

UNDERSTANDING DIABETIC AND DIABETIC CHARCOT FOOT RECONSTRUCTION

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INTRODUCTION

Diabetic and diabetic Charcot foot deformities occur in a wide variety of forms and in widely differing degrees of diabetic involvement. Charcot joint changes may affect virtually any joint of the foot.

The hallux interphalangeal joint is the most commonly affected joint of the digits (Fig. 1). All of the metatarsophalangeal joints are susceptible as well, but the most commonly involved metatarsophalangeal joints appear to be those which are under the greatest stress. The incidence of dislocation of the metatarsophalangeal joints and mal perforans ulcers appears to be increased with Charcot metatarsophalangeal joints.



Fig. 1. Charcot hallux interphalangeal joint is a frequent finding in diabetic neuroarthropathy.

Lisfranc's joint is especially vulnerable to Charcot joint collapse (Fig. 2A-B). Most often the first ray collapses and is followed in predictable patterns by collapse of the remaining tarsometatarsal joints with progressive abduction and dorsiflexion of the forefoot. A rockerbottom foot develops and is most often associated with the development of mal perforans ulcers beneath the collapsed joints.



Fig. 2A-B. Sixty-eight year old insulin-dependent diabetic male with Charcot Lisfranc's and midtarsal joints. Foot is shown after more than two months of wheelchair confinement to allow healing of mal perforans ulcer and equilibration of soft tissue and bone with opposite foot.



Fig. 2B.

Collapse of the midtarsal and subtalar joints may precede or may be intimately associated with collapse of Lisfranc's joint. Such collapse is most often seen in conjunction with ankle equinus. It is the restriction in ankle joint dorsiflexion which supplies the pathologic force to encourage collapse. This may be either congenital or may be a direct result of motor neuropathy. In either event, loss of ankle dorsiflexion inflicts severe compression forces on the ankle, the subtalar and midtarsal joints in addition to Lisfranc's joint (Fig. 3A-B).

Charcot ankle joint collapse is most often associated with collapse of major tarsal and tarsometatarsal joints. When the ankle is involved, the deformity is usually quite severe.

The etiology of diabetic Charcot joints is still widely debated. Despite such debate, there is more than ample evidence to implicate mixed neuropathy as the principle etiology.

Autonomic (sympathetic) neuropathy causes a dramatic increase in peripheral circulation. This increase appears due to loss of vasomotor tone. Increased circulation results in the bone mineral being washed-out more rapidly than it can be replaced. Bone strength is greatly weakened by mineral depletion, making it much more vulnerable to fracture or collapse under stress.

Sensory neuropathy provides for a decrease in sensations related to standing and walking. Stresses on joints that would normally cause a patient to shift weight to the other foot or to adjust posture are either not felt or are felt with less urgency. The early warning system is thus compromised adding to the vulnerability of the peripheral joints.

Motor neuropathy causes the development of an imbalance in strength. In the thigh, the quadriceps tend to weaken early. This often compromises knee function. In the leg, the anterior muscles weaken more rapidly, leaving the posterior muscles with a mechanical advantage and resulting in progressive limitation of ankle dorsiflexion. Such limitations cause increased pronation with severe increases in compression forces at the ankle, the subtalar and midtarsal, and at Lisfranc's joints.

In the foot, motor neuropathy results in weakening of the intrinsic muscles. Clawtoe or hammertoe deformity results and retrograde force of the toes against the metatarsal heads produce Charcot dislocations of the metatarsophalangeal joints and the development of plantar mal perforans ulcers.

It should be apparent that the interplay of all three forms of neuropathy contribute to develop a Charcot type joint collapse. Autonomic neuropathy weakens bone by increasing peripheral circulation. Sensory neuropathy allows a patient to tolerate increased compression forces of the joints without the usual discomfort that would cause postural accommodation. Motor neuropathy causes uneven muscle atrophy which results in equinus of the ankle and in clawing of the toes. These latter changes supply severe compression forces to the already compromised joints. The result is Charcot joint collapse.

Little needs to be said about peripheral circulation in the diabetic Charcot foot. In fact, one may say that it would be virtually impossible to develop Charcot changes without increased circulation. The Monckeberg sclerosis of blood vessels often visualized on radiographs of the foot does not indicate decreased circulation. The medial sclerosis of Monckeberg is probably due to loss of vasomotor tone which facilitates calcification of the medial coat of the arterial wall. Such patients

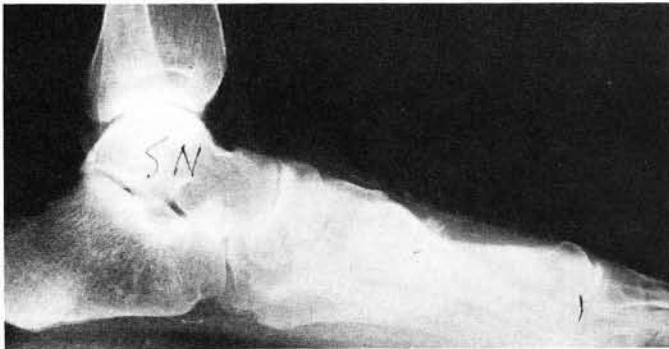


Fig. 3A-B. Thirty year old insulin-dependent diabetic who demonstrates significant ankle equinus. Forefoot breakdown is seen with multiple metatarsal fractures and early collapse at Lisfranc's joint in right foot. Charcot metatarsophalangeal joints are seen in right second, third, and fourth joints.



Fig. 3B.

appear to have greatly increased circulation even in the presence of pipe stem arteries.

Certainly there are many diabetic patients who do develop severe peripheral vascular compromise. These patients are not the ones who develop diabetic Charcot joints.

MATERIALS AND METHODS

Surgical Evaluation

Any surgical evaluation must consider the diabetic patient both from a general medical and a podiatric surgical standpoint. Patients with mixed neuropathy may have associated cardiac or gastrointestinal disorders of a neuropathic origin. The severity of the peripheral neuropathies fails to correlate well with the degree of systemic involvement.

Podiatric surgical evaluation must consider peripheral circulation, muscle function and biomechanical compromise, edema and induration, condition of soft tissues, and patient temperament and reliability.

Evaluation of peripheral circulation cannot end with palpation of pulses. Indeed, the pulses may not be palpable even with the presence of increased circulation. This is predictable in Monckeberg's medial sclerosis. The same patient on Doppler examination will evidence increased circulation. Capillary rebound and venous filling time should be assessed and noninvasive vascular studies used as needed.

Severe edema and induration are often present with diabetic Charcot foot collapse. The inflammatory response of bone attempting to heal in the presence of constant motion maintains inflammation and contributes to induration of soft tissue. During this stage, bone is quite soft due to the presence of inflammatory soup. This is not the time to perform surgery.

Since any reconstructive surgery will involve considerable convalescence, it is important to determine whether a patient is psychologically prepared for the time and effort that will be necessary. It is far better to avoid surgery than to perform it on a patient who cannot be relied upon to follow necessary postoperative instructions. The equilibration period prior to surgery provides an excellent opportunity to determine whether or not the patient is adequately committed to the planned reconstruction.

Whether to Operate

A decision to operate should be based on an analysis of risks vs. potential gain. In many instances the patient has the choice of amputation of the foot, amputation of the foot and leg, or an attempt to reconstruct and stabilize the foot. If the patient is physically and emotionally able to withstand the surgery, and if the foot can be salvaged, then the patient's entire life expectancy may be extended. It is well recognized that the loss of a limb places the opposite limb at an increased risk for complications and amputation.

When to Operate

Providing the patient is physically and emotionally suited for surgery and the lengthy convalescence that may be required, the question then is not whether to operate, but when?

As already mentioned, when the foot is swollen, inflamed, hot, indurated, and with or without ulceration one should defer surgery. Typically, the patient should be placed on total non-weight bearing with wheelchair or bed rest until the involved foot equilibrates with the noninvolved side (Fig. 2A,B). If a wheelchair is used it must have leg rests. Even the weight of the leg pressing the foot against the foot rest will delay or prevent equilibration. To operate in the presence of significant inflammation makes fixation of the involved bones nearly impossible, similar to trying to fix jello to ice cream.

After four to eight weeks of total non-weight bearing without allowing the foot to touch down, one can expect resolution of inflammation, elimination of edema, return to normal or near normal skin temperature, softening of induration, and remobilization of skin and subcutaneous tissue. Additionally, the bone undergoes considerable hardening which will facilitate rigid internal fixation. The foot is then in condition to undergo surgical reconstruction.

Surgical Approaches

Development of a comprehensive plan is of paramount importance in reconstructing a diabetic Charcot foot. The foot may be collapsed at Lisfranc's joint and at midtarsal and subtalar joints, but if significant equinus is present, one must include correction of equinus as a central part of surgical reconstruction. Otherwise, residual equinus will result in breaking the foot at the same or other level once weight bearing resumes (Fig. 4 A-G).

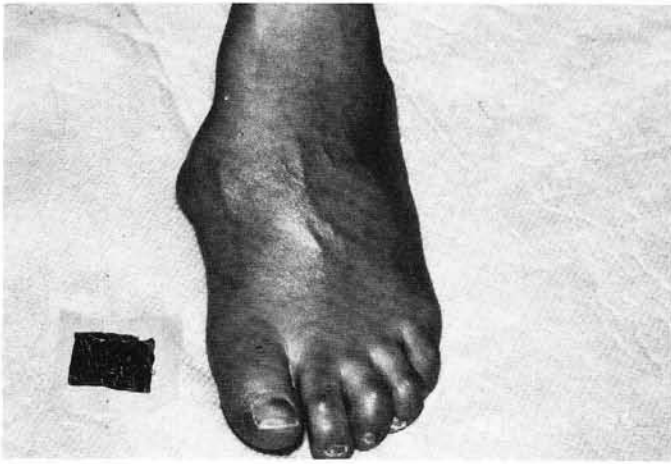


Fig. 4A-G. Shows 41 year old insulin-dependent female who presented with triceps equinus and Charcot joint collapse of Lisfranc's joint. A-C illustrates preoperative appearance.



Fig. 4D-F illustrate excellent correction of Lisfranc's joint with multiple arthrodeses.



Fig. 4B.

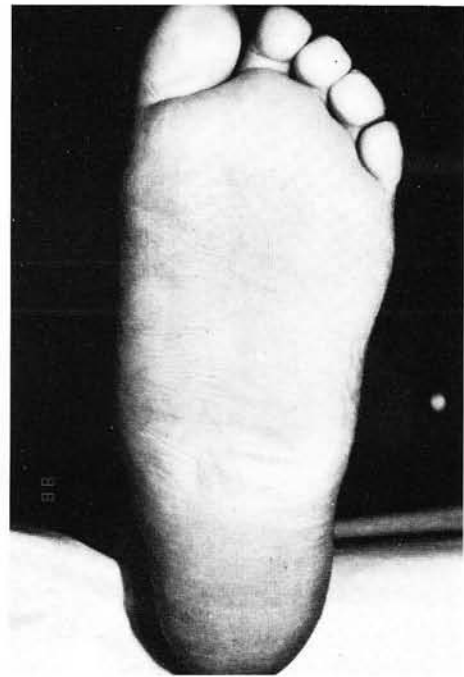


Fig. 4E.



Fig. 4C.

The surgical plan should include a realistic list of everything that may need to be done. If the amount of surgery appears excessive for either the patient or the surgeon then staging of the surgery may need to be considered. Often two teams of surgeons may be used where one team operates for the first four hours and a second and equally well-qualified team completes the surgery. There is nothing wrong with staging the surgery in two or three intervals if this appears safer for the patient or is more comfortable for the surgeon.



Fig. 4F.

tendon transfers to compensate for anterior muscle group weakness.

Digital surgery most often involves arthrodesis of the first through fourth proximal interphalangeal joints and arthroplasty of the fifth. Stabilization arthrodesis of the toes not only provides permanent correction of the alignment of the toes but additionally facilitates improved function of the metatarsophalangeal joints. The long and short flexors are much more effective in plantarflexion of the metatarsophalangeal joints after arthrodesis of the interphalangeal joints. The fifth toe should not be arthrodesed because of almost certain irritation from shoes. Improved alignment of the toes contributes greatly to prevention of mal perforans ulcers beneath the metatarsal heads.

Forefoot reconstruction commonly includes pan metatarsal head resections. It is imperative that resections be carefully planned then tested at surgery to be certain to maintain equal weight bearing beneath all bones. Pan metatarsal head resections, appropriately planned and combined with digital stabilizations, can do much to eliminate plantar ulcerations and mal perforans lesions (Fig. 5A-C).

Midfoot arthrodesis (Lisfranc's) is one of the more commonly required surgical reconstructions. It is also one of the more difficult, delicate, and time consuming ones. Fitting of the joints is as difficult as is the fixation. Getting all five metatarsal heads on the same transverse plane is essential, but tedious. Any patient undergoing midfoot arthrodesis should be evaluated carefully for ankle equinus. Unless equinus is corrected one can expect recurrent breakdown at Lisfranc's joint or at some other level (Fig. 4). Equinus must not be ignored.

Midfoot arthrodesis must frequently be combined with rearfoot arthrodesis and often with forefoot reconstruction and digital stabilization (Fig. 6A-D).

Rearfoot arthrodesis is necessary in those feet where collapse of the subtalar and midtarsal joints or parts of those joints has occurred. Often the navicular bone will have collapsed or dislocated. The entire midtarsal joint may be collapsed, or the talus may have collapsed through the calcaneus. Arthrodesis of the rearfoot complex including extensive bone grafting is often necessary to salvage the limb. Equinus is a considerable influence in the deformity and must be addressed. Rearfoot arthrodesis is commonly combined with digital and forefoot surgery as well as with midfoot arthrodesis. The combinations of multiple procedures requires knowledgeable and aggressive incisional planning and an orderly approach to instrumentation.

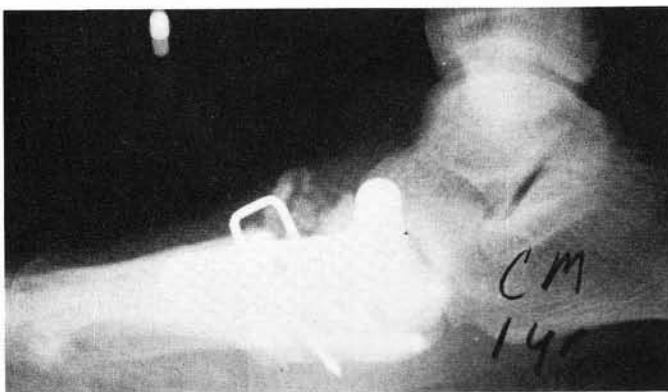


Fig 4G. Illustrates complete collapse of correction one year later. We now feel that such collapse is predictable if equinus is not addressed.

Where staging is used, it is important that key procedures be accomplished in appropriate order. There is little to be gained by reconstructing the forefoot or the toes if Lisfranc's joint is totally collapsed. Logic will dictate an appropriate order to the surgical stages.

Specific surgical procedures that are commonly required include digital stabilizations, forefoot reconstruction, midfoot stabilization, rearfoot arthrodesis, ankle repair or arthrodesis, correction or equinus, and muscle



Fig. 5A-C. Foot of 25 year old insulin-dependent diabetic female. She presented with large mal perforans ulcer beneath first and with near rigidus of first, second, and third metatarsophalangeal joints. Patient has previously undergone surgery on first metatarsophalangeal joint with subsequent development of hallux limitus and Charcot second and third metatarsophalangeal joints. Since earlier surgery, the patient had experienced recurring ulcerations at this site.



Fig. 5C.



Fig. 5B-C. Pan metatarsal head resections with planned length pattern restored near equal weightbearing beneath metatarsal heads.

Ankle repair may involve either bony or ligamentous considerations. Ligaments are commonly avulsed from bone and repair may involve primary repair by reattachment to bone. More often some bony collapse is involved. Not all unstable ankles require stabilization procedures. It depends greatly on the degree of instability, and it is also dependent on whether or not the patient

is expected to have a normal propulsive gait after surgery. In many instances patients already show motor neuropathy with resulting equinus. Lengthening of the Achilles tendon may be absolutely essential, yet to do so will likely result in a less propulsive or even an apropulsive gait. A moderate amount of ankle instability is tolerated well in patients who are relatively apropulsive.

Where there is frank collapse of the ankle joint, an ankle arthrodesis or pan talar arthrodesis may be necessary. If the ankle must be fused, it should be fused at as near a right angle as possible. Following surgery, a rocker sole should be ordered on the shoe to minimize bending and compression forces in the lesser tarsal and tarsometatarsal areas. Otherwise, the rigid ankle may encourage breakdown in a new area.

Equinus cannot be ignored. No one likes to lengthen the Achilles tendon on a patient already exhibiting motor neuropathy. Failure to lengthen this tendon can result in such devastation of the foot that one is left with little choice when equinus is present. The patient should be prepared in advance for strengthening exercises over a period of several months following cast removal. The patient should also be aware of the possible necessity for upright bracing should the triceps not respond to strengthening exercises. After all, the main concern is salvage of a limb, not the production of a cross country runner.

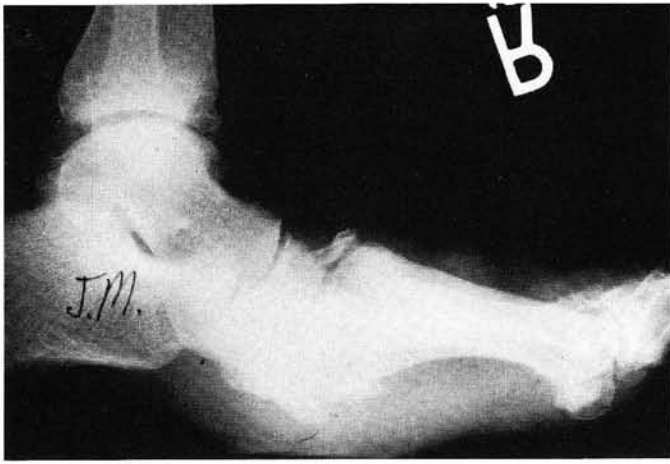


Fig. 6A-D. Sixty-eight year old insulin-dependent diabetic male (same patient as in Fig. 2). **A-B** illustrate degree of forefoot, midfoot, and rearfoot collapse. Patient also exhibits severe ankle equinus with -10 degrees of ankle dorsiflexion.



Fig 6B. Note second metatarsophalangeal joint collapse.

Muscle/tendon transfers are often combined with stabilizing operations. These are especially helpful where anterior leg muscle weakness has resulted in a dropfoot condition. Peroneus longus and tibialis posterior tendon transfers have generally worked well.

Postsurgical Management

Depending largely on the area of involvement and the extent of reconstruction carried out, postsurgical care can be rather simple and non-confining or it may be very complex and protracted.



Fig. 6C-D. At surgery bone was still too soft to allow screws to purchase adequately. Pins and staples provided adequate, though not good, internal fixation. Extensive autogenous bone graft from iliac crest was used to replace lost bone.

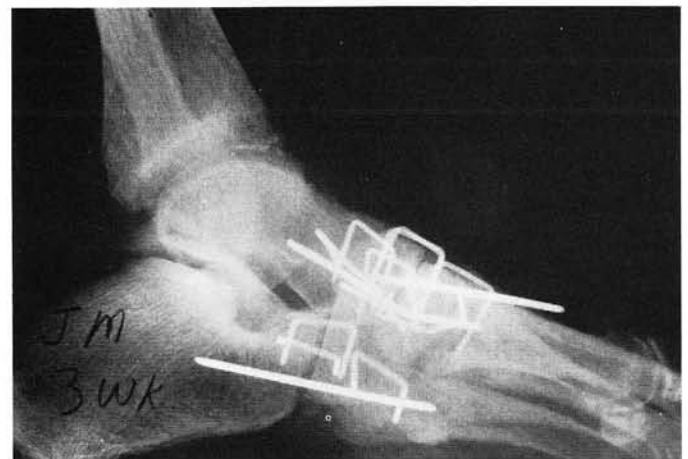


Fig. 6D.

Digital and forefoot reconstruction aftercare can usually be handled on a limited weight bearing basis. Kirschner wires typically protect the arthrodeses of the toes and the metatarsophalangeal joint alignment. A Darco trauma shoe is built up with a 1/2 inch thick insole all the way forward to the sulcus beneath the toes (Fig. 7). The insole is then cut abruptly to allow the toes to float over the end. Additionally, the toes are protected from bending force by molded splinting that is applied underneath the forefoot and toes after the dressing and bandage are in place. Splinting is held in place with additional kling bandage.

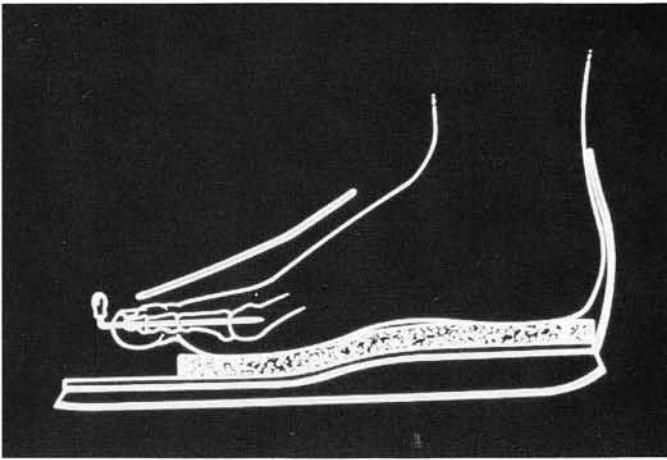


Fig. 7. Darco trauma shoe is built up with 1/2 inch insole to float end of toes and prevent dorsiflexion force against wires used to fixate toes and metatarsophalangeal joints.

The built-up insole protects the Kirschner wires from reactive force of the ground, while the splinting protects from the flexion force that results from loading the metatarsals. Both forms of protection are important to defend the wires against metal fatigue.

Arthrodesis of midfoot and rearfoot joints requires cast immobilization. Typically an above-the-knee cast is used for the first four to six weeks followed by below-knee casting for an additional six to twelve weeks. In both instances the cast is bivalved, and once the incision lines are well healed, the patient is encouraged to remove the cast and use a hot tub (carefully controlled) once or twice daily for 15 - 20 minutes. By this time only an ace bandage is used for mild compression and a second bandage holds the bivalved cast in place and facilitates easy removal (Fig. 8A-B).

At about two months postoperatively, an impression of the foot is made and a full length accommodative orthotic device ordered that can be used in a boot or depth shoe. Often it may be used in a high top padded basketball shoe. The device should be prepared and ready to use when the cast is discontinued.

Elastic bandaging should be encouraged for three to four months after the cast is removed, as should a high top shoe or boot. Elastic bandaging helps to control edema and the high top shoe helps to add stability to the weakened ankle.

Weight bearing should be avoided on both feet throughout the period of casting. By far the most common complication we have seen in the Charcot foot reconstruction has been collapse of the opposite foot. We now insist on a wheelchair throughout the casting



Fig. 8A-B. Bivalved cast is held together with 6-12 inch elastic bandage. Split cast facilitates removal for bathing and for gentle range of motion exercise.

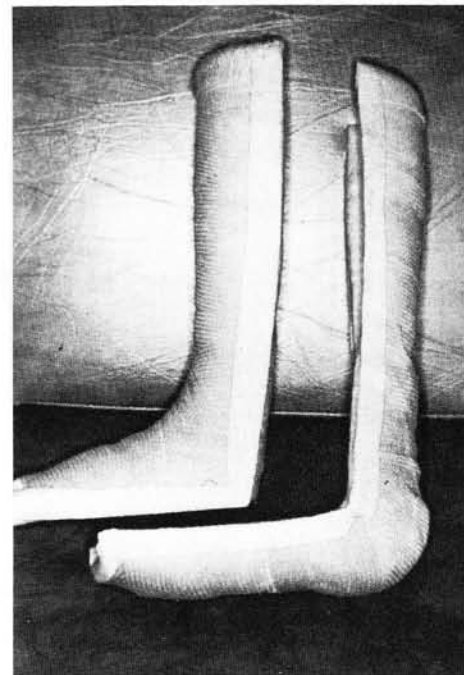


Fig. 8B.

period. When weight bearing is begun, it is with walker assistance. One graduates to crutches in a few weeks and to a cane on the affected side after approximately two months. Muscle strengthening exercises are carried out from the inception of weight bearing and for several months thereafter.

Ankle and pan talar arthrodesis may require a month or six weeks longer immobilization than is typical for other joints. This is largely because fusion of the ankle provides a new source of compression and bending force to the other joints of the foot as well as to the newly fused joints. One must, therefore, be quite certain of firm bony union before subjecting the joints to the bending and compression forces that will be generated. A rocker sole shoe is especially helpful in minimizing the compression and bending forces of the midtarsal and tarsometatarsal areas.

Muscle /tendon transfers require some special consideration. By six weeks post surgery, one can usually institute unresisted active and isometric exercises to strengthen the muscle. One must be certain that loading of the fused joints is averted at this early stage. Active unresisted exercises carried out several times daily can greatly facilitate muscle tone and speed convalescence.

CASE HISTORIES

The following are a sampling of cases from the 103 feet treated to date. They are chosen to represent the types of deformities one may encounter.

Case 1, Digital Charcot Joint

D.R. is a 40 year old non-insulin dependent diabetic male. He was first seen on referral by an infectious disease specialist after the patient had been hospitalized numerous times for debridement of the hallux interphalangeal joint, ostensibly for osteomyelitis. Yet no organisms were seen on gram stain and none were cultured.

Clinical examination showed moderate motor neuropathy.

Sensory and autonomic neuropathy were also apparent. Radiographs on that date demonstrated what could have easily represented either osteomyelitis or diabetic Charcot joint of the hallux interphalangeal joint (Fig. 9A). A plantar ulcer was present beneath the joint. Cultures were obtained but proved negative. The skin was warm over all the toes and capillary rebound was instantaneous.

The patient was placed on total non weight bearing for ten days and the wound healed completely.



Fig. 9A. Radiograph of Charcot hallux interphalangeal joint in 40 year old non-insulin dependent diabetic male. Patient had experienced recurring ulceration beneath joint.



Fig. 9B. Hallux interphalangeal joint arthrodesis followed by an accommodative orthosis resolved deformity and there have been no recurrent ulcerations at two years post surgery.



Fig. 10A-C. Represents 44 year old insulin-dependent diabetic female. **A.** Preoperative dorsoplantar view shows Charcot joint changes at third and fourth metatarsophalangeal joints and at first and second cuneometatarsal joints. First and second rays are unstable. Second toe had been amputated several years earlier.



Fig. 10B. Four months postoperative dorsoplantar radiograph.

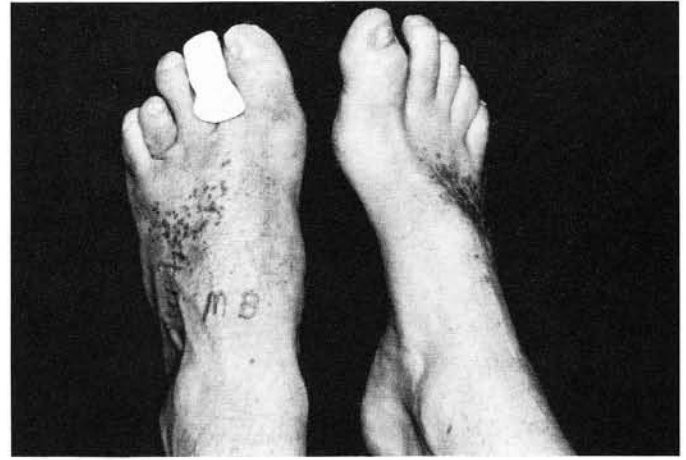


Fig. 10C. Dorsal view of foot at four months with silicone toe replacement.

Treatment consisted of arthrodesis of the hallux interphalangeal joint (Fig. 9B) followed six weeks later by a full length latex mold to equalize plantar stress. At two years post surgery the patient has had no recurrence of ulceration and has learned the art of proper daily inspection of his feet to permit early care for any area of irritation.

Case 2, Forefoot and Midfoot Charcot Joints

M.B. is a 44 year old insulin dependent diabetic female. Patient was first seen on referral from Dr. David Buchan, Columbus, Ohio (Fig. 10A-C). The patient complained of multiple forefoot deformities, primarily on the left foot though the right foot did demonstrate Charcot joint collapse of the hallux interphalangeal joint (Fig. 1).

Figure 10A demonstrates Charcot joint involvement at metatarsophalangeal joints three and four and at the first and second cuneometatarsal joints. The second toe had been removed several years earlier.

Clinical examination evidenced excellent peripheral circulation, but with dry scaly skin frequently associated with sympathetic neuropathy. Motor and sensory neuropathy were also in evidence.

Surgical treatment involved pan metatarsal head resections with a planned length pattern. The second was subsequently shortened slightly to compensate for its plantar protrusion after realignment (Fig. 10B). The middle two toes were arthrodesed and arthroplasty performed on the fifth. The first and second cuneometatarsal joints were arthrodesed in a realigned position. At four months post surgery the foot is seen to have satisfac-

tory realignment. Follow up treatment included an accommodative orthotic device and a silicone prosthetic toe (10C).

At one year follow up evaluation the patient appeared well controlled on the left foot and was planning surgical repair of the right great toe (Fig. 1).

Case 3, Rearfoot and Midfoot Charcot Joints

M.C. is a 34 year old insulin dependent diabetic female (Fig. 11A-G). Patient was first seen on referral by her endocrinologist. At that time she presented with a large plantar ulceration beneath the collapsed arch of the left foot. More than a year of limited weight bearing and plastizote insoles in inlay depth shoes had failed to show any progress with the mal perforans ulcer.

Clinical examination showed a vastly swollen, erythematous, indurated left foot. The plantar ulcer did not appear infected but showed a typical mal perforans base. Pulses were palpable and exceedingly strong bilaterally. Capillary rebound to the toes was instantaneous. Sensory perception was decreased and significant motor neuropathy was evident. Autonomic (sympathetic) neuropathy was also quite evident with dry scaly skin and dystrophic nails.

Range of motion examination of the foot showed total instability of the forefoot on the rearfoot. Ankle dorsiflexion was quite limited with most dorsiflexion occurring at the midfoot.

Radiographic examination showed collapse through the midtarsal, lesser tarsal, and tarsometatarsal joints (Fig. 11A, 11B).

The patient's left foot was treated by non weight bearing contact casts for nine months while attempting to get consolidation of the fractures. The ulcer healed quickly. The patient was limited to crutches with no weight on the left foot. Figure 11C shows collapse of the opposite foot which is the most common complication we have experienced in treating diabetic Charcot feet. The same neuropathy which was present in the left foot also placed the right foot at risk, and the same equinus (motor neuropathy related) which was present in the left foot provided the pathologic force in the right foot.

Following two months of total non-weight bearing, surgery was performed on the right foot. A tendo Achillis lengthening and mid tarsal and first ray arthrodesis were performed. Bony union appeared solid at three months,

but weight bearing resulted in fatigue fracture of the Steinmann pin at the naviculocuneiform joint indicating motion. The patient was returned to non-weight bearing status for six additional weeks. At five months postoperatively satisfactory healing is evident (Fig. 11 D-G).

This case illustrates only too well the necessity to be acutely aware of management of the opposite foot while treating a unilateral Charcot foot collapse. The opposite foot is usually at high risk. It is the opposite foot which has provided us with our most frequent complications in managing the Charcot foot.

Case 4, Rearfoot/midfoot Collapse

S.H. is a 49 year old insulin dependent diabetic female (Fig. 12A-F). The patient was first seen on referral from a regional diabetes hospital after nearly two years of unsuccessful attempts at conservative treatment with accommodative orthoses and depth shoes, with casts, and finally with 9 months of wheelchair confinement. The patient indicated that any attempt at weight bearing caused pain and recurrent swelling. Pain is not a usual complaint of the patient with Charcot foot deformity.

Clinical examination demonstrated advanced polyneuropathy with motor and sensory being particularly severe. Autonomic (sympathetic) neuropathy was also evidenced by warm feet with dry scaly skin.

Radiographic examination confirmed Charcot joint collapse of the first ray. The first cuneiform had avulsed free from the second and had likewise avulsed a large section of the navicular bone (Fig. 12A-B).

The patient was confined to a wheelchair for six additional weeks to allow full equilibration with the opposite foot. Surgery was then performed to stabilize both the rearfoot and mid foot. A tendo Achillis lengthening was also performed. A clinical view of the foot three days postoperatively shows the rather benign edema that is typical for patients with sympathetic neuropathy following major reconstructive surgery (Fig. 12C). A clinical view four months later shows the patient shortly after resuming weight bearing with the use of a walker (Fig. 12D). The walker was continued for an additional month to ease the strain of weight bearing on both feet. Accommodative orthoses and depth oxfords with 1 inch heels were used so as to lessen stress to the opposite foot from triceps equinus.

Figures 12E and F demonstrate satisfactory union with some improvement in osteopenia. Patient was immobi-

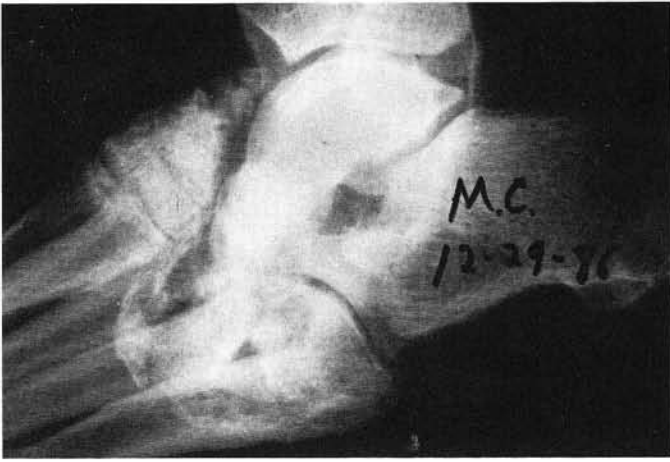


Fig. 11A-G. A and B. Show Charcot foot collapse on left foot of 34 year old insulin dependent diabetic female.



Fig. 11C. Shows collapse of opposite foot which occurred while protecting left foot from weight bearing.



Fig. 11D-G. Show right foot five months after stabilization arthrodesis of mid-tarsal and first ray joints and Achilles lengthening. Early weight bearing caused fatigue of Steinmann pin with fracture. This necessitated an extra six weeks of non-weight bearing. Total non weight bearing was four and one-half months.

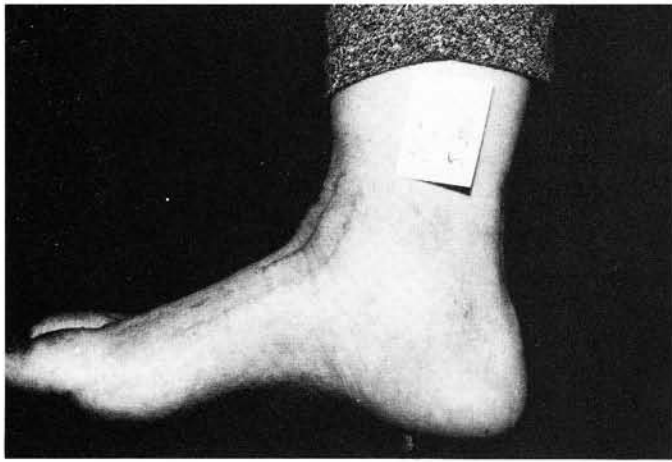


Fig. 11E.



Fig. 11F.

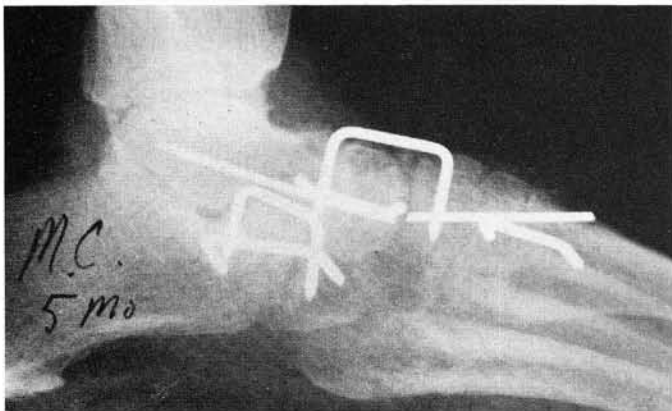


Fig. 11G.

lized in a bivalved cast and was allowed to remove the foot and leg for exercise throughout recovery. This may account for the improvement in osteoporosis.

Because of weakness related to lengthening of the triceps the patient still required a cane at six months after surgery. She is on an exercise program to strengthen the weakened triceps muscle, but it is uncertain the degree to which it will recover.

Regardless of whether the triceps recovers satisfactory strength it was necessary that the Achilles be lengthened. To have ignored this need would have simply resulted in the foot breaking somewhere else when weight bearing was resumed.

RESULTS

At this writing 103 feet have undergone reconstruction for Charcot foot collapse at the Podiatry Institute. Thus far we have not lost a single toe or foot. We are keenly aware of the high risk patient with whom we are dealing, and we attempt to make each patient aware that should the surgery fail they would likely go on to below knee amputation. For most patients this is what had been proposed by others before being referred to The Podiatry Institute.

We have been impressed with the speed of healing in this series of patients. This is true of bone and soft tissue, but is more dramatic as regards soft tissue. Post surgical edema and inflammation are minimal compared to the average non diabetic patient. Bone healing appears rapid as well but is more difficult to measure since the surgery performed is far more traumatic than is usually necessary on the non Charcot foot.

We feel that a key concept to remember is that the autonomic (sympathetic) neuropathy which caused osteopenia to begin with will continue to be present. The same vasomotor loss which causes osteopenia will continue. One must, therefore, be on constant guard against stresses that can result in new areas of collapse.



Fig. 12A-F. Presents 49 year old insulin dependent diabetic female. **A-B.** Left foot is seen two years after Charcot foot collapse. Patient has been unable to tolerate resumption of weightbearing for more than a few steps due to unusual pain and edema. First ray is totally unstable and has avulsed navicular along with first cuneiform. Midtarsal joint is also unstable but is not fractured.



Fig. 12C. Clinical appearance three days after rearfoot and first ray stabilization arthrodesis and tendo Achillis lengthening. Note rather excellent appearance of foot for three days postoperative.

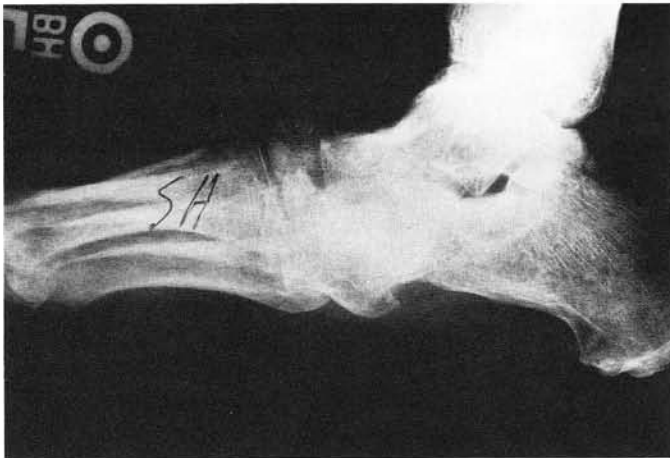


Fig. 12B.



Fig. 12D. Clinical appearance of the foot at four months after surgery.



Fig. 12E-F. Radiographic evidence of solid bony union, one month after removal of cast. Note some minor improvement in osteopenia.

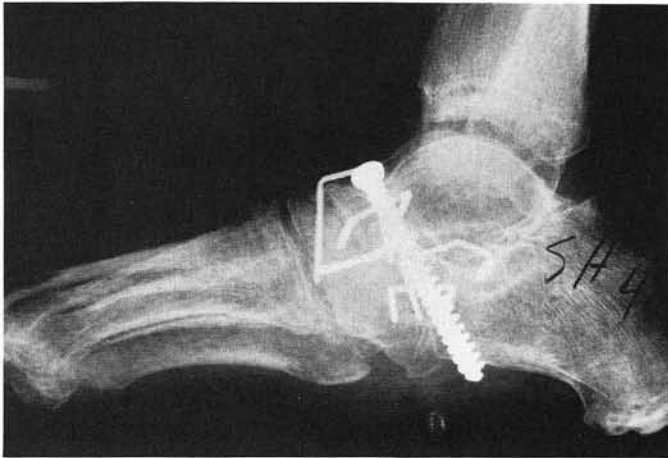


Fig. 12F.

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