bodies were noted.

Chondromalacia was graded as described by Outerbridge[21] and unstable chondral flaps were debrided. Free chondro-osseous fracture fragments were removed. Articular surface lesions were assessed for degree of injury and stability. Fragments devoid of bone were excised at the discretion of the senior author and the fracture bed gently abraded or percutaneously drilled with a Kirschner wire, if there were concerns about vascularity to the site. Intra-articular ligament integrity and syndesmosis stability were also evaluated arthroscopically. Ligament tears were documented and debrided when appropriate.

Percutaneous manipulation of fracture fragments under direct visualization was performed using fluoroscopy as needed. If arthroscopically assisted reduction of the medial malleolus or distal tibia fracture was successful, cannulated screw fixation under fluoroscopic control was undertaken without open exposure of the fracture site. Caution was exercised to protect and expose as necessary regional neurovascular structures, such as the saphenous vein or nerve and superficial peroneal nerve branches. If the fracture morphology precluded arthroscopic reduction, open reduction and fixation were then performed. The arthroscope was subsequently used to confirm anatomic reduction of the articular fracture fragments. The joint was again irrigated and portals closed with cutaneous sutures.

Postoperatively, the ankle was routinely immobilized in a posterior splint or cast for 2 weeks. A removable splint was provided subsequently for 2 weeks and directed-active and active-assisted range-of-motion exercises introduced. Weight bearing was not permitted in the initial 4 postoperative weeks. At 1 month, progressive weight bearing to tolerance was initiated with the limb in a short leg cast or brace dependent on fracture stability and patient compliance and continued until satisfactory fracture healing.

Statistical Analysis

Patient demographics, fracture classification, and associated ligamentous injuries were correlated with articular surface damage. Statistical comparisons among noncontinuous parameters were performed using χ-square analysis. Paired comparisons among continuous variables were performed using the Fisher exact test. Significance level (α) was selected as $P = .05$.

RESULTS

Intra-articular pathology identified at arthroscopy included chondromalacia and articular surface lesions of the tibial plafond and talus, free osseous or chondro-osseous bodies, and intra-articular and capsular ligament tears (Table 1). Chondromalacia was identified in 9 of the 48 ankles. Three ankles had grade I and 3 had grade II-III chondromalacia of the talar dome. One ankle had grade I changes on the central tibial plafond; 2 had grade I injury to both articular surfaces. Fracture debris and hemarthrosis were noted in virtually all cases and cleared with lavage and gentle debridement. Free fragments requiring operative extraction were noted in 13 ankles. Traumatic articular surface lesions (TASLs), including chondral defects and osteochondral lesions measuring greater than 5 mm in diameter, were identified in 30 of the 48 ankles (63%), with 11 lesions localized to the tibia and 19 noted on the talus. Excluded were intra-articular extensions of metaphyseal or epiphyseal fractures, such as Tillaux or triplane. Tibial lesions were at the posterior syndesmotic ligament origin in 6 cases, at the anterior capsular insertion in 3 cases, and at the central articular surface in 2 cases. Of the 19 talar lesions, 15 involved the medial dome and 4 involved the lateral articulation. TASLs of the talus in this series were uniformly unstable or displaced and virtually devoid of subchondral bone precluding satisfactory internal fixation (Fig 1). No significant statistical association was identified between individual patient demographic parameters (including age, sex, injury side, or mechanism) and intra-articular pathology. There were no untoward effects of fluid extravasation in any case.

Under the Lauge-Hansen classification, 24 injuries were supination-external rotation (SER), 10 were pronation-external rotation (PER), 5 were supination-adduction (SAD), and 4 were pronation-abduction (PAB). According to the Danis-Weber system, 2 fractures were type A, 24 were type B, and 11 were type C. One triplane and 4 Tillaux fractures were unclassifiable in either scheme along with 6 isolated medial malleolus fractures not classified by Danis-Weber criteria.

The incidence of TASLs of the talus was highest in fractures that disrupted the distal tibiofibular syndesmosis. Of the 10 PER fractures, 7 had unstable or displaced talar TASLs, 5 involving the medial and 2 the lateral articulation. A lateral talar dome TASL was identified in the sole PAB3 fracture in the series (Fig 2A). Thus, of the 1 Danis-Weber C fractures, 8 sustained full-thickness talar articular damage (Fig 2B). Additionally, the only SER or B injury with an unstable syndesmosis requiring fixation had a medial talar TASL. Therefore, 9 of 12 fractures with syndesmosis disruptions sustained a documented talar chondral defect. The statistical association between syndesmosis disruption and a talar articular lesion was highly significant ($P = .01$).

Ten of the 24 SER or Danis-Weber B fractures were found to have talar TASLs, 9 medial and 1 lateral (Fig 2B). One of the 5 SAD and 1 of the 4 PAB injuries had medial and lateral dome TASLs, respectively. No TASLs of the talus were found in the 2 Danis-Weber A, 1 triplane, or 4 Tillaux fractures. Of the 36 ankles with a clinically stable syndesmosis, 10 had talar TASLs; 9 of these fractures were SER injuries.

A medial malleolus fracture or deltoid ligament disruption, irrespective of injury classification, did not appear to predispose or protect from articular chondral injury ($P > .9$). A TASL of the talus was noted in 13 of 31 cases with either fracture of the medial malleolus fracture or disruption of the deltoid ligament and in 6 of 17 injuries with no medial injury. Furthermore, an isolated or combined fracture of the medial malleolus was not in itself associated with a higher incidence of talus lesions ($P > .7$). Only 4 of 12 cases with a fractured medial malleolus had an arthroscopically identified TASL of the talus compared with 15 of 36 injuries with an intact medial malleolus.

Articular surface lesions of the lateral talar dome were relatively more common following pronation than supination injury mechanisms. Lateral talar

Table 1. *Intra-articular Pathology Identified at Arthroscopy*

Case No.	Lauge-Hansen Class*	Danis-Weber Class	Medial Malleolus Fracture	Articular Surface Lesion Talus	Articular Surface Lesion Tibia	Chondromalacia (Grade)	Ligament Tear†	Loose Body	Internal Fixation‡
1	SER	B	no	none	none	Talus (II)	S	none	LM
2	SER	B	no	Lateral	none	none	none	none	LM
3	SER	B	no	none	Posterior	none	none	none	LM
4	SER	B	no	none	Anterior	none	none	none	LM
5	SER	B	no	none	none	none	D	none	LM
6	SER	B	no	Medial	none	none	D	none	LM
7	SER	B	no	Medial	none	none	D	none	LM
8	SER	B	no	none	Posterior	none	S	none	LM
9	SER	B	no	none	none	none	S	yes	LM
10	SER	B	no	Medial	none	Tibia (I)	D/S	none	LM
11	SER	B	no	none	none	none	D/S	none	LM
12	SER	B	no	none	none	Talus (I)	D/S	yes	LM
13	SER	B	no	Medial	none	none	D/S	none	LM/S
14	SER	B	no	none	none	Talus/Tibia (I)	D	none	LM
15	SER	B	no	none	none	none	D	none	LM
16	SER	B	no	Medial	none	none	D	none	LM
17	SER	B	no	none	Anterior	none	D/S	yes	LM
18	SER	B	no	Medial	none	Talus (I)	S	none	LM
19	SER	B	no	none	none	none	D	none	LM
20	SER	B	no	none	none	none	D/S	yes	LM
21	SER	B	no	Medial	none	none	D	yes	LM
22	SER	B	yes	Medial	none	none	none	yes	MM/LM
23	SER	B	yes	Medial	none	none	none	yes	MM/LM
24	SER	B	yes	none	none	none	none	none	MM/LM
25	PER	C	no	none	Posterior	none	S	none	LM/S
26	PER	C	no	Lateral	none	Talus (III)	D/S/ATFL	none	LM/S
27	PER	C	no	Medial	none	none	S	none	LM/S
28	PER	C	no	Medial	none	none	D/S	none	LM/S
29	PER	C	no	none	Posterior	none	D/S	none	S
30	PER	C	no	Medial	none	none	D/S	yes	S
31	PER	C	no	Medial	none	Talus (II)	S	yes	S
32	PER	C	no	Medial	none	none	S	yes	S
33	PER	C	yes	Lateral	none	none	S	none	MM/S
34	PER	C	yes	none	none	Talus/Tibia (I)	S	none	MM/LM/S
35	SAD	NA	yes	Medial	none	none	S	none	MM
36	SAD	A	yes	none	none	none	none	yes	MM
37	SAD	NA	yes	none	Central	none	none	none	MM
38	SAD	NA	yes	none	none	none	none	yes	MM
39	SAD	A	no	none	none	none	D	none	LM
40	PAB	NA	yes	none	Central	none	N	yes	MM
41	PAB	NA	yes	none	Anterior	Talus (I)	ATFL	none	MM
42	PAB	NA	yes	none	none	none	none	none	MM
43	PAB	C	no	Lateral	none	none	S	none	LM/S
44	TILLAUX	NA	no	none	none	none	none	none	Ant Tubercle
45	TILLAUX	NA	no	none	none	none	none	none	Ant Tubercle
46	TRIPLANE	NA	no	none	none	none	none	none	Distal Tibia

*SER, supination-external rotation; PER, pronation-external rotation; SAD, supination-abduction; PAB, pronation-abduction.

†S, syndesmotic ligaments (including anterior and posterior inferior tibiofibular ligaments); D, deltoid ligament; ATFL, anterior talofibular ligament.

‡LM, lateral malleolus plate osteosynthesis; S, distal tiobiofibular syndesmosis screw fixation; MM, medial malleolus screw fixation.

TASLs were identified in 3 of 14 Lauge-Hansen pronation fractures. In contrast, only 1 such lesion was noted in 29 supination injuries. This association approached yet did not attain statistical significance ($P = .09$).

TASLs of the tibia were identified in the central

Image available only in print

FIGURE 1. Medial dome TASL of the talus in a Lauge-Hansen SER or Danis-Weber B fracture (case 10). (A) Preoperative anteroposterior radiograph and (B) arthroscopic appearance of a right ankle viewed anteromedially (OLT, osteochondral lesion; Ta, talus). (C) Simulated mechanism. Note that the chondral fragment with minimal subchondral bone was sheared from the talar surface, creating a full-thickness articular defect on the medial dome. An osseous defect was not apparent on roentgenographic views. (Reprinted with permission.[11])

articular surface in only 2 cases: 1 SAD and 1 PAB injury. Peripheral tibial TASLs were essentially incomplete osteochondral avulsions of the posterior inferior tibiofibular ligament origin in 6 cases and the anterior capsule in 3 cases. All 6 posterolateral tibial TASLs were identified in external rotation injuries (SER, PER, or Tillaux). Concomitant syndesmosis ligament tearing was identified arthroscopically in 3 of the 4 adult posterolateral tibial articular lesions (Table 1). However, an incompetent

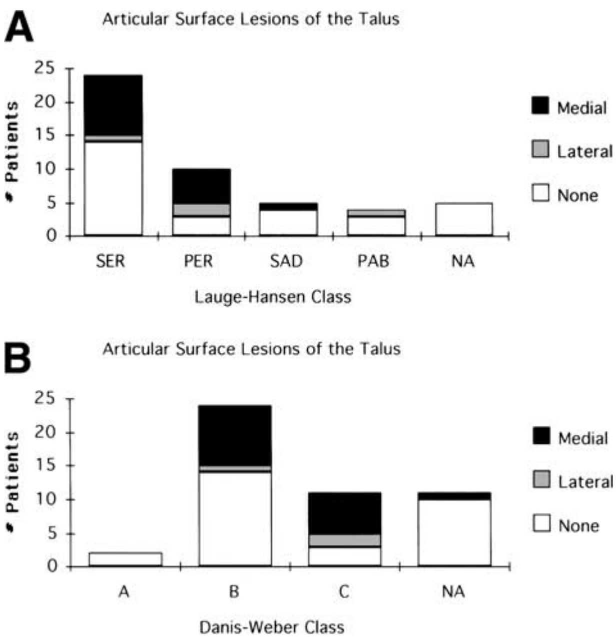

FIGURE 2. Incidence of TASLs of the talus categorized by fracture classification. (A) Lauge-Hansen and (B) Danis-Weber. Note particularly the significantly increased incidence of chondral damage in injuries defined by syndesmosis disruption: Lauge-Hansen PER or Danis-Weber C ($P < .05$).

syndesmosis complex was not significantly related to tibial articular damage ($P > .9$).

Visualized intra-articular ligament injuries were in general consistent with the fracture mechanism as described by Lauge-Hansen. Of the 24 SER fractures, 10 had sustained at least partial interstitial disruption or incomplete articular avulsion of the syndesmotic ligaments with damage visualized in both the anterior and posterior inferior tibiofibular ligaments (Fig 3). Only 1 SER injury had an unstable syndesmosis necessitating fixation. In all PER fractures and the sole PAB3 injury, a disrupted and unstable syndesmosis ligament complex was documented arthroscopically and clinically with the application of an external rotation stress at the foot. In only 20% of cases could the abnormal pathology seen at arthroscopy be identified at the time of ORIF.

DISCUSSION

Previously, the extent and type of intra-articular damage in an acute fracture of the ankle was unclear. This is one of the first prospective studies to show that ankle fractures have a high incidence of concomitant intra-articular pathology. Traumatic articular surface lesions of the talar dome were noted in 10 of 24 Lauge-Hansen SER or Danis-Weber B injuries in this series. Moreover, in Danis-Weber C fractures, articular lesions of the talus were noted in 8 of 11 cases. Overall, 9 of 12 ankle fractures that disrupted the integrity of the distal tibiofibular syndesmosis sustained a full-thickness articular defect of the talus. Concomitant medial malleolus fractures or deltoid ligament tears did not correlate statistically with the occurrence of such articular lesions. Talar dome lesions were medial in 15 and lateral in 4 cases. Pronation injuries were associated with a 6-fold higher risk of a lateral dome talar injury compared with supination injuries. This trend failed to attain statistical significance, likely reflecting the relatively few lateral talus lesions observed.

The precise mechanism producing TASLs of the talus is not conclusively known. Based on our observations, more than 1 mechanism may be associated with similar appearing lesions. Berndt and Hardy[22] proposed torsional impaction as the principal force. These investigators reasoned that, with the tibia internally rotated, forced dorsiflexion and inversion impacted and compressed the lateral talar margin against the medial articular surface of the fibula, resulting in a shear force potentially displacing a lateral osteochondral fragment. Conversely, with the tibia in external rotation, plantarflexion and inversion impacted the medial articulation of the talus against the posteromedial tibia, causing a shear force at the medial dome.

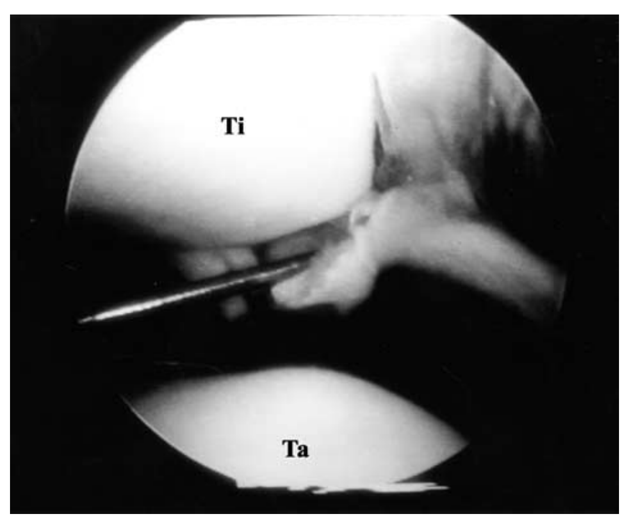

FIGURE 3. Torn posterior inferior tibiofibular ligament in Lauge-Hansen SER or Danis-Weber B fracture (case 20) left ankle viewed anterolaterally. Hemorrhage was apparent within the interosseous membrane. The syndesmosis was clinically stable, requiring no fixation (Ti, tibia; Ta, talus).

Yao and Weis[23] suggested that lateral lesions were caused by eversion with the ankle dorsiflexed and the tibia internally rotated on the talus; medial lesions were, then, produced by inversion with the ankle plantarflexed.

Our data regarding the distribution of TASLs of the talus with regard to injury mechanism are not entirely concordant with those presented by Berndt and Hardy or Yao and Weis. Clearly, no single mechanism can explain each case. Unfortunately, neither fracture classification used accurately accounts for the relative tibiotalar flexion at the time of injury. We found a preponderance of medial talar dome TASLs regardless of fracture mechanism. Pronation mechanisms were associated with an increased incidence of lateral lesions. Perhaps pronating the foot loads the lateral talar dome against the lateral tibial plafond and medial fibula, predisposing this site to initial compression and shear forces producing an articular surface injury (Fig 4). Interestingly, unstable syndesmosis injuries were associated with a significantly high risk of articular surface injury to the talar dome. In this subgroup of patients, it is impossible to discern if the TASL was produced instantaneously before or after the syndesmosis was disrupted. Primary or coupled rotational and translational forces must be considered.

An external force imparted to the ankle joint creates a shear stress on the articular surface. A chondral or osteochondral fracture may occur along this vector. The extent of injury depends on both the magnitude and direction of shear. If the shear stress is greater than the ultimate shear strength of bone but less than that of articular cartilage, the chondral surface momentarily deforms but remains intact as the underlying bone fractures. However, if the shear stress is greater than the ultimate shear strength of both bone and articular cartilage, a complete and potentially displaced osteochondral lesion occurs.[24] A shear stress with a relatively large horizontal component and small vertical component predominantly parallels the ankle joint surfaces. If this stress exceeds the ultimate shear stress of articular cartilage, an isolated chondral injury may be created. In no case in our series did preoperative ra-

A

Image available in print only

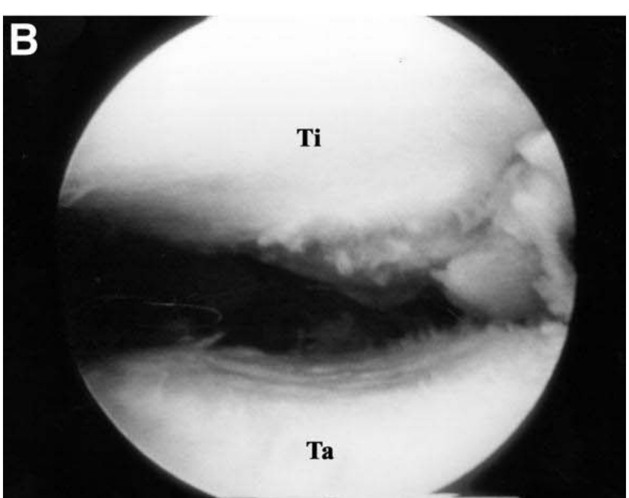

Figure 4. Lateral dome TASL of the talus in a Lauge-Hansen PER or Danis-Weber C fracture (case 26). (A) Proposed mechanism (modified and reprinted with permission[11]). (B) Arthroscopic photograph of a right ankle viewed anteromedially. Note the initial localized compression and rotation moments and resultant shear force causing the articular surface injury to the talus. Subchondral bone was exposed over a considerable area of the lateral articulation (Ta, talus; Ti, tibia).

diographs suggest an osteochondral fracture. Arthroscopic findings showed that most articular lesions were virtually devoid of subchondral bone. The surface injuries identified represented considerable chondral delamination alone or beyond the circumference of the osseous injury. This is consistent with an extensive horizontal shear component, acting primarily in parallel to the talar articular surface (Fig 1). Unfortunately, salvage of such fragments by satisfactory internal fixation is not currently feasible.

Tibial TASLs were noted in 11 of the 48 cases. Only 2 acute ankle fractures had central tibial articular lesions, occurring each in adduction and abduction injuries. No central lesion was identified in an external rotation mechanism, possibly reflecting a relatively greater degree of axial loading at the central plafond in coronal plane injuries. The remaining tibial TASLs represented incomplete articular avulsions of the anterior capsule or posterior inferior tibiofibular ligament. All 6 posterolateral tibial TASLs were identified in external rotation injuries (SER, PER, or Tillaux). A concomitant intrasubstance syndesmosis ligament injury was identified arthroscopically in 3 of the 4 adult posterolateral margin TASLs. This latter finding correlates the observations of Ogilvie-Harris and Reed[25] who noted similar pathologic features in patients with chronic isolated syndesmosis injuries, including tearing of the posterior inferior tibiofibular ligament and chondral fracture of the posterolateral portion of the tibial plafond. An external rotation mechanism is most consistent with this pattern of injury.

The purpose of our study was to document arthroscopically the incidence and location of occult intra-articular injuries associated with acute ankle fractures. Although few authors have addressed this subject, Feldman[26] recently reported a high incidence of articular surface pathology in such injuries. Of 15 extra-articular ankle fractures in his series, 14 were categorized as SER injuries and 1 classified as a PER injury. Articular damage at arthroscopy was noted in 11 patients. Chondral fractures of the talar dome were found in 7 patients and osteochondral lesions of the tibia were identified in 4. Six patients had combined articular injuries.

Without extended clinical follow-up, it is not possible to determine if early arthroscopic intervention will minimize poor outcomes following ankle fractures. Ideally, a prospective randomized study could answer this. The concept, however, is intuitively appealing. The prognostic importance of traumatic articular lesions is unclear, although studies suggest such injuries may be a source of refractory functional deficits.[7,8] Optimal outcome of operatively treated ankle fractures depends on an anatomic reduction. However, an unrecognized concomitant chondral damage may adversely influence the clinical result. Lantz et al.[8] noted a 49% incidence of injury to the talar dome articular cartilage in isolated malleolar fractures. Despite anatomic reduction and internal fixation, patients with talar dome chondral injuries overall had significantly diminished joint motion and function. Taga et al.[7] found chondral lesions in 89% of acute lateral ankle ligament injuries examined arthroscopically. Moreover, on follow-up evaluation of chronic lateral ligament tears, 50% with chondral lesions greater than half the thickness of the articular cartilage complained of persistent pain.

Complete or partial tears of the anterior and posterior inferior tibiofibular ligaments were noted arthroscopically in 22 of 48 cases, although only 12 of these injuries were clinically unstable requiring syndesmosis fixation. Previous reports have documented the occurrence of syndesmotic pain[25] and, in particular, anterolateral impingement following ankle injury.[27-31] Surgical exploration has identified hyalinized connective tissue extending from the anterior inferior tibiofibular ligament, entrapped in the distal tibiofibular and talofibular articulations.[28,31] Posteriorly, fibrosis and damage to the posterior inferior tibiofibular ligament, chondral fracture of the posterolateral plafond, and disruption of the interosseous ligament have been described.[25] Given the marked incidence of arthroscopically visible injury to the syndesmotic ligaments, this damage and subsequent fibrosis and inflammation may account for such post-traumatic impingement syndromes. In fact, the frequency of injury to the anterior inferior tibiofibular ligament may be underestimated. Only 20% of the ligament is intra-articular.[20] Consequently, injury to the extra-articular portion may remain occult and predispose to later symptomatology. Again, the effect of early debridement is not yet known. Resection of intra-articular pathology in the late convalescent period, however, has proved substantially beneficial.[25,27-30]

Interstitial tears of the deltoid ligament were identified arthroscopically in 19 adult injuries without a medial malleolus fracture. Ruptured ligament fibers were debrided from the medial gutter before open fixation of the fibula. At times, an interposed deltoid ligament may impede reduction of the fibula or talus, requiring a medial arthrotomy. Although uncommon, the incidence of this circumstance has been reported. Routine arthroscopic evaluation and debridement of the medial gutter may preclude the need for an open medial approach to address interposed tissue. No patient in our study required a medial arthrotomy following intra-articular debridement in the setting of a deltoid ligament rupture. Feldman,[26] moreover, reported the deltoid ligament infolded into the medial gutter in 3 of 15 patients with extra-articular ankle fractures evaluated arthroscopically. When present, the deltoid ligament lesion was debrided to allow anatomical reduction.

Given the high incidence of unstable articular surface lesions, loose chondro-osseous bodies, and intra-articular ligament tears, early arthroscopic interventions may optimize healing and function. Our recommendations for arthroscopic examination of acute ankle fractures include all intra-articular and extra-articular ankle injuries where the occurrence of articular damage is probable. We have shown that the likelihood of chondral lesions is highest in injuries with an unstable distal tibiofibular syndesmosis, but it is not infrequent in other fracture types. Certainly, ankle arthroscopy is indicated in the context of a known displaced osteochondral lesion.

Advantages of arthroscopic evaluation of acute ankle fractures include minimal surgical trauma and excellent visualization of the joint surfaces for assessing and treating the articular surface damage. The procedure also permits evaluation and debridement of ligament tears and removal of loose debris that may cause third body articular damage. Furthermore, arthroscopic visualization may facilitate reduction and internal fixation in certain fracture patterns.[10-14] Absolute contraindications include open fractures, neurovascular injury, and severe swelling. Arthroscopy should be performed with caution in the acutely fractured ankle to avoid excessive fluid extravasation and compartment ischemia. In our experience, extravasation has not been a problem when adequate inflow and outflow are maintained. Only extended follow-up evaluation can determine if early arthroscopic intervention will improve clinical outcome. Nevertheless, arthroscopy is a valuable tool in identifying and treating intra-articular damage that would otherwise remain unrecognized and may provide prognostic information regarding the functional result of these injuries. The surgeon should have adept skills for routine ankle arthroscopy before implementing these techniques in fracture management.

REFERENCES

1. Ali MS, McLaren CA, Rouholamin E, O'Connor BT. Ankle fractures in the elderly: Nonoperative or operative treatment. *J Orthop Trauma* 1987;1:275-280.
2. Hughes JL, Weber H, Willenegger H, Kuner EH. Evaluation of ankle fractures: Non-operative and operative treatment. *Clin Orthop* 1979;138:111-119.
3. Yde J, Krisensen KD. Ankle Fractures. Supination-eversion fractures of stage IV. Primary and late results of operative and non-operative treatment. *Acta Orthop Scand* 1980;51:981-990.
4. Anderson IF, Crichton KJ, Grattan-Smith T, Cooper RA, Brazier D. Osteochondral fractures of the dome of the talus. *J Bone Joint Surg Am* 1989;71:1143-1152.
5. Renström PAFH. Persistently painful sprained ankle. *J Am Acad Orthop Surg* 1994;2:270-280.
6. Stone JW. Osteochondral lesions of the talar dome. *J Am Acad Orthop Surg* 1996;4:63-73.
7. Taga I, Shino K, Inoue M, Nakata K, Maeda A. Articular cartilage lesions in ankles with lateral ligament injury. An arthroscopic study. *Am J Sports Med* 1993;21:120-126.
8. Lantz BA, McAndrew M, Scioli M, Fitzrandolph RL. The effect of concomitant chondral injuries accompanying operatively reduced malleolar fractures. *J Orthop Trauma* 1991;5: 125-128.
9. Ferkel RD, Scranton PE Jr. Current concepts review: Arthroscopy of the ankle and foot. *J Bone Joint Surg Am* 1993;75: 1233-1242.
10. Ferkel RD, Orwin JF. Ankle arthroscopy: A new tool for treating acute and chronic ankle fractures. *Arthroscopy* 1993; 9:352-353.
11. Ferkel RD, Orwin JF. Arthroscopic treatment of acute ankle fractures and postfracture defects. In: Ferkel RD, ed. *Arthroscopic surgery: The foot and ankle*. Philadelphia: Lippincott-Raven, 1996;185-200.
12. Holt ES. Arthroscopic visualization of the tibial plafond during posterior malleolar- fracture fixation. *Foot Ankle* 1994;15:206-208.
13. Miller MD. Arthroscopically assisted reduction and fixation of an adult Tillaux fracture of the ankle. *Arthroscopy* 1997;13: 117-119.
14. Whipple TL, Martin DR, McIntyre LF, Meyers JF. Arthroscopic treatment of triplane fractures of the ankle. *Arthroscopy* 1993;9:456-463.
15. Lauge-Hansen N. "Ligamentous" ankle fractures: Diagnosis and treatment. *Acta Chir Scand* 1949;97:544-550.
16. Lauge-Hansen N. Fractures of the ankle. III. Genetic roentgenologic diagnosis of fractures of the ankle. *AJR Am J Roentgenol* 1954;71:456-471.
17. Danis R. *Theorie et pratique de l'osto-synthse*. Paris: Masson, 1947.
18. Weber BG. *Die Verletzungen des oberen Sprunggelenkes. Aktuelle Probleme in der Chirugie.* Ed 2. Bern: Verlag Hans Huber, 1972.
19. Stetson WB, Ferkel RD. Ankle arthroscopy: I. Technique and complications. *J Am Acad Orthop Surg* 1996;4:17-23.
20. Ferkel RD. Diagnostic arthroscopic examination. In: Ferkel RD, ed. *Arthroscopic surgery: The foot and ankle*. Philadelphia: Lippincott-Raven, 1996;103-118.
21. Outerbridge RE. The etiology of chondromalacia patellae. *J Bone Joint Surg Br* 1961;43:752-757.
22. Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg Am* 1959;41:988-1020.
23. Yao I, Weis E. Osteochondritis dissecans. *Orthop Rev* 1985; 14:190-194.
24. Stauffer RN. lntraarticular ankle problems. In: Evarts CM, ed.

Surgery of the musculoskeletal system. Vol 3. New York: Churchill Livingstone, 1983.

25. Ogilvie-Harris DJ, Reed SC. Disruption of the ankle syndesmosis: Diagnosis and treatment by arthroscopic surgery. *Arthroscopy* 1994;10:561-568.
26. Feldman MD. Evaluation of intra-articular damage in displaced extra-articular ankle fractures. Presented at the Annual Meeting of the Eastern Orthopaedic Association, Scottsdale, Arizona, 1997.
27. DeBerardino TM, Arciero RA, Taylor DC. Arthroscopic treatment of soft-tissue impingement of the ankle in athletes. *Arthroscopy* 1997;13:492-498.
28. Ferkel RD, Karzel RP, Del Pizzo W, Friedman MJ, Fischer SP. Arthroscopic treatment of anterolateral impingement of the ankle. *Am J Sports Med* 1991;19:440-446.
29. Meislin RJ, Rose DJ, Parisien JS, Springer S. Arthroscopic treatment of synovial impingement of the ankle. *Am J Sports Med* 1993;21:186-189.
30. Stetson WB, Ferkel RD. Ankle arthroscopy: II. Indications and risks. *J Am Acad Orthop Surg* 1996;4:24-31.
31. Wolin I, Glassman F, Sideman S. Internal derangement of the talofibular component of the ankle. *Surg Gynecol Obstet* 1950; 91:193.