CHARCOT JOINTS

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Charcot joint deformities have always posed a difficult problem for those treating the foot. Unfortunately, physicians have often been unequal to the challenge. Initial therapy typically begins with molded shoes and other supportive and palliative measures and when these procedures fail amputation follows. More recently the members of the Podiatry Institute have been driven by a feeling that there must be something more available to these patients.

Surgical reconstruction is not a new concept, but has rarely been advocated or pursued with any interest. The following is a review of the surgical alternatives for patients with neuropathic joint deformities. A thorough review of the etiology and other basic information about Charcot joints has been previously offered (1, 2).

Surgical Intervention

In the neuropathic foot there are three primary indications for surgery:

Biopsy
Exostectomy
Elective reconstruction (arthrodesis)

1. Biopsy

The diagnosis of Charcot joints may at times be difficult for there are a number of conditions which may mimic this syndrome both radiographically and clinically. The picture is classically complicated by the presence of persistent ulcerations which may have led to the development of osteomyelitis. Both septic arthritis and osteomyelitis may occur simultaneously with neuroarthropathy.

The definitive diagnosis of Charcot joints rests upon a synovial biopsy. The specimen will demonstrate the presence of multiple shards of bone and cartilage embedded within the deeper layers of the synovium. If osteomyelitis is of concern then a bone biopsy is essential for diagnosis.

2. Exostectomy

A simple approach to ulcerative lesions within the lower extremity is to excise the offending osseous prominence. Preferably this is done through an incision site removed from the ulceration to minimize bacterial contamination of the bone. More recently complete elliptical excision of the ulceration with exostectomy and primary wound closure has been described (3).

The authors feel that exostectomy may be applicable in some instances. However, it appears at best a temporary means by which one can alleviate an ulcerative problem. The chief concern is what happens to the foot over the long term. Isolated metatarsal head resection is bound to result in transfer lesions at a later date.

Tarsal exostectomy does nothing to correct the severe pes valgo planus deformity which may be the chief cause of the osseous prominence. The authors feel that many of these patients would be better served by reconstructive procedures which eliminate deformities.

3. Elective Reconstruction (Arthrodesis)

In selected patients reconstruction of the Charcot foot may result in the creation of a functional part and prevent amputation. We have been traditionally led to believe that nothing definitive can be accomplished with the Charcot foot and consequently many such patients have succumbed to amputation. The key to successful reconstruction is proper patient and procedural selection. The following factors must be considered prior to surgery.

Procedural selection:

Arthroplasty is generally contraindicated in the neuropathic foot as the end result of this procedure is an unstable joint space; a situation which already exists in this foot type. Unlike the average patient with osteoarthritis or joint contracture, Charcot patients have no limitation of motion. On the contrary, the motion is typically quite excessive in the neuropathic joint. Implants are generally contraindicated and implant arthroplasty in general is associated with a very high rate of failure when performed in patients with neuropathic hip joints (4, 5).

The overwhelming procedure of choice in Charcot joints is arthrodesis. Joint fusion provides for correction and realignment of deformities, elimination of instability, improved function, as well as maintenance of correction.

Patient compliance:

Above all the patient must be willing to cooperate. Although this seems a foregone conclusion, many patients

may not understand the need for a period of nonweightbearing postoperatively as the foot is generally pain free.

Charcot state:

Surgery should be postponed until the Charcot state is quiescent. In the acute phase the Charcot foot is warm and edematous with the osseous structures being soft and resorption occurring secondary to hyperemia and joint injury. The surgeon should wait until the acute phase has passed and the osseous structures have had an opportunity to consolidate and solidify. This may require a period of nonweightbearing prior to surgery. The remodeling process may be monitored by assessing the temperature differences in the patient's feet. Once a symmetric gradient has returned then primary remodeling is complete.

Bone quality:

One needs to carefully examine the quality of bone prior to surgery. In order to ensure the highest rate of arthrodesis, good healthy osseous tissue must be present. Any bone which is suspect must be resected and grafts used if necessary to achieve suitable alignment. Provisions may need to be made prior to surgery to harvest good corticocancellous autogenous bone for grafting.

Fixation:

When performing arthrodesis in the neuropathic foot the most rigid form of fixation suitable to the procedure is necessary. Preferably this involves the use of screws and/or plates.

Etiology:

As most Charcot joints occur unilaterally it is felt that trauma plays an important role in initiating the process. Usually the event is not remembered by the patient as the neuropathy tends to mask or minimize symptoms. Consequently, the patient may not seek medical attention and appropriate therapy is not rendered. Joint instability is thus created and the Charcot process begins.

Another form of injury may be chronic microtrauma which may be seen in a hypermobile foot. Bone is weakened due to the hyperemia that follows autosympathectomy (6,7,8). Joint sensation is lost secondary to neuropathy, and instability leads to the cycle of fracture and repair.

These two mechanisms (acute vs. chronic trauma) are not mutually exclusive. The acutely injured part may suffer substantial injury; certainly enough to cause the ligamentous instability necessary for initiating Charcot joint. The joint degeneration may later be exacerbated by a number of deforming influences. Ankle equinus is probably the most common and severe destructive influence which will be seen.

Equinus alone may cause sufficient forces upon the foot to initiate Charcot destruction. The equinus may be an inherited shortage of dorsiflexion or an adaptive contracture of the triceps. Secondary contracture of the posterior leg musculature is most likely to occur as a result of the anterior muscle group weakness that accompanies diabetic peripheral polyneuropathy. The effects of ankle equinus may be aggravated by diabetic amyotrophy. This is a proximal weakness which primarily affects the hip and anterior thigh muscles. As the hamstrings are typically spared, knee flexion imbalance may become evident and further exacerbate the disproportionate strength of the posterior leg muscles.

Associated deformities must be addressed at the same time as arthrodesis. Patients with severe equinus who are undergoing Lisfranc arthrodesis will have the stress shifted to other joints if the heel cord is not lengthened.

Circulation:

The circulatory status is of concern in most diabetic patients. We have been programmed to believe that all diabetics have impaired circulatory ability. By definition a patient with a Charcot foot possesses good circulation. Pulses in most of these patients will be strongly palpable and when not palpable such state is most likely secondary to vascular calcification. Arterial calcification does not necessarily imply that there is intimal damage to the vessels. Calcification may occur within the media, and therefore, not primarily affect the arterial diameter.

Clouse et al, in 90 patients with diabetic Charcot foot/ankle deformities, found that 78% had radiographic evidence of vascular calcification (9). One of our patients had such severe vascular calcification that upon attempts to obtain ankle/arm indices the pedal arteries could not be occluded. This individual had complete Lisfranc reconstruction, bled freely during surgery, and healed without consequence.

Discussion of the patient's vascular status would not be complete without mentioning the role of the autonomic nervous system in controlling blood flow to the foot. Sympathetic fibers maintain a constrictive tone upon the vessels of the foot. Diabetic neuropathy will at times affect the sympathetic nerves in addition to the other nerves in the body. Once sympathetic function is impaired the vasomotor tone of the vessels as well as the sweat glands are affected. This results in a well perfused, warm, anhidrotic foot.

Clinical evidence of circulation may also be obtained by examining the feet for evidence of inflammation when an ulcer is present. If the ulcer is associated with edema and erythema then the patient has the capacity to mount an immune response. Inflammation is a vascular dependent process and the degree of inflammation will most likely be proportionate to the adequacy of the blood supply.

Unilateral and nonweightbearing:

Absolute nonweightbearing on the extremity is recommended until adequate healing is assured. Surgery will induce trauma as well as an acute hyperemic response. Early ambulation on the foot will encourage resorption at the arthrodesis site and risk failure. It may also encourage Charcot degeneration in joints which were not fused. The surgical procedure may be selective, but the Charcot process is not, thus all areas of the foot are rendered susceptible. It must be remembered that pain can not be used as an indicator of postoperative progress in these patients. Therefore, one should be hesitant to allow weightbearing too early in the postoperative period and if in doubt tend toward overprotection of the part.

Muscle Function

Muscle function needs to be evaluated prior to reconstructive surgery to determine that adequate power remains to control the reconstructed part. When inadequate function remains the patient needs to be prepared to wear an ankle/foot/orthosis or other appropriate supportive device. Or in many instances additional stabilization arthrodeses may be necessary.

Surgical Technique

Since the Charcot joint is by nature unstable most surgical procedures involve arthrodesis. Additional procedures may include tendon lengthening or tendon transfer to remove deforming influence or to provide power to correct conditions such as "foot drop."

Tarsometatarsal Collapse (Charcot Lisfranc's Joint)

Collapse of Lisfranc's joint presents a frequent and difficult reconstructive challenge. Not only are the five tarsometatarsal joints collapsed, but the intercuneiform and the cuneocuboid joints are likewise destroyed. The metatarsals are dislocated from the tarsal bones and are angulated dorsally and laterally.

There are several difficulties in reconstructing this deformity. The intertarsal joints must first be stabilized in order to provide a suitable foundation for stabilization of the metatarsals. The tarsometatarsal joints must be cleared of all diseased bone followed by anchoring of the five metatarsals onto the tarsal foundation. And the metatarsals must be so aligned that all five bear weight on the same transverse plane at their distal ends.

Intertarsal Stabilization

Before tarsometatarsal arthrodesis can be affected it is necessary to provide a stable foundation. Diastasis of the intercuneiform and cuneocuboid joint is thus reduced as the first priority in restructuring Lisfranc fracture-dislocations. The approach requires adequate exposure to accommodate all possible reconstruction which may be required:

1. Three longitudinal skin incisions provide access to the lesser tarsals and to the tarsometatarsal joints. One incision is placed over the first ray from navicular to mid-metatarsal. A second is placed

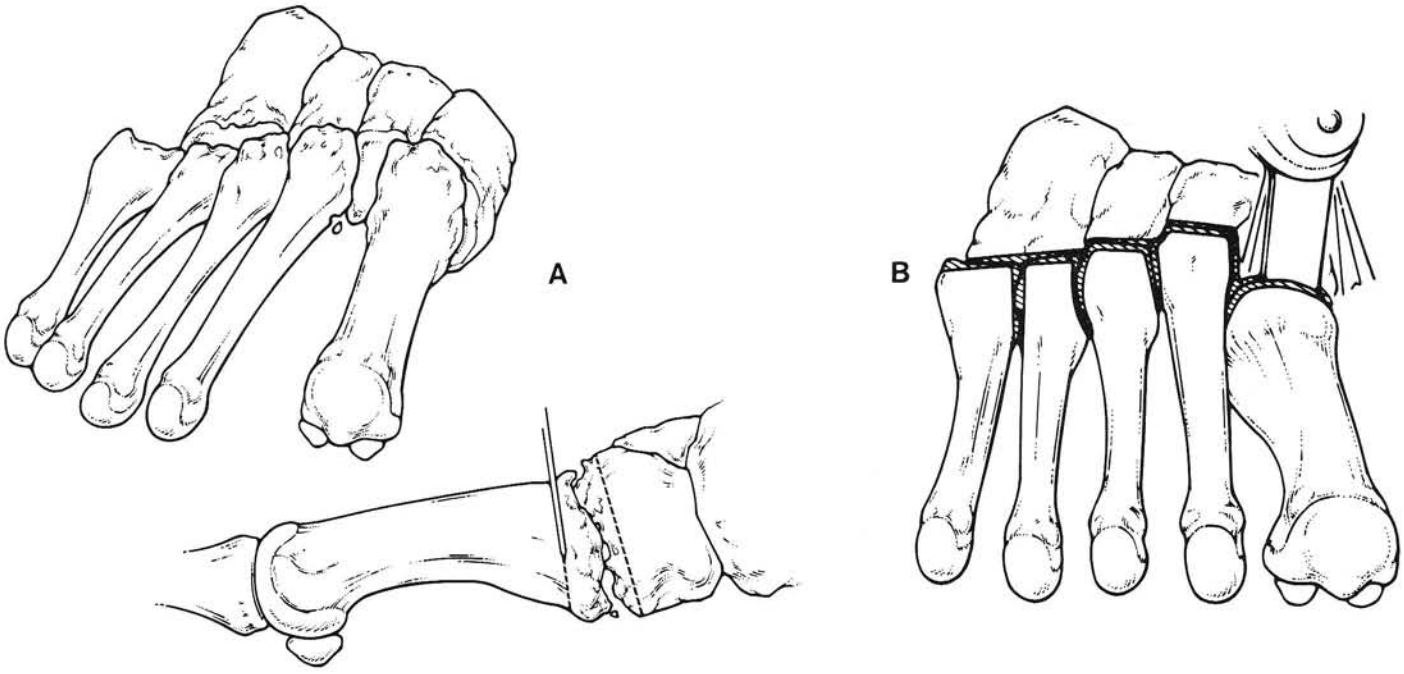


Fig. 1. A. All diseased bone is resected to a level of healthy corticocancellous bone. This includes the interfaces between the lesser tarsal bones. **B.** Reciprocal planing is demonstrated in achieving a near perfect fit of the first cuneometatarsal joint. Reciprocal planing is similarly used in the refitting of all of the involved joints.

over the third ray and extends from the lateral navicular to the neck of the third metatarsal. A third incision is placed over the fifth ray and extends from the calcaneocuboid joint distally to the neck of the fifth metatarsal.

2. Soft tissue dissection follows anatomic planes to the level of periosteal-ligamentous covering.
3. Joint spaces are outlined with the scalpel providing as clear an outline of the bones as is possible with distorted anatomy. Exposure is facilitated by initiating subperiosteal dissection out over the individual metatarsal bones where contours remain normal and extending dissection proximally to outline the diseased joints.
4. A power oscillating saw is used to resect all tarsometatarsal joint surfaces (Fig. 1 A.).
5. The intercuneiform and cuneocuboid joints are next resected. This is often accomplished by holding the joint closed and using coaxial *reciprocal planing* to shave away the interfaces of both bones.
6. The intercuneiform and cuneocuboid joints are then closed and compressed with transfixing screws. In the rare instance where there is no diastasis of the lesser tarsal joints this maneuver is unnecessary.
7. With the lesser tarsals stabalized one can proceed to reconstruction of the tarsometatarsal joints.

Two major technical problems occur in anchoring the metatarsals to the lesser tarsal foundation. First, there are five metatarsals. Their length needs to be controlled to a reasonable parabola. More importantly the five metatarsal heads need to be lined up reasonably close to the same transverse plane. This is necessary to assure even weight-bearing and to minimize the potential for ulceration beneath prominent metatarsals. Second, the metatarsals need to be fitted tightly at the interfaces between the five bases. This encourages intermetatarsal base arthrodesis while also arthrodesing the tarsometatarsal joints. Additional strength to the metatarsal structure is obviously gained by such fitting.

Transverse Plane Alignment and Fitting

After appropriate resection of diseased bone transverse plane alignment is planned as follows:

1. The first metatarsal is placed in position on the first cuneiform. The surgeon holds the desired position of the metatarsal while an assistant utilizes *reciprocal planing* to produce a perfect fitting of the joint surfaces to that alignment (Fig. 1 B). An .062 Kirschner wire is placed obliquely across the first metatarsocuneiform joint as temporary fixation (Fig. 2 A.).

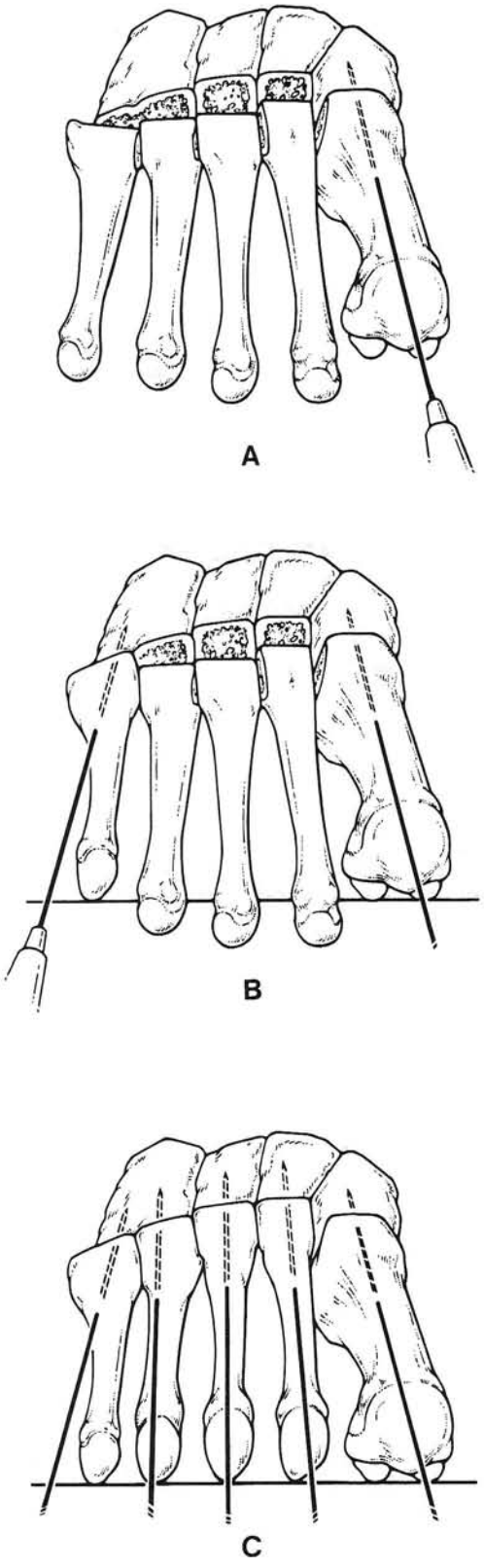


Fig. 2. A. First cuneometatarsal joint is temporarily fixated with a .062 Kirschner wire. **B.** The fifth metatarsal is the second to be fixated with appropriate attention to angle of declination of the bone. This then establishes a transverse plane for fixation of the intermediate metatarsals. **C.** All five tarsometatarsal joints are temporarily fixated before placing permanent fixation screws.

2. The fifth metatarsocuboid joint is held in the desired alignment while the assistant utilizes *reciprocal planing* to fit the surfaces perfectly. A second .062 Kirschner wire is used to temporarily fixate the fifth metatarsocuboid alignment (Fig. 2 B.). This then establishes alignment for a transverse plane. A flat object can be held across the plantar of the foot from first to fifth metatarsal to assist the in-plane alignment.
3. The second metatarsal is next held in position by the surgeon. The assistant first *reciprocally planes* the interface between the first and second metatarsal bases. Next the interface between the second metatarsal and the second cuneiform is *reciprocally planed*. A temporary .062 Kirschner wire is used to fixate the precise position of the second metatarsal.
4. The third metatarsal is aligned, held by the surgeon and the medial base interface with the second metatarsal *reciprocally planed*. The cuneometatarsal joint is then fitted and temporary fixation obtained.
5. The fourth metatarsal is fitted and temporarily fixated by the same maneuvers while being held firmly on the transverse plane.
6. If a tight fit is not present the fourth and fifth metatarsal is refitted against the fourth metatarsal and refixated. This is usually necessary since the fitting of the bases should result in some narrowing of the medial to lateral measurement (Fig. 2 C.).
7. Rigid internal fixation with lag screws and plates is then accomplished as appropriate.
8. The limb is placed in an above knee bivalved non-weightbearing cast to facilitate close observation and redressings.
9. By 4-6 weeks postoperatively the cast can usually be cut to below knee.
10. Non weightbearing is continued until radiographic evidence of bony union is present.
11. A well fitted accommodative orthotic device is used in a padded counter basketball shoe for the first two months following return to full weightbearing.
12. An ankle/foot/orthosis may be employed for several months or until return of muscle strength if muscle-tendon lengthening has been performed.
13. Appropriate muscle strengthening exercises are ordered where residual function suggests that it may be fruitful.
14. A cane is suggested until patient regains coordinated function and adequate muscle strength, especially where achilles lengthening has been performed.

Charcot Peritalar Joints

Peritalar Charcot joints, like Lisfranc's collapse is especially common in the foot which presents with significant equinus and in which compensation for restricted ankle dorsiflexion occurs at the subtalar and midtarsal joint (Figs. 3 A-C.). The ankle joint may or may not be affected by the degenerative changes. The principles of reconstruction here are no different than those described in restructuring Lisfranc's joint. Diseased bone must be resected to a level exposing healthy cortical and cancellous bone. If bony gaps remain they may be filled with autograft or high quality allograft. Rigid fixation is essential and deforming influences must be recognized and corrected or accommodated (Fig. 3 C.).

If indeed equinus is present it must be addressed, despite the fact that recovery from resulting muscle weakness will be slow if achilles lengthening is performed. Failure to correct equinus, whether bony or soft tissue in origin will predictably result in breakdown at another level. Equinus is therefore corrected as a part of the surgical reconstruction. The patient is prepared for the fact that recovery of muscle strength will be slow and in some instances may necessitate permanent bracing. In our experience such bracing only becomes necesary in the most severe instances.

Where profound anterior muscle weakness contributes to equinus or to overt dropfoot deformity consideration should be given to anterior transfer of one or more posterior leg muscles. Obviously, that which can be transferred is dependent on the muscle inventory.

Triple Arthrodesis

Triple arthrodesis is the mainstay of rearfoot reconstruction in the Charcot peritalar collapse. If the ankle joint is also involved then pan talar arthrodesis is considered (Figs. 3 A-C.).

We have no reluctance to perform rearfoot arthrodesis in such patients. The alternative is usually below knee amputation. There is, therefore, much to gain and little to lose.

Following triple or pantalar arthrodesis the patient is retained in an above knee bivalved cast for 4-6 weeks followed by an additional 6-8 weeks in a below knee bivalved cast with bathing and soaking and active unresisted range of motion exercise prescribed.

In the patient who is unreliable and who may be subject to placing weight on the foot prematurely an above knee cast with 45 degree flexion at the knee is recommended. Such casting should be continued until arthrodesis is confirmed radiographically.

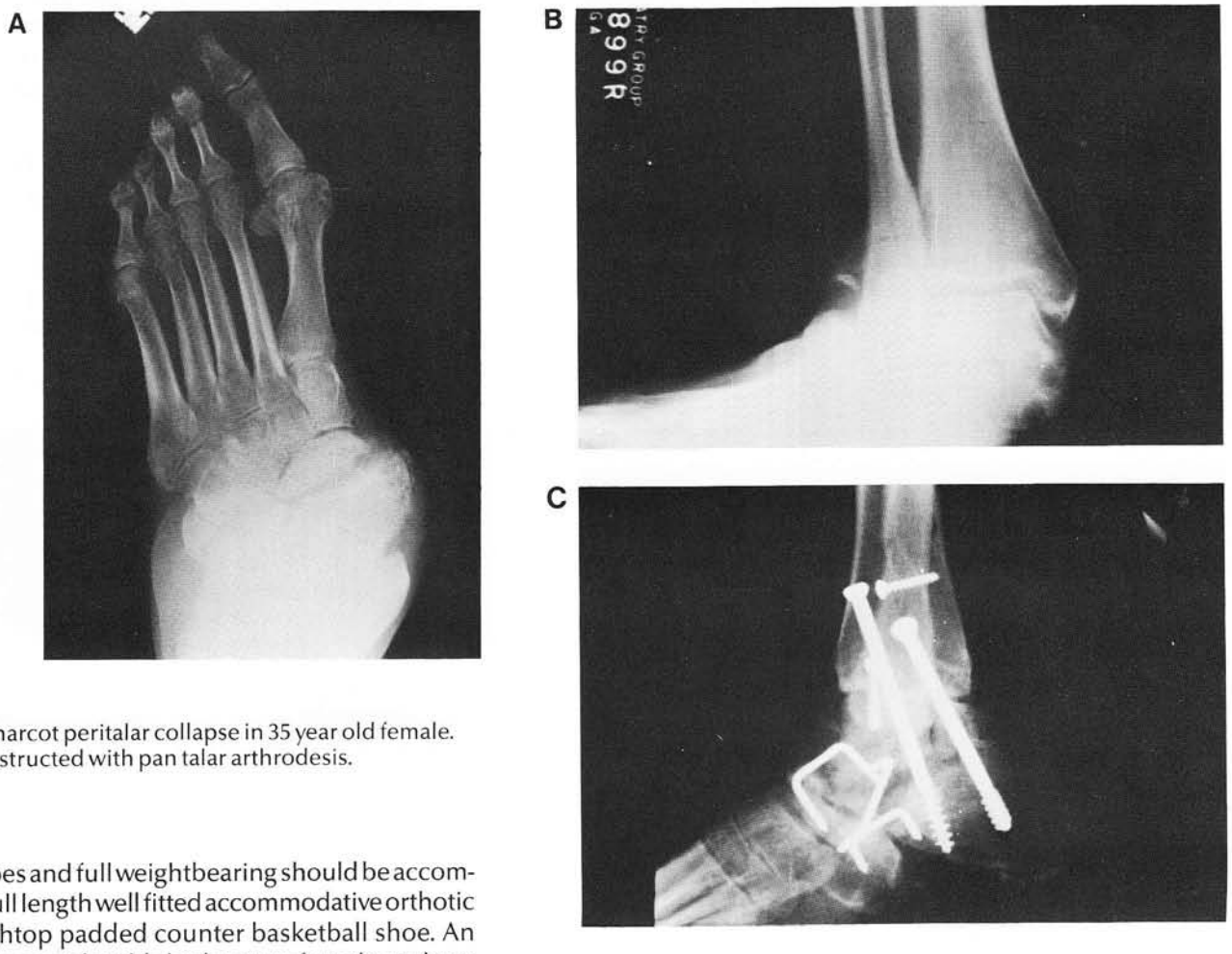


Fig. 3. A, B, C. Charcot peritalar collapse in 35 year old female. Surgically reconstructed with pan talar arthrodesis.

Return to shoes and full weightbearing should be accompanied with a full length well fitted accommodative orthotic device in a hightop padded counter basketball shoe. An elastic ankle support should also be worn for at least three months. A cane is encouraged until there is sufficient muscle tone to permit safe ambulation without such.

Exercise, physical therapy, control of diabetes and any other measures which may encourage some return of function should be recommended.

Case Histories

Case 1 (from Gerald Falke,D.P.M., Hagerstown, Maryland)

G.K. is a 58 year old insulin dependent diabetic of long standing. He is seen on referral from Maryland for reconstructive surgery. He provides a history of progressive increase in peripheral neuropathy over the past several years. He had been quite athletic as a youth and in recent years has worked with youth league baseball. Despite proper shoes and well fitted orthotic devices he has seen progressive collapse of the right arch (Figs. 4 A-C.).

Clinical examination showed moderately advanced neuropathy of both feet and legs. Vascular examination revealed strong palpable posterior tibial and dorsalis pedis pulses. The skin was dry and warm. Capillary rebound at the pulp of the toes was instantaneous.

Posterior leg muscles appeared to retain normal strength while the anterior muscles showed significant weakness. Range of motion examination demonstrated profound ankle equinus with severe restriction of ankle dorsiflexion. Crepitas was pronounced when the forefoot was moved on the rearfoot.

Weightbearing examination showed a rockerbottom foot with total collapse at Lisfranc's joint and with failure of the heel to make full contact. The opposite foot showed severe pronation and the presence of equinus, but the foot was still structurally controllable.

Radiographic examination demonstrated complete collapse of Lisfranc's joint as well as diastasis of the of the intercuneiform and cuneocuboid joints. The first cuneiform bone showed near total destruction (Figs. 4 A-C).

A diagnosis of diabetic neuroarthropathy by the referring physician was found accurate and the patient cleared for the planned reconstructive surgery.

Surgical considerations included the need to plan for an autogenous bone graft to replace the totally collapsed first cuneiform. Additionally, tendo Achillis lengthening was

deemed necessary since the severe equinus present would have assured breakdown of other joints if left uncorrected.

Surgical Reconstruction

The principle reconstruction included:

1. Resection of all five metatarsal bases to a level of healthy corticocancellous bone (Fig. 4 D).
2. Resection of the distal portion of the cuneiform and cuboid bones to a level of healthy bone (Fig. 4 D).
3. Complete excision of the first cuneiform bone.
4. Reciprocal planing of the interfaces between cuneiforms and cuneocuboid joints to a level of raw denuded cancellous bone surfaces (Fig. 4 D).
5. Replacement of first cuneiform with iliac crest corticocancellous graft.
6. Temporary fixation of first and fifth metatarsals with .062 Kirschner wires to establish a transverse plane.
7. Manual alignment of each of the intermediate tarsometatarsal joints while having an assistant *reciprocally plane* the interface to a perfect fit (Fig. 1 B).

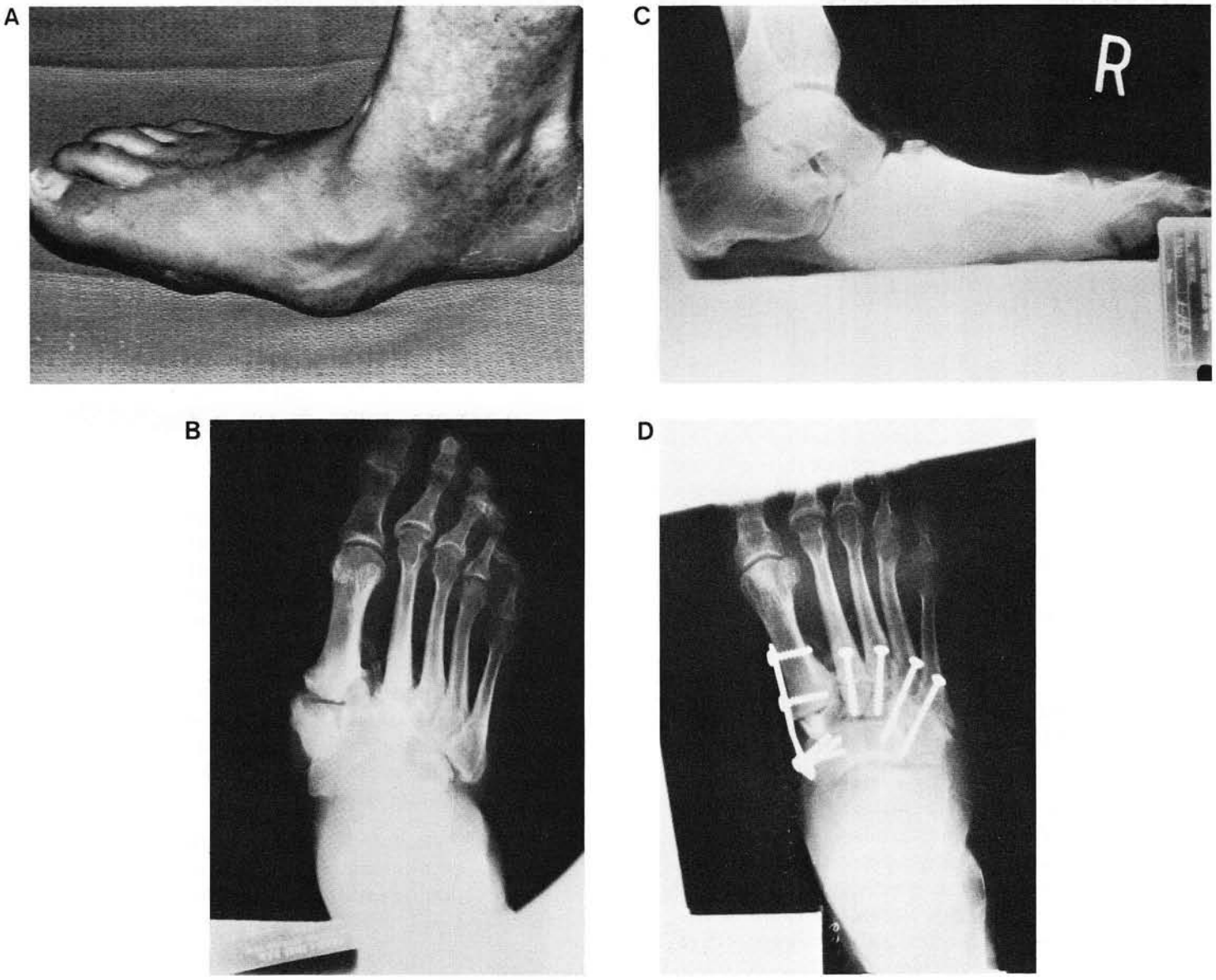


Fig. 4. A, B, C. Patient is a 58 year old male insulin dependent diabetic of long standing duration. Clinical and radiographic appearance of foot after Lisfranc's joint collapse. **D.** Lisfranc's joint resection included resection of all diseased bone from the intercuneiform joints, distal ends of the cuneiforms and cuboid, and the proximal ends of the metatarsal bones. Autogenous iliac crest graft is used to replace destroyed first cuneiform. Screw and plate fixation provide rigid internal fixation.

8. The interfaces between the metatarsal bases were also *reciprocally planed* to provide for raw bone-to-bone fitting (Fig. 2 C.).
9. Screw and plate fixation to provide rigid internal fixation (Fig. 4 D).

Post operative follow up was relatively uneventful. The patient was discharged from the hospital on the sixth post operative day. Because of the size of the bone graft it was expected that 4-6 months would be required before unassisted weightbearing was placed on the foot. Actually, the graft appeared solidly incorporated and at 5 months the patient returned to shoes with a supportive device. One year following surgery he was walking well with an accommodative orthotic device in the shoe (Figs. 5 A, B.). Some two years post surgery the patient continues to do well. He has been able to return to coaching youth baseball.

Case 2

W.F. is a 43 year old white insulin dependent diabetic female. She was first seen on referral 2-4-87 with a severely swollen, erythematous right foot and ankle (Figs. 6 A, B). A large plantar ulcer was present beneath the first cuneometatarsal joint with purulent discharge. The foot showed a rocker-bottom configuration with the apex of the bulge beneath the cuneometatarsal joints.

The patient indicated that she had been under treatment to heal the ulcer for 11 months with contact casting changed at regular intervals and was currently scheduled for below knee amputation of the limb.

A

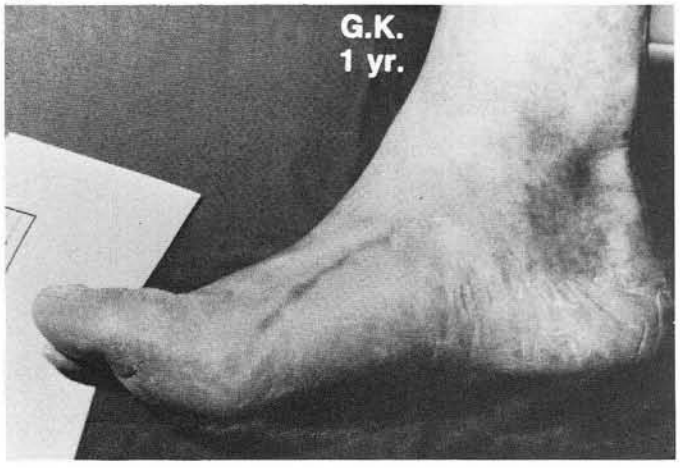


Fig. 5. A, B. Follow up radiographic and clinical appearance at one year following surgery.

With the extreme edema, heat, and redness present it was evident that adequate circulation was present in the limb. Palpation revealed the presence of a deep central compartment abscess in the arch of the foot (Fig. 6 A).

Radiographs of the foot showed Charcot tarsometatarsal joints as well as intertarsal joints (Figs. 7 A, B).

Range of motion examination revealed a minus 15 degree dorsiflexion of the ankle, confirming severe equinus related to profound neuropathy with anterior muscle weakness. Compensation for the equinus was greatest at the tarsometatarsal joints with collapse of the joints. Compensation also occurred at the midtarsal and subtalar joints. The heel failed to make contact with the weightbearing surface in stance.

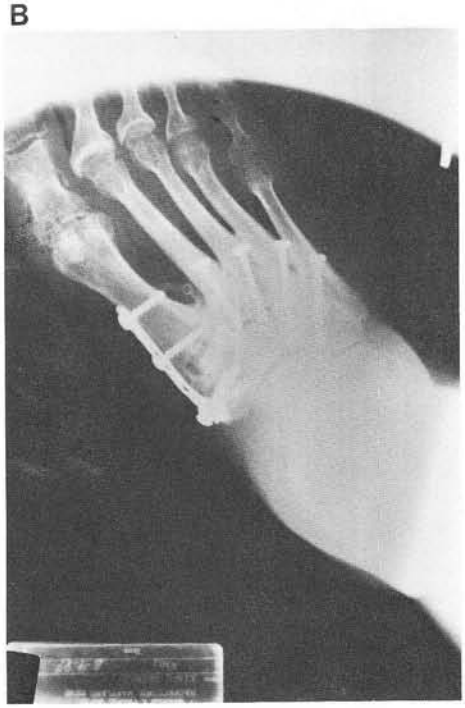
Muscle testing demonstrated rupture of the tibialis posterior tendon with loss of stability of the midtarsal and subtalar joints.

The patient was taken to surgery the same day for deep drainage and debridement followed by daily irrigation and packing. Within a matter of hours the foot improved greatly with a dramatic decrease in edema, erythema, and temperature. Within 2 weeks the ulcer and the deep plantar incision were well healed and little sign of inflammation remained. The foot was also approaching normal size. Throughout this period total non weightbearing was required for the involved foot.

Reconstruction

A period of two months total non weightbearing was

B



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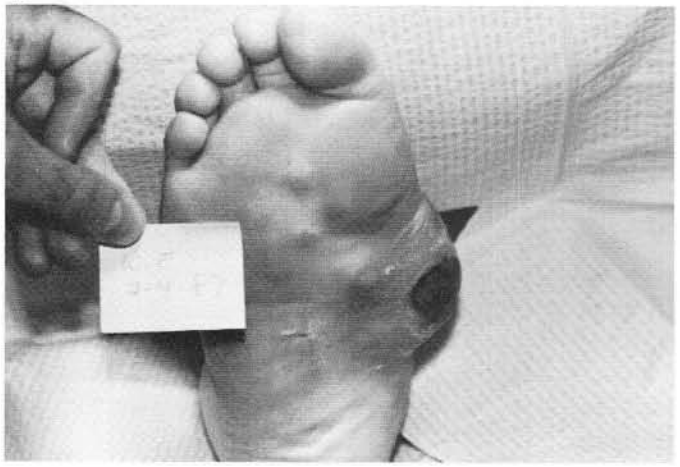

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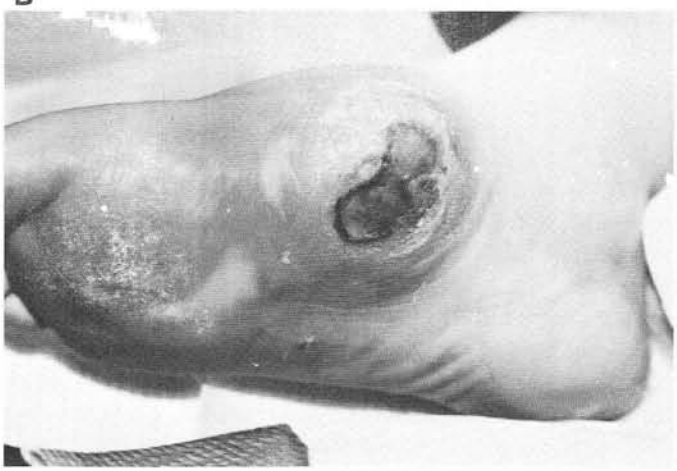

Fig. 6. A, B. Forty-three year old female insulin dependent diabetic with profound peripheral neuropathy. Patient is seen with acutely inflamed, severely swollen right foot with plantar ulceration and deep central compartment abscess which required immediate incision and drainage.

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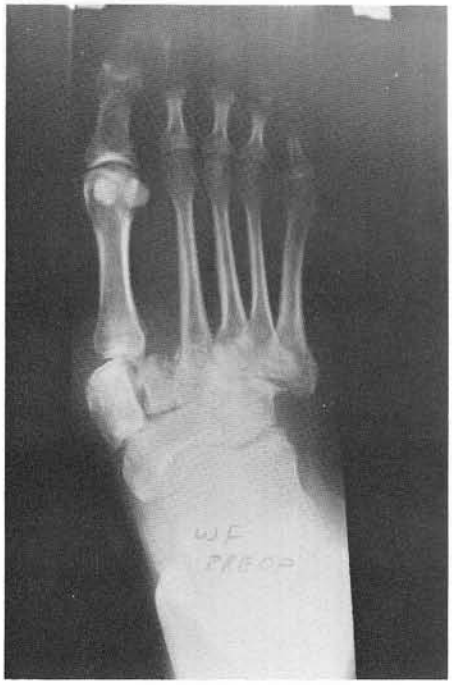

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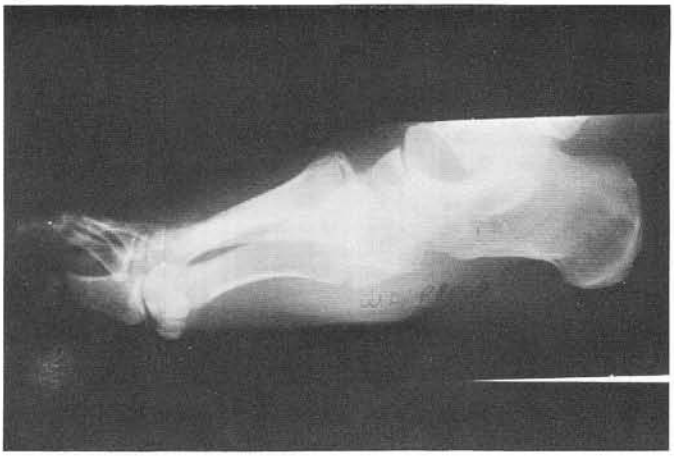


Fig. 7. A, B. Radiographs show Charcot tarsometatarsal and intertarsal joint destruction.

allowed for all inflammation to subside and tissues to return to a supple normal. Patient was then returned to surgery for the first of two stage surgery.

Stage One

Procedures included:

1. Arthrodesis of intertarsal joints, intercuneiform and cuneocuboid. The joints were stabilized with lag screw fixation (Fig. 8).
2. Resection of all diseased bone followed by arthrodesis of all tarsometatarsal joints. No bone grafting was necessary.
3. Tendo Achillis lengthening.

Recovery was uncomplicated and patient was maintained in an above knee bivalved cast. At 9 weeks post surgery radiographs showed solid healing of all arthrodeses (Figs. 9 A, B). Ten weeks postoperatively little edema and no erythema remained (Figs. 9 C, D). The patient was placed in an ankle brace and a hightop shoe with a full length orthotic device and allowed weightbearing with a cane. Ankle bracing was felt necessary since the midtarsal and subtalar instability has yet to be addressed.

Stage Two

Three months after stage one reconstruction the patient was returned to surgery for talonavicular arthrodesis to provide midtarsal stability in the absence of a functional tibialis posterior tendon (Fig. 10).

Discussion

The above patient illustrates several clinical points worth repeating. First, the patient mounted a pronounced inflammatory response to infection when first seen. This said a great deal about her circulation. Without satisfactory circulation the pronounced inflammatory response would not have been seen.

Bone healing and indeed skin and other soft tissue healing occurred in slightly over one-half the time required by a normal healthy patient. This is attributable to the fact that the patient's pronounced neuropathy (anesthetic below knee) had produced an autosympathectomy. Thus, the small blood vessels stood open wide allowing a very pronounced blood flow to the periphery. This same increased blood flow is thought responsible for areas of pronounced bone absorption and softening and facilitates the collapse of joints that are under excessive compression. This, of course, tends to explain the occurrence of Charcot joints in the feet at points of greatest forced compensation.

The same hyperemia responsible for the absorption of bone will also lead to continuing softness of bone following surgery. It is therefore essential that arthrodesis be quite solid before exposure to stresses of weightbearing.

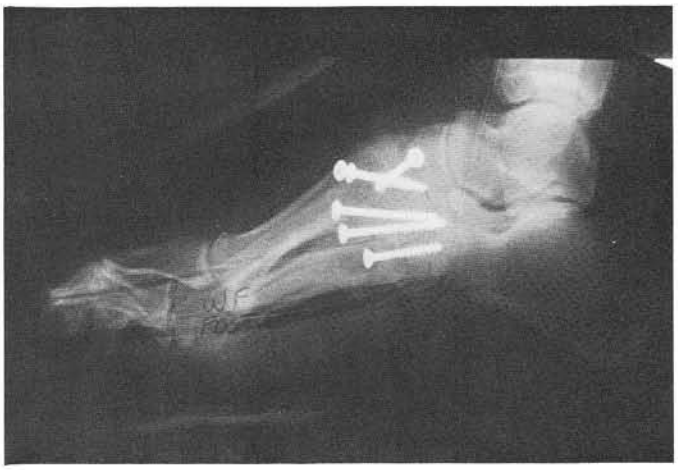


Fig. 8. Postoperative radiograph following first stage of reconstruction.

The degree of neuropathy in this instance was sufficient to allow the drainage of a deep plantar abscess and two subsequent reconstructive surgeries, without the necessity for anesthesia. The anesthesiologist monitored the patient throughout surgery and was prepared to provide spinal anesthesia if required. It was not needed.

Summary

Patients with neuropathic joint collapse can often be reconstructed. Such patients tend to have very adequate circulation; indeed hyperemia is most often present. It is necessary to confirm the presence of competent circulation before engaging in surgery.

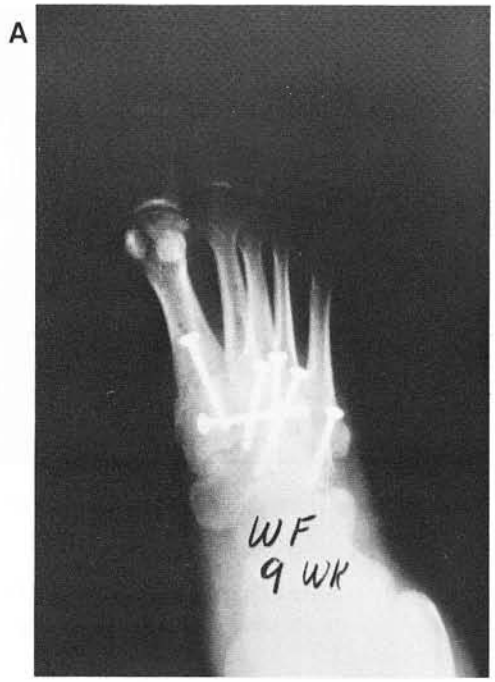
Reconstructive surgery usually involves arthrodesing procedures as the primary approach to reconstruction. One essential is that all diseased bone must be resected.

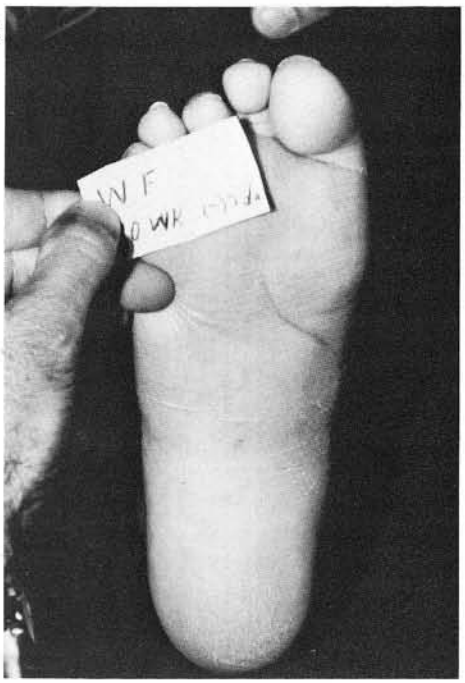
It is equally important to identify and correct deforming influences such as equinus which if uncorrected would result in repeat collapse at the same or another level within the foot or ankle.

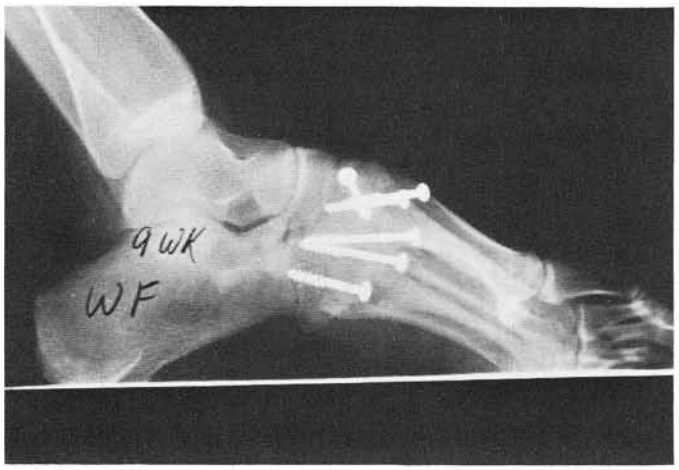
Where ruptured tendons such as the tibialis posterior are present it is unwise to expect tendon repair to be effective. More often joint stabilization (in this case talonavicular or triple arthrodesis) can provide stability.

In patients with dropfoot deformity resulting from anterior muscle weakness it is often wise to transfer a posterior muscle to the dorsum of the foot. Such transfers can contribute greatly to returning the patient to a functional capacity without upright leg bracing.

Motor and sensory neuropathy do not occur at the same rate, and there is often very good posterior muscle strength







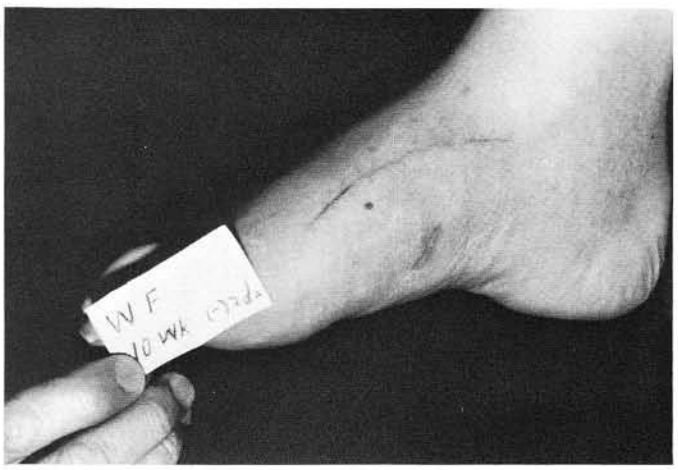


Fig. 9. A, B. Radiographs at 9 weeks post surgery show good consolidation of arthrodeses. **C, D.** Clinical appearance of foot at 10 weeks following stage one of surgical repair.

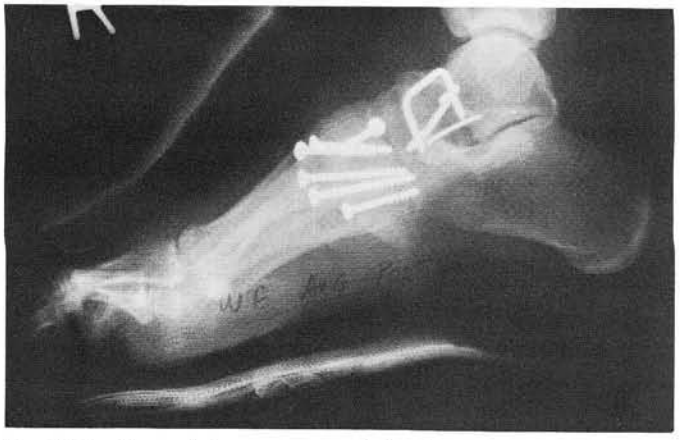


Fig. 10. Radiographic appearance following stage two of surgical reconstruction. Talonavicular arthrodesis is evident and was required because of ruptured tibialis posterior tendon.

in the presence of profound sensory neuropathy.

The same neuropathy which places the foot at risk for the foregoing complications also increases the risks of complications following surgery. It is essential that the patient be monitored closely during convalescence to prevent complications caused by constriction or pressure. A bivalved cast that facilitates frequent inspection is helpful.

The neuroarthropathic foot can be reconstructed but must be done with respect for the pathology and caution for any slight complication.

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