CHAPTER 9

TALAR FRACTURES REVISITED

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Talar fractures have posed a challenge to physicians for years. From osteochondral talar dome fractures to open, dislocated talar neck fractures, each type has its own associated morbidity. Before one can approach the treatment of talar fractures, it is important to have an understanding of the anatomy of the talus, its vascular supply, and the different types of talar fractures.

ANATOMY OF THE TALUS

The talus is composed of three parts: body, neck, and head. The superior surface of the body is referred to as the trochlea or talar dome which articulates with the tibial plafond to form the ankle joint. The dome continues medially to articulate with the medial malleolus, and laterally to articulate with the lateral malleolus. Ligamentous structures inserting medially include the portions of the deltoid ligament (anterior and posterior talotibial) and laterally the anterior and posterior talofibular ligaments. The posterior talar process projects off of the body, and the flexor hallucis longus tendon courses through a groove in the middle of the posterior aspect of the process. The lateral talar process is located on the inferior portion of the lateral aspect of the body. Articulations include the fibula, superiorly and laterally, and the posterior facet of the calcaneus inferiorly. The lateral talocalcaneal ligament inserts into the apex and the anterior and posterior talofibular ligaments insert superior to the apex of the lateral process.

The inferior surface of the body has three articular facets, (anterior, middle, and posterior), that articulate with the calcaneus, forming the subtalar joint. The neck projects from the body anteriorly. The ankle joint capsule inserts on the superior surface of the neck. The inferior surface of the neck forms the tarsal canal with the calcaneus. The interosseous talocalcaneal ligament inserts here. The talar head articulates with the navicular forming the talonavicular joint. The entire talus has no muscular origins or insertions.

VASCULAR SUPPLY OF THE TALUS

Mulfinger and Trueta described the vascularity of the talus consisting of extraosseous and intraosseous supplies (Fig. 1). Extraosseous arteries are branches of the three major arteries which supply the talus: posterior tibial artery, dorsalis pedis artery, and peroneal artery. The posterior tibial artery provides branches to the posterior process (calcaneal branches), the body (artery of the tarsal canal), and the medial surface of the body (deltoid branch). Branches of the dorsalis pedis artery supply the superior surface of the neck (medial tarsal artery or anterior medial malleolar artery), the neck and body (artery of the tarsal sinus), and the head (lateral tarsal artery). An anastomotic network exists in the sinus tarsi formed by the lateral tarsal (dorsalis pedis), perforating peroneal (peroneal), and anterior lateral malleolar (anterior tibial) branches. The branches of the peroneal artery supply the posterior process and the tarsal canal.

The intraosseous arterial patterns are divided into head and body. The head is supplied by two sources: the branches from the dorsalis pedis or anterior tibial artery supply the medial superior half, and the sinus tarsi anastomosis or lateral tarsal artery supply the inferior and lateral half. The body

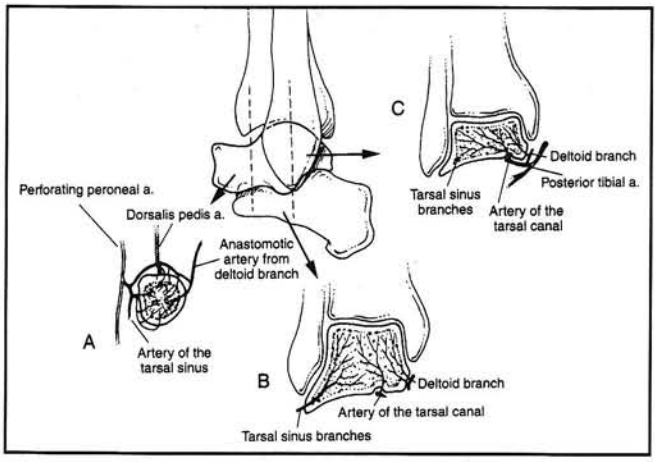

Figure 1. Blood supply of the talus.

is mostly supplied by the artery in the tarsal canal which gives four to five main branches into the body curving posterolaterally, supplying almost all of the middle and lateral thirds of the body and a portion of the lateral trochlear surface. The deltoid branches enter the medial periosteal surface of the body and supply the medial third of the body. The sinus tarsi anastomosis sends branches that enter the lateral anterior surface and supply the lateral inferior aspect of the body including most of the posterior facet. The anastomosis also sends arteries which enter the superior surface of the neck and supply the middle of the anterior superior aspect of the trochlea. Several small branches from the posterior periosteal network enter and supply the posterior tubercle. The multitude of anastomoses between the three major arterial supplies make it difficult for avascular necrosis to occur.

TYPES OF TALAR FRACTURES

Fractures of the Talar Neck

The second most common type of talar fractures are talar neck fractures. This fracture was coined "aviator's astralgus" by Anderson in 1919. The mechanism of injury involves hyperdorsiflexion of the foot with impingement of the talar neck against the anterior edge of the tibial plafond. In 1970, talar fractures were classified by Hawkins based on associated dislocation of the proximal fragment (Fig. 2).

With luxation of each joint, more vascular supply to the talus is disrupted, increasing the potential for developing avascular necrosis. The three groups include: Group I - a vertical neck fracture without displacement, Group II - a vertical neck fracture with dislocation of the talar body from the subtalar joint, and Group III - a vertical neck fracture with dislocation of the talar body from the subtalar joint and ankle joint. Canale and Kelly proposed a fourth group to this classification that includes vertical neck fractures with dislocation of the talar body from the subtalar joint and ankle joint, and dislocation of the talar head from the talonavicular joint. Their reported rates of avascular necrosis are shown in Table 1.

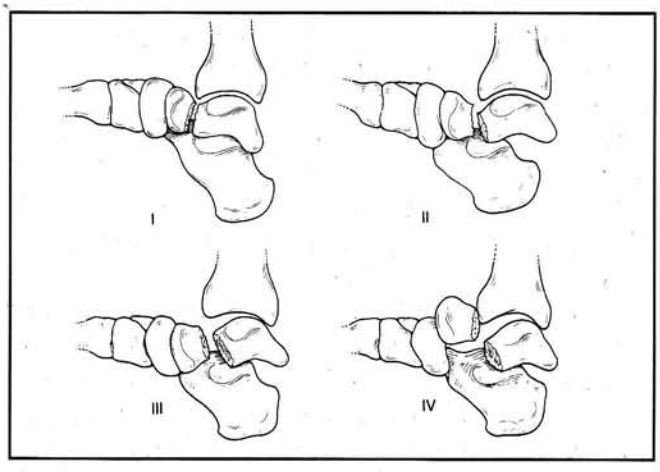

Figure 2. Hawkins classification of talar neck fractures: modified by Canale & Kelly in 1978.

Table 1

RATES OF AVASCULAR NECROSIS IN TALAR NECK FRACTURES

	HAWKINS	CANALE & KELLY
Group I	0%	13%
Group II	42%	50%
Group III	91%	84%
Group IV	-	*50%

*Only 3 cases reported. One treated with total talectomy. One of the remaining two resulted in AVN. Need more cases to accurately assess true rate of occurrence.

Group I talar neck fractures should be treated with application of a non-weight bearing, below-knee cast for 6 to 12 weeks with additional non-weight bearing range of motion exercises for 2 to 5 months. Group II talar neck fractures should be treated by an attempt at closed reduction. If successfully reduced, the foot should be casted non-weight bearing until evidence of an osseous union is identified. If closed reduction is unsuccessful, open reduction with internal fixation via compression screws should be employed. Some authors have postulated that this method re-establishes the vascular supply to the body of the talus across the actual fracture line. Group III & IV fractures show marked improvement when treated with anatomic reduction and rigid internal fixation.

The prognostic indicator for re-establishment of vascular supply to the talar body is subchondral atrophy seen in the talar dome on the anterior-posterior view of the ankle around 6 to 8 weeks after injury. The blood supply washes out the subchondral bone creating a disuse osteopenia. This is referred to as a positive "Hawkins sign."

Fractures of the Body of the Talus

Talar body fractures include three types: compression, crush, and shearing fractures (Fig. 3). Compression and crush fractures generally end in poor results because of the loss of bone substance and disruption of the articular surface created by the injury. These can be treated with open reduction and bone grafting, to augment bone loss and restore height of the talus, and fixated internally or externally. Despite acute surgical treatment, these injuries usually require subtalar or ankle arthrodesis at a later date.

Shearing type fractures are more successfully treated with open reduction and internal fixation due to the presence of a definite fracture line and larger fragments. End results depend on restoration of vascularity to the talar body.

Fractures of the Lateral Process

Lateral process fractures are relatively uncommon, however, this fracture is one of the most commonly misdiagnosed fractures in emergency rooms. The most popular theory for mechanism of injury is dorsiflexion and inversion as described by Hawkins. Ruch proposed a mechanism involving an eversion and external rotation forces on a dorsiflexed foot. Following disruption of the deltoid ligament or avulsion of the medial malleolus, the lateral aspect of the posterior facet impacts against the lateral talar process, and potentially the lateral talar process against the fibula. Early treatment is aimed at excision of small fragments and rigid internal compression fixation of large fragments. Late treatment may consist of arthroplasty or arthrodesis.

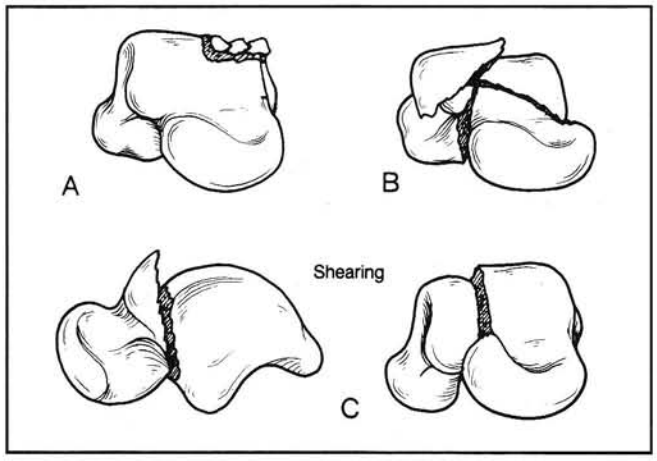

Figure 3. Types of talar body fractures. A. Compression, B. Crush, and C. Shearing.

Fractures of the Posterior Process

Posterior process fracture or "Shepherd's Fracture" is similar to chronic injury to the synchondrosis of an os trigonum. Both result in posterior ankle pain. Both can appear identical on conventional radiographs and tomograms. Most often, patients with posterior process fractures relate a history of ankle trauma. Range of motion of the first metatarsophalangeal joint results in pain in the posterior ankle due to the flexor hallucis longus tendon coursing through the groove in the posterior process.

Mechanisms of injury include forced plantarflexion causing direct impingement of the posterior margin of the tibia on the posterior process, direct trauma to the area, excessive dorsiflexion causing increased tension on the posterior talotibial ligament (medial tubercle) or posterior talofibular ligament (lateral tubercle), and repetitive microtrauma secondary to excessive subtalar joint pronation.

Treatment includes non-weight bearing, below-knee casting for 6 to 8 weeks, local/steroid injections, and NSAIDs. If symptoms persist, excision of fragments results in improvement of symptoms. If the fragment is large enough, it may be corrected acutely with open reduction and internal fixation.

Osteochondral Fractures of the Talar Dome

Talar dome lesions were described and classified by Berndt and Harty. They described medial lesions that were noted on the posterior third of the talar curve caused by inversion and plantarflexion of the foot with external rotation of the tibia on the talus. Lateral lesions occurred at the middle third of the talar curve and were caused by inversion and dorsiflexion of the foot in the ankle. The lesions were staged as follows: Stage I - compression of subchondral bone with intact cartilage, Stage II - partially detached osteochondral fragment, Stage III - completely detached osteochondral fragment that remains in the defect, and Stage IV - displaced osteochondral fragment (Fig. 4). Plain films, CT, MRI, and arthroscopy can assist in the diagnosis and staging of talar dome lesions.

Stage I and II lesions are treated with cast immobilization for 4 to 12 weeks. Medial Stage III lesions should be treated with cast immobilization as well. If symptoms persist after conservative care, surgical excision and curettage is recommended. Lateral Stage III and all Stage IV lesions should

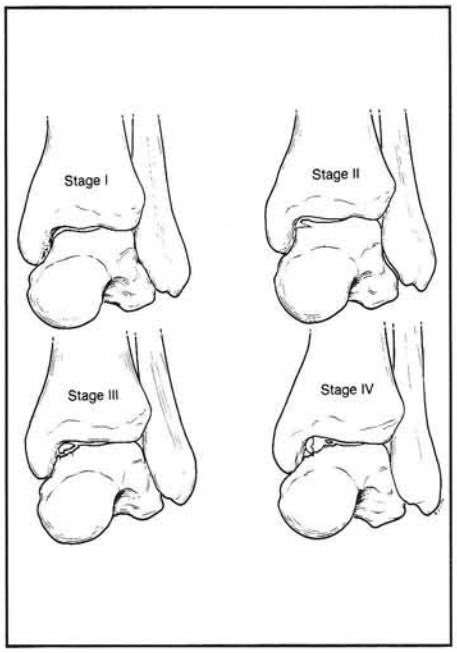

Figure 4. Osteochondral fractures of the talar dome Berndt & Harty classification.

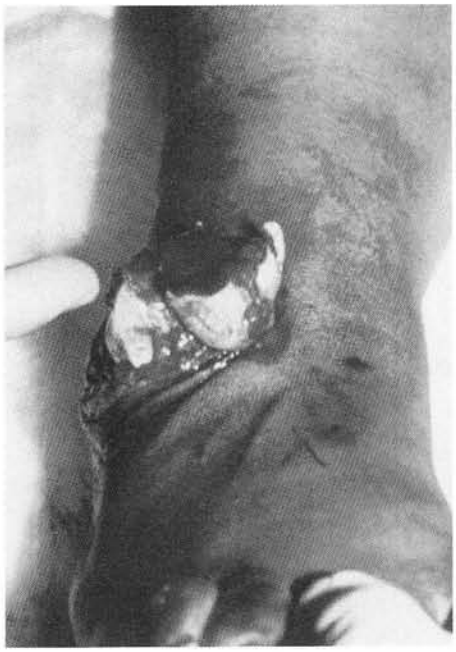

Figure 5A. Clinical appearance on presentation to emergency room.

undergo early surgical intervention. Larger fragments can be fixed with a small compression screw. The head of the screw should be recessed into subchondral bone, well below the articular cartilage surface. Following excision of fragments, the exposed subchondral bone should be drilled with multiple small drill holes to promote migration of fibroblasts to the surface for production of fibrocartilage. Talar dome lesions may be approached either open or arthroscopically.

Chip and Avulsion Fractures

Chip and avulsion fractures are by far the most common of all talar fractures. They result from avulsion of ligaments attaching to the talus. These fractures are generally successfully treated with cast immobilization for 4 to 6 weeks. Persistent symptoms may warrant excision, although this rarely is the case.

CASE STUDIES

Case 1

A 23-year-old woman sustained an open injury to her right foot secondary to a head-on motor vehicle accident. She was brought into the emergency room in stable condition. The only other injuries sustained were lacerations to both hands. The patient's neurovascular status was intact to the level of the digits on the right foot. An open laceration was noted on the anterior lateral aspect of the ankle, with bone exposed (Fig. 5A).

Plain films revealed a transverse fracture through the body of the talus, with dislocation of the body from the subtalar joint and ankle joint, and dislocation of the head from the talonavicular joint (Figs. 5B, 5C). The anterior fragment was displaced anteriorly, superiorly, and laterally. The patient was taken to surgery. After copious lavage, ORIF of the fracture with two 4.0mm partially-threaded cancellous screws was performed using fluoroscopy. A midsubstance rupture of the anterior talofibular and calcaneofibular ligaments was repaired with 2-0 Dexon suture. A partially ruptured peroneus brevis tendon was repaired using 2-0 Dexon suture. Tissue layers were closed anatomically, and a sterile dressing was applied, followed by application of a Jones compression cast. Cultures taken intra-operatively after irrigation were negative for bacterial growth. The patient was kept on cefazolin and gentamicin for 3 days and was discharged from the hospital at 4 days postoperative (Figs. 5D, 5E).

The patient remained non-weight bearing with crutches for 4 1/2 months, and used a bone stimulator for 8 months (Fig. 5F). An arteriogram was performed at 4 months postoperative, and revealed no supply from the peroneal artery or portion of the anterior tibial artery with the

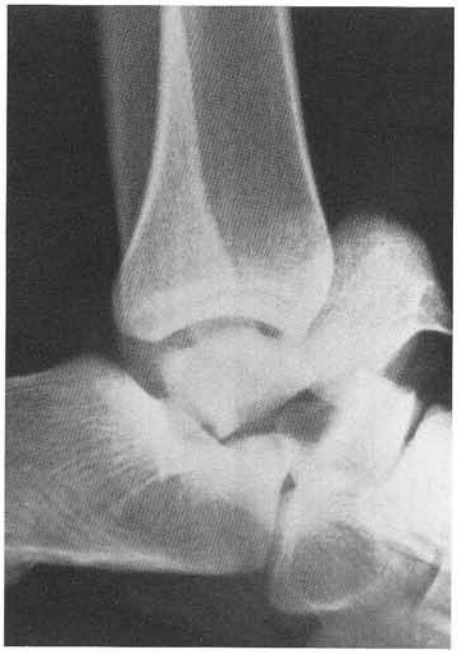

Figure 5B. Pre-reduction AP radiograph.

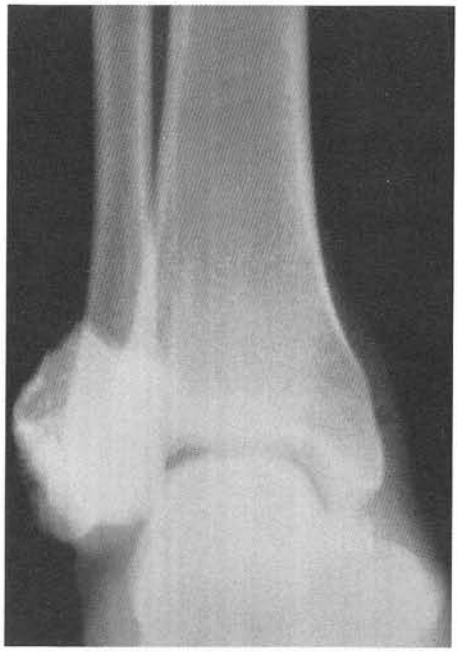

Figure 5C. Pre-reduction lateral radiograph

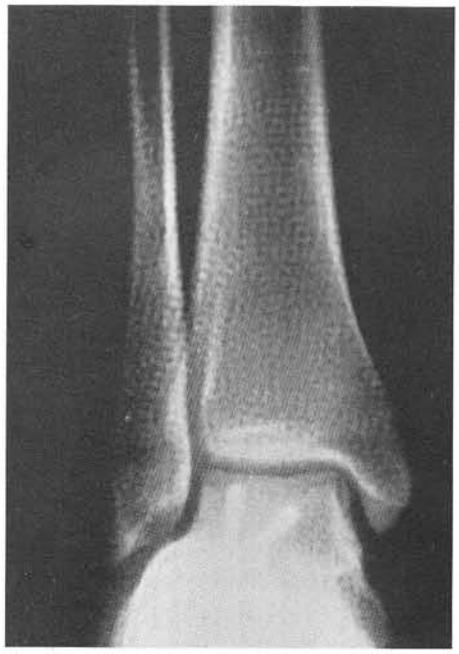

Figure 5D. Immediate postoperative AP radiograph.

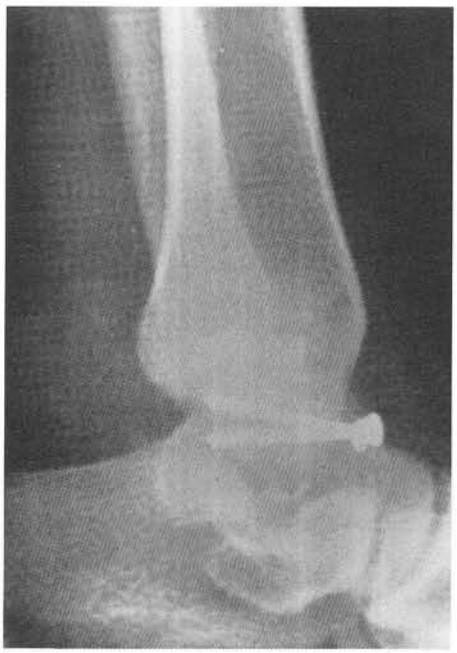

Figure 5E. Immediate postoperative lateral radiograph.

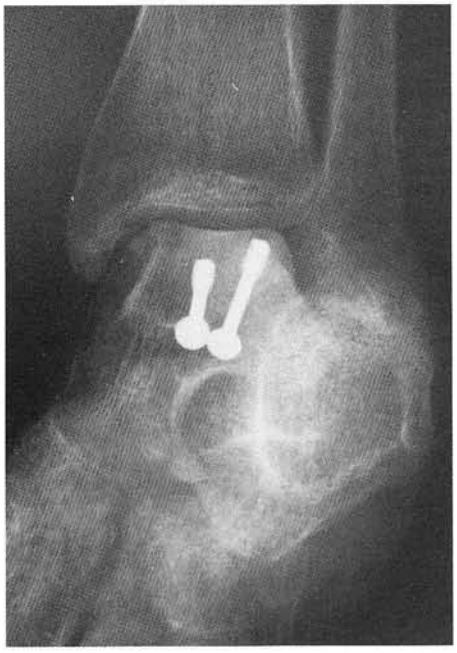

Figure 5F. Eight weeks postoperative AP radiograph. Note the "washed out" appearance of the subchondral bone.

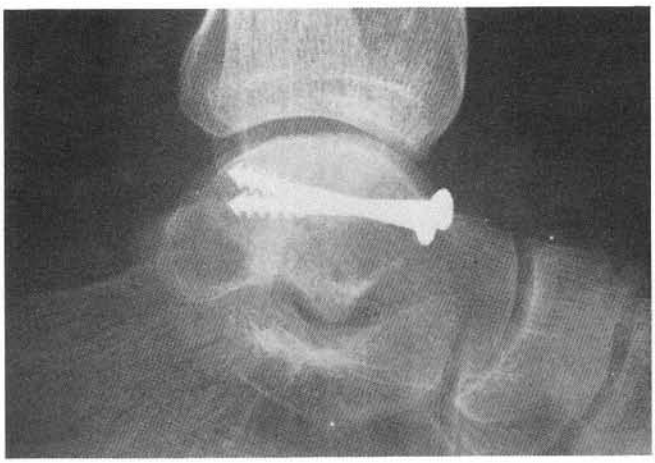

Figure 5G. Lateral radiograph at eighteen months postoperative.

remaining vasculature to the talus showing patency with circulation going into the talus. Physical therapy was started 4 months postoperative. The patient resumed-partial weight bearing in a Cam walker for an additional 3 1/2 months. At eight months postoperative, the patient was placed in a high-top shoe and allowed full weight bearing. The patient was released at eighteen months (Fig. 5G) postoperative when radiographic evidence of complete healing of the fracture was apparent, and range of motion of the ankle was five degrees with knee extended, 10 degrees with knee flexed on the right and two degrees with knee extended, and fifteen degrees with knee flexed on the left. Subtalar joint range of motion was ten degrees of pronation and thirty-four degrees of supination on the right, and twenty degrees of pronation and thirty-seven degrees of supination on the left. The patient's only complaints were being unable to wear high heels, and being unable to stand up on one forefoot.

Case 2

A 24-year-old woman suffered an injury to her right ankle at age 14 while playing basketball. She complained of pain at the anterior and anterolateral aspect of the ankle, aggravated by heels, excessive walking, and running. She also related a "catching sensation" in the ankle joint. Plain films and MRI revealed a medial Stage 3 osteochondral fracture of the talus (Figs. 6A, 6B). The patient wore an ankle brace when running, wore inserts in all shoes, and took NSAIDs. Conservative therapy failed to relieve her symptoms and she underwent surgery.

A tibial malleolar osteotomy was made to allow for exposure of the posteromedial lesion. The fragment measured 5 mm x 7 mm. After excision of the fragment, the fibrous tissue was removed from the subchondral plate. Fenestration of the subchondral bone was performed with a 0.035 Kirschner-wire. The tibial osteotomy was fixated with two 4.0 mm partially-threaded cancellous screws, and tissues were closed in anatomic layers. The patient was placed in a sterile dressing and a non-weight-bearing Jones compression cast.

The patient was taken out of the cast on the fifth day postoperatively and started on a continuous passive motion device. The patient was kept non-weight bearing for 4 weeks, then minimal weight bearing in a Cam walker for 2 weeks. At 6 weeks postoperative, the patient was allowed full weight bearing in an air cast, then gradual return to activities at 8 weeks. At 5 months, the patient was running 3 miles a day with full, pain-free range of motion (Figs. 6C, 6D).

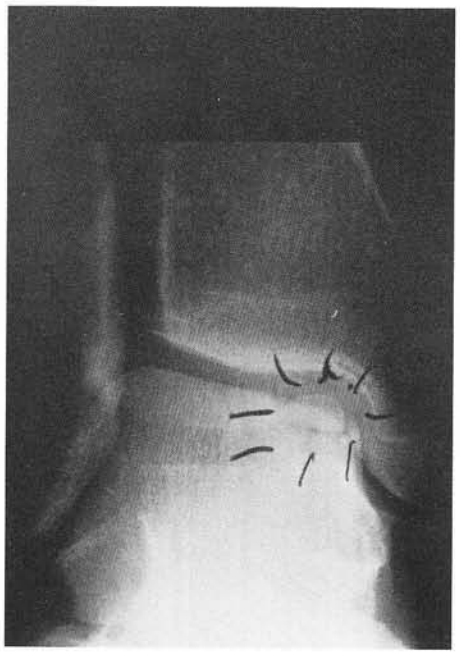

Figure 6A. Preoperative AP radiograph

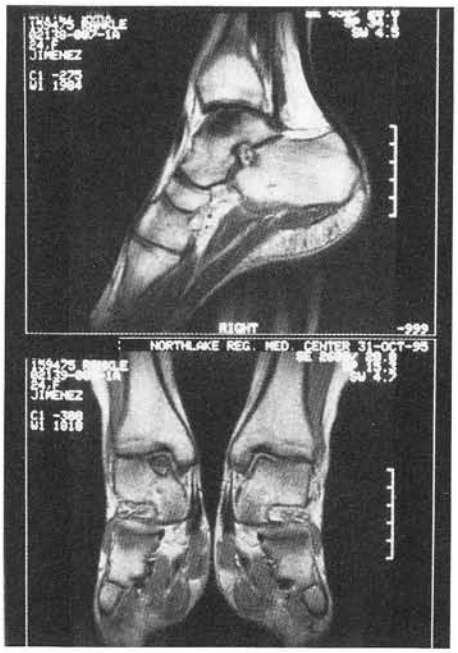

Figure 6B. Sagittal and coronal MRI.

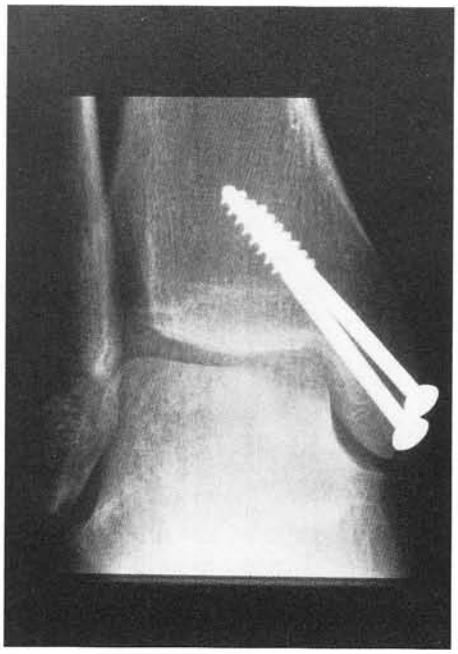

Figure 6C. Five month postoperative AP radiograph

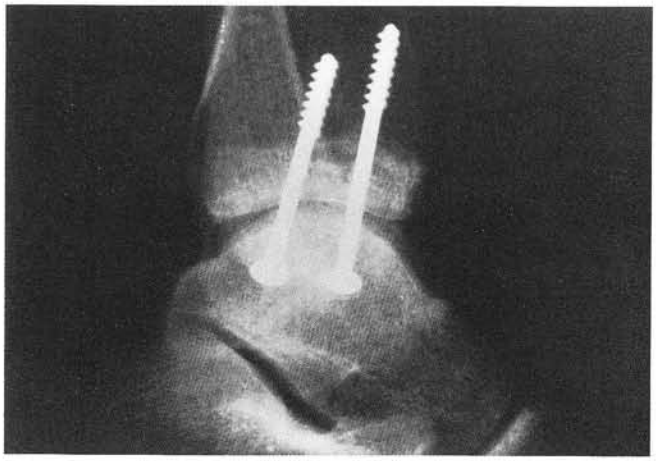

Figure 6D. Lateral radiograph.

Case 3

A 41-year-old woman was involved in a motor vehicle accident and presented to the emergency room with an open laceration over the sinus tarsi of the right foot approximately 5 cm in length. The talus was exposed through the wound (Fig. 7A). Vibratory and sharp/dull sensations were intact. Dorsalis pedis and posterior tibial arteries were monophasic via hand-held Doppler. The foot felt cool and digits began to appear dusky with a capillary refill time of five seconds. Plain films (Figs. 7B, 7C) revealed a comminuted fracture of the posterior process and posterior portion of the talar body, with dislocations of the subtalar and talonavicular joints. The patient was taken to surgery for closed reduction and irrigation of an open talar fracture. The subtalar and talonavicular joints were relocated (Figs. 7D, 7E). Immediately following reduction, both pulses became palpable, although both remained monophasic via Doppler. The intermediate dorsal cutaneous nerve was observed at the medial aspect of the laceration. The anterior talofibular, calcaneofibular, and lateral subtalar joint ligaments were all noted to be disrupted. The peroneal tendons were noted to be intact. The wound was irrigated with sterile normal saline and reapproximated with Steri-Strips. A dry, sterile dressing and non-weight bearing below-knee cast was applied. The patient was placed on cefazolin and gentamicin for three days. A CT scan was taken the next day, and revealed a significant amount of comminution of the posterior portion of the talar body (Fig. 7F).

The patient was discharged from the hospital on the third day postoperative on dicloxicillin and oral ciprofloxacin for five days. The patient was kept non-weight bearing for six weeks. The laceration healed uneventfully and neurologic sensation to the intermediate dorsal cutaneous nerve remained normal.

Two months later, the patient had painful, decreased ankle and subtalar joint range of motion, and decreased first metatarsophalangeal joint range of motion. The patient underwent surgery for excision of the malunited partially healed posterior fracture fragment (Fig. 7G). The patient was allowed to bear weight after two weeks. The patient had improvement in pain with range of motion, however, range of motion remained painful to a certain degree. The patient was followed for fourteen months with treatment consisting of anti-inflammatory medication, formal physical therapy, and range of motion exercises at home. At sixteen months after initial injury, the patient had significant arthrosis with decreased, crepitant, and somewhat painful range of motion at the subtalar joint and ankle joint (Fig. 7H).

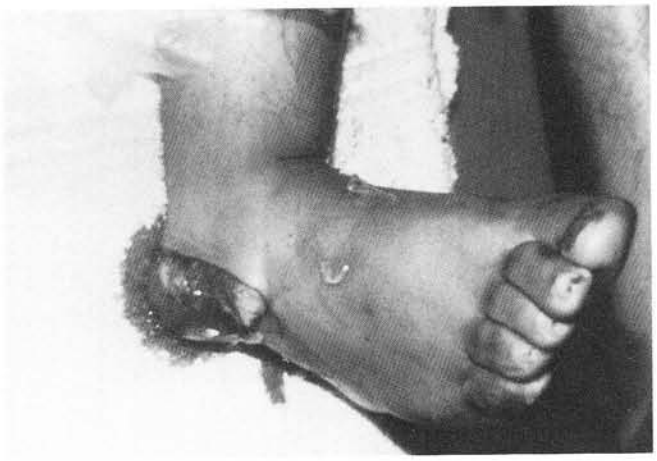

Figure 7A. Clinical appearance on presentation to emergency room.

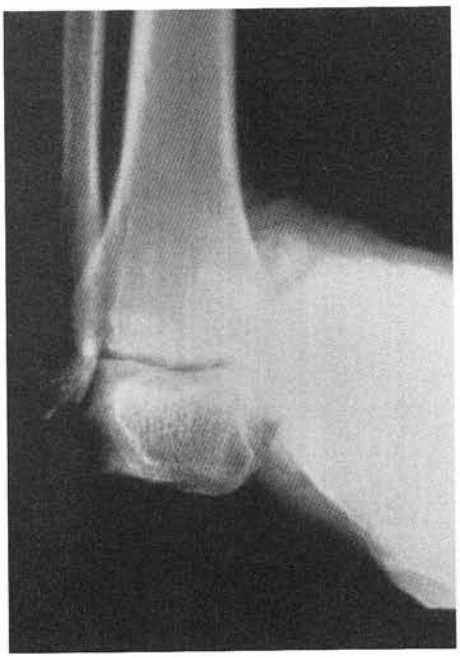

Figure 7B. Pre-reduction AP radiograph.

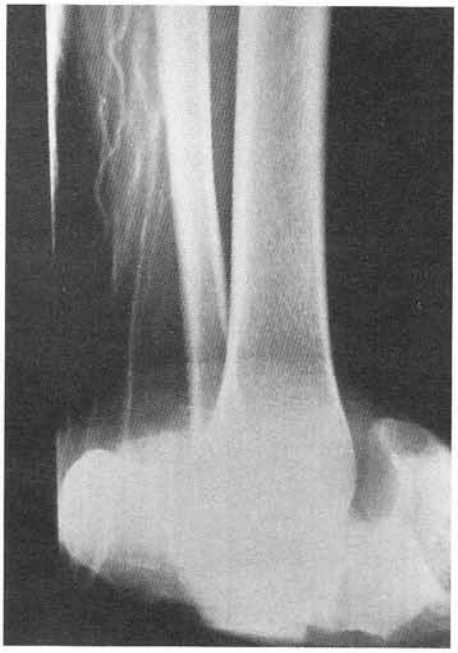

Figure 7C. Lateral radiograph.

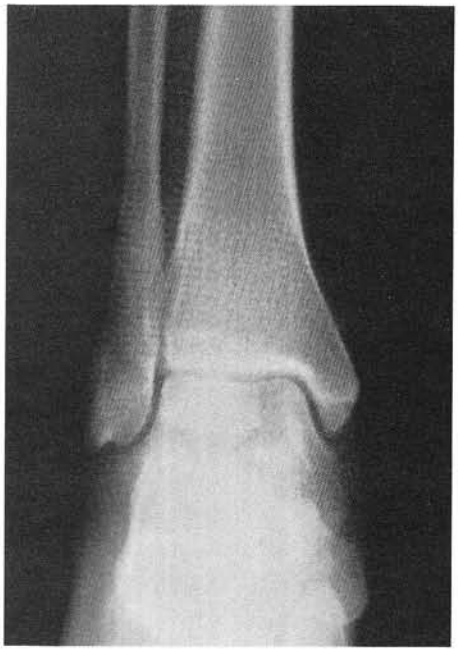

Figure 7D. Post-reduction AP

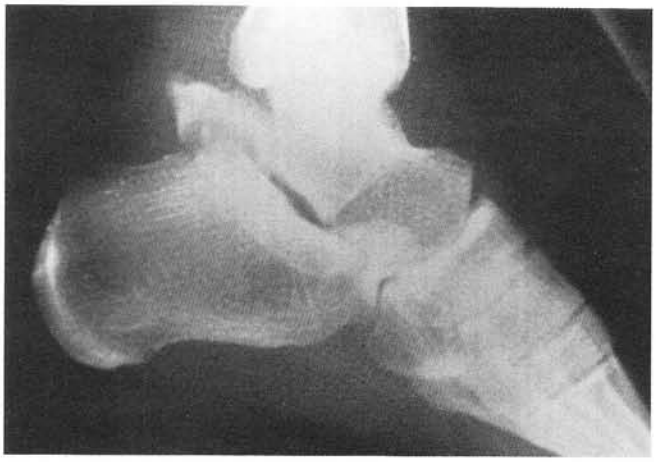

Figure 7E. Lateral radiograph.

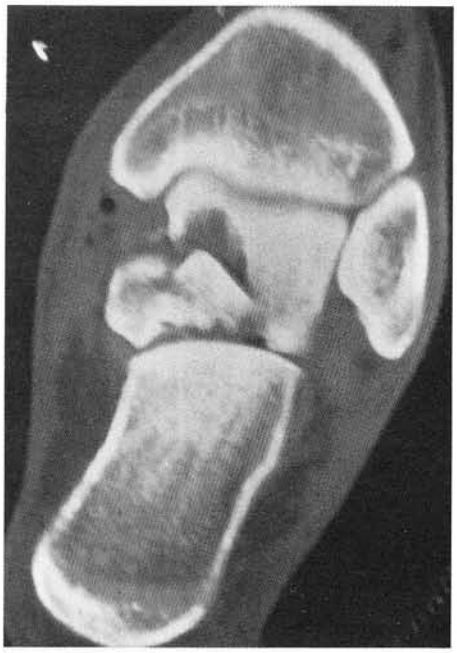

Figure 7F. Post-reduction CT. Note the severe comminution of the posterior portion of the talar body.

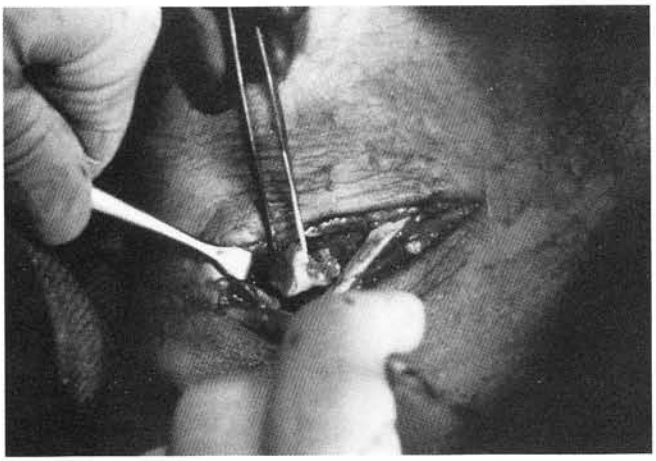

Figure 7G. Intra-operative excision of fracture fragment.

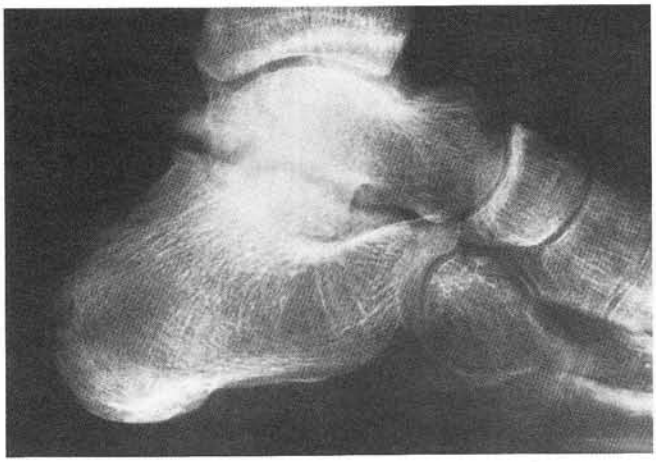

Figure 7H. Sixteen months postoperative lateral radiograph. Note the severe degenerative joint disease of the subtalar and ankle joints.

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