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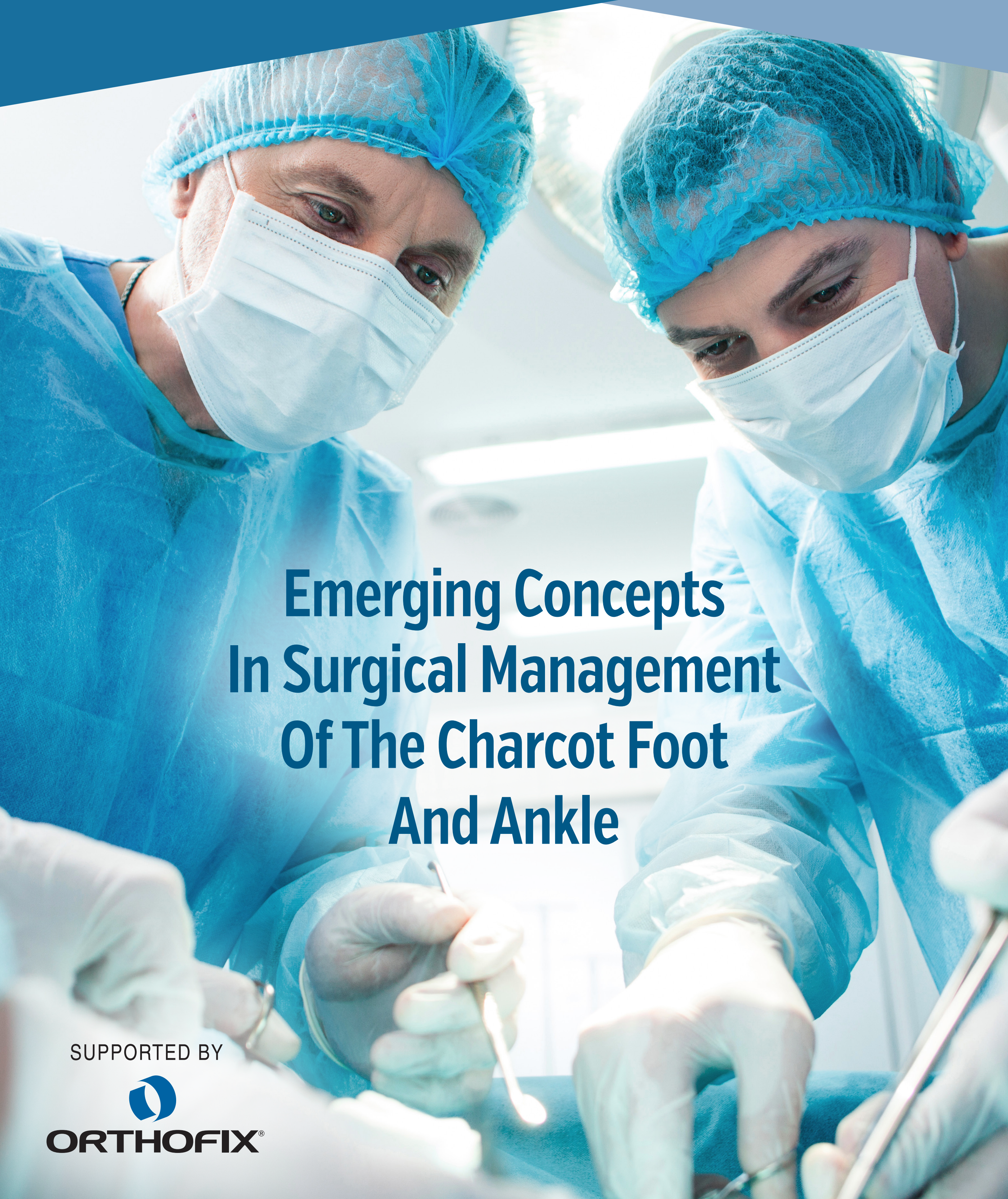

Emerging Concepts In Surgical Management Of The Charcot Foot And Ankle

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When Patients Have Charcot Osteoarthropathy And Osteomyelitis

Given the challenges of treating this patient population, these authors discuss principles of diagnostic testing and pertinent factors in formulating an effective treatment algorithm.

By Peter A. Blume , DPM, FACFAS, and Ryan J. Donegan, DPM, MS, AACFAS

Charcot osteoarthropathy was first described in 1883 and remains a poorly understood and frequently overlooked complication of diabetes.[1] Recognition in the earliest stage is problematic as many cases are misdiagnosed. An estimated 7 percent of the United States population has diabetes and along with the increased life expectancy of this population, diabetes-associated complications such as foot ulcerations, peripheral arterial disease, infections and Charcot osteoarthropathy are increasing in prevalence.[2,3]

Charcot osteoarthropathy is a relatively painless, progressive and degenerative arthropathy of a single or multiple joints caused by underlying neurologic deficits, most commonly affecting peripheral joints. Current estimates of prevalence range from .08 percent in the general diabetic population to 13 percent in high-risk diabetic patients.[4] Charcot osteoarthropathy usually occurs eight to 12 years after the diagnosis of diabetes, occurs more frequently in men during the fifth and sixth decades, and has recurrence rates between 12 to 33 percent.[5-7]

Diabetic patients with Charcot osteoarthropathy are complex patients with many comorbidities. When severe infection is concurrent, morbidity and mortality rates can be as high as 35 percent, even when there is appropriate management of the infection.[8] In contrast to Charcot osteoarthropathy, osteomyelitis itself is an infection in the bone. People who have diabetes most often develop osteomyelitis in their feet as a result of foot ulcers.[9] A team-based approach, including hospitalists, vascular specialists and infectious disease physicians in addition to foot and ankle surgeons, is critical in providing the most successful outcomes for this at-risk population.

Diagnosing Charcot Osteoarthropathy

Charcot osteoarthropathy is a diagnosis by clinical examination. One should use imaging to stage and supplement evaluation of the progression of the condition. In practical clinical application, there are acute and chronic stages of Charcot osteoarthropathy.[10-14] In the patient with acute-stage Charcot, osteoarthropathy and osteomyelitis are extremely difficult to diagnose when they occur concurrently as they appear to have similar presentations both clinically and with imaging modalities. As with any pathology, clinicians would use a stepwise process to obtain an accurate diagnosis, leading to correct treatment.

While a history of infections and open wounds can increase suspicion of osteomyelitis, this does not exclude a concomitant Charcot osteoarthropathy process just as unremarkable clinical tests do not exclude infection. One can employ laboratory testing for affirming as well as monitoring treatment. Advanced imaging also plays a role in the difficult task of differentiation between Charcot osteoarthropathy and osteomyelitis. Magnetic resonance imaging (MRI) allows simultaneous evaluation of soft tissue and osseous structures as well as defining the anatomic location with good accuracy and localization.[15]

However, differentiating between acute Charcot osteoarthropathy and osteomyelitis is difficult due to similar signal intensity changes.[16] Bone scintigraphy is highly sensitive but lacks specificity in the diagnosis of Charcot osteoarthropathy.[17] Clinicians mainly use bone scintigraphy to rule out osteomyelitis in diabetic patients with open wounds and the use of leukocyte-labeled bone scans offers a distinct advantage over MRI in patients with metal implants. With bone scans, there is no artifact generated from imaging of metal implants but this not the case with MRI as the artifact can obscure imaging results.

Although controversy has emerged concerning the accuracy of the "gold standard" bone biopsy, researchers still recommend the modality and withholding antibiotics for 48 hours prior to culturing.[18] Lavery and colleagues report 95 percent sensitivity and 99 percent specificity for osteomyelitis with a mean of 1.6 isolates per patient, and *Staphylococcus aureus* (33 percent) and *Enterococci* (12 percent) being the most common isolates.[19] The table "A Closer Look At Diagnostic Imaging Studies" on page 5 offers a summary of relevant imaging studies.

Addressing Perfusion, Osteomyelitis, Wound Coverage And Reconstruction

When formulating a treatment algorithm for Charcot osteoarthropathy with osteomyelitis, it is imperative to address all factors that may have an effect on the outcome. The goal is as close to full erad-

Here one can see radical resection of infected bone.

ication of osteomyelitis before final reconstruction takes place. During the initial treatment of osteomyelitis, assessment of perfusion is critical as this is ultimately the most important factor for a successful outcome. One needs to close wounds, manage tissue deficits and address osseous instabilities and areas prone to breakdown. The overall strategy for surgically managing a severe diabetic foot infection is infection control through aggressive and extensive surgical debridement, a comprehensive vascular assessment with possible vascular surgery and/or endovascular intervention, and soft tissue and skeletal reconstruction after the eradication of infection to obtain wound closure and limb salvage.

The need for adequate perfusion is obvious. Many times, limb salvage requires a combination of infection management, wound closure and surgical reconstruction. All three of these factors are dependent upon the perfusion of the lower extremity, allowing for adequate antibiotic delivery and osseous and skin healing. Perfusion should be greater than 30 mm Hg because lower values of arterial perfusion are associated with impaired wound healing.[25]

If the patient has inadequate perfusion, you need to work closely with a vascular or interventional radiologist. Do not assume patients with Charcot osteoarthropathy have proficient perfusion. The ankle-brachial index measurement is considered the most accurate noninvasive diagnostic method for evaluating peripheral arterial disease (PAD).[26] This provides a quantitative evaluation of distal flow. In contrast, angiography can provide a definitive diagnosis of PAD by showing a road map of the arteries.[8] Revascularization should be angiosome-directed and with improved techniques such as retrograde endovascular approaches, even small distal arteries are now accessible.

A severe diabetic foot infection carries a 25 percent risk of major amputation and one should involve infectious disease specialists as quickly as possible.[27] Antibiotics do not penetrate devascularized bone. Therefore, adequate surgical debridement, in addition to antimicrobial therapy, is necessary to cure chronic osteomyelitis. The length of treatment for osteomyelitis depends upon clean margins as well as culture positive and culture negative specimens. The standard recommendation for treating chronic osteomyelitis is six weeks of tailored parenteral antibiotic therapy.[28]

A Closer Look At Diagnostic Imaging Studies

Study	Results	Conclusions
SPECT/CT coupled with bedside percutaneous bone biopsy when positive scan obtained.[20]	Sensitivity and specificity for combined method 88.0 percent and 93.6 percent respectively. Positive predictive value (PPV) and negative predictive value (NPV) of 91.7 percent and 90.7 percent respectively.	Coupling of 67Ga SPECT/CT imaging and bedside percutaneous bone puncture accurate for diagnosing diabetic foot osteomyelitis
Suspected osteomyelitis or exacerbation of known osteomyelitis investigated with CT and SPECT/CT.[21]	Sensitivity, specificity and accuracy for CT of 77, 86, and 79 percent. For SPECT/CT, sensitivity, specificity and accuracy of 100, 86, and 98 percent.	SPECT/CT significantly more accurate compared with CT
T1-weighted MRI features associated with diabetic pedal osteomyelitis present in histologically proven cases non-pedal osteomyelitis.[22]	93 percent of cases demonstrated T1-weighted imaging features typical of pedal osteomyelitis with confluent region of decreased signal intensity, hypointense or isointense, relative to skeletal muscle in a geographic pattern with medullary distribution	Cases that did not demonstrate typical T1-weighted features predominantly secondary to hematologic mechanism of infection
Investigated FDG PET/CT for diagnosis of osteomyelitis in the diabetic foot.[23]	FDG PET/CT sensitivity, specificity and accuracy of 100, 92, and 95 percent in a patient-based analysis and 100, 93, and 96 percent in lesion-based analysis	Foci sites of acute infection precisely localized with PET/CT allowing correct differentiation between osteomyelitis and soft-tissue infection
Investigated bone scintigraphy to MRI for detecting osseous lesions.[24]	Inflammatory lesions were detected in 74.1 percent of symptomatic regions by bone scintigraphy and 98.1 percent of symptomatic regions by MRI. Sensitivity of MRI compared to bone scintigraphy was superior in detecting lesions in the long bones of the thigh and the lower legs (100 percent vs 78.4 percent respectively).	MRI rather than plantar bone scintigraphy for detection of chronic osteomyelitis

The Reconstructive Ladder For Wound Closure

Free Flap
Tissue Expansion
Pedicle Flap
Local Flap
Skin Graft
Dermal Matrices
Negative-Pressure Wound Therapy
Closure by Secondary Intention
Primary Closure

Ascending the ladder, closure becomes more technically difficult and morbidities are increased. One should choose a closure technique that provides the least morbidity and most durability, but this does not always involve starting at the lower steps.

Note the reconstruction of the deformity to prevent recurrent wounds. Resection of bone, the exchange of bone cement, bone grafting, osteotomies and arthrodesis are all options to achieve a stable foot.

However, oral antibiotics have now become available that achieve adequate levels in bone, achieving similar cure rates. Antibiotic-loaded bone cement represents another antibiotic delivery vehicle, ideally providing antibiotic delivery while simultaneously contributing to the process of bone regeneration.[29]

Diabetic foot and ankle reconstruction closure requires a thorough knowledge of flap and grafting techniques. One must be vigilant with appropriate patient selection and a thorough workup prior to surgery will assist in obtaining optimal results. If there is any question about the patient's vascular status, angiogram and ankle-brachial index (ABI) are crucial, and one can utilize these findings with angiosome principles to plan flaps and closures. The goal of wound healing is to obtain the best closure through the least morbid means. The decision for wound closure depends on the location of the wound and host factors (i.e. tissue extensibility and the individual's healing potential).

Adjunctive therapy with dermal matrices and other biologics, along with negative-pressure wound therapy (NPWT), have played a large role in reducing the need for more involved flaps. Still, large deficits of tissue, exposed bone/tendon and plantar weightbearing wounds do not have good outcomes with skin grafting. Successful closure requires the removal of biofilm and a vascularized granular wound bed along with the prevention of seroma and sheer forces. Orthofix flap frames with quick adjust struts ideally combine the rigidity and protection required while simultaneously allowing easy access. See "The Reconstructive Ladder For Wound Closure" above.

When Charcot osteoarthropathy is in the presence of an open wound, a stepwise approach is required. The first step involves radical resection of clinically infected bone (see photo on page 5). Tissue cultures from the resected bone guide antibiotic therapy, involving any combination of intravenous, oral and implantable bone cement/antibiotic-loaded beads/bone void filler with antibiotics. After clearance of osteomyelitis, the focus becomes reconstruction of the foot/ankle in a stable plantigrade position. Resection of bone, exchange of bone cement, bone grafting, osteotomies and arthrodesis are all available to achieve a stable foot (see above radiograph). Surgeons can also employ soft tissue balancing and gradual correction with Orthofix hexapod frames when long-standing deformities, chronic soft tissue contractures and peripheral scarring of the neurovascular bundle are present.

There are many different ways of maintaining deformity correction in reconstruction. Surgeons may use Steinmann pins to maintain position, achieve compression with external fixation through midfoot, hindfoot and/or ankle joints, and achieve stabilization with beaming bolts and dual-purpose antibiotic-coated intramedullary nails (see left photo on page 7). These are all viable options. One would usually maintain fixation for a period of eight weeks in foot deformities and a minimum of 12 weeks when the ankle is involved.

The final outcome should be a limb

Note the use of external fixation to maintain reconstruction.

The final outcome should be a limb with all biomechanical factors addressed in order to provide a functional, plantigrade, wound-free limb.

with all biomechanical factors addressed to provide a functional, plantigrade, wound-free limb done in an economically responsible way (see right photo above). Using this protocol, Pinzur and colleagues were able to achieve 95.7 percent limb salvage with ambulation in commercially available therapeutic footwear.[30]

Maximizing Outcomes

Diabetic patients with Charcot osteoarthropathy are complex patients with many comorbidities other than osteomyelitis. A proactive, cooperative, co-management model for the perioperative management of high-risk patients undergoing complex surgery can improve the quality and efficiency metrics associated with the delivery of service to these complicated patients.[31]

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Gradual Deformity Correction In The Charcot Foot

Citing the merits of gradual correction of Charcot deformities, this author emphasizes accurate assessment of the deformity's magnitude, a strong awareness of at-risk structures and appropriate ex fix selection to help achieve optimal outcomes.

By Philip Wrotslavsky, DPM, FACFAS

Gradual correction of foot and ankle deformities is a topic that is not often discussed. The decision of when to perform gradual correction in the foot requires a thorough understanding of deformity correction. When the surgeon is faced with a complex disease process such as Charcot foot in addition to a multiplanar deformity, one can appreciate the difficulty in choosing the appropriate procedure. Once one becomes aware of the magnitude of deformity associated with the Charcot foot and the actual amount of correction needed to obtain a stable foot, then the decision regarding acute versus gradual correction can be appreciated.

There is controversy about when to proceed with surgery, the type of correction and fixation constructs. In the literature, researchers have reported a failure rate of as much as 50 to 80 percent for Charcot foot reconstructions.[1] While it was previously believed that the Charcot foot had "bounding pulses" and more than adequate blood flow, Wukich and colleagues recently noted a 40 percent prevalence of peripheral arterial disease (PAD) in patients with diabetic Charcot neuroarthropathy.[2]

Previous authors have postulated that an acute deformity correction of the Charcot foot may lead to complications of ischemia. To prevent ischemia in the Charcot limb, multiple authors have described a two-stage approach to Charcot reconstruction involving gradual correction of the deformity followed by internal fixation.[3-5] The authors believe that gradual correction allows for accurate deformity correction while providing less risk to neurovascular structures. In my opinion, not only does there need to be a concern during an acute correction for arterial structure compromise but the physician should also take venous congestion, skin stretching and even nerve structures (even though we are dealing with a neuropathic foot) into consideration. One can make an analogy to the field of orthodontics and braces. No one would find it acceptable to acutely correct misaligned teeth. That is why braces are applied and the teeth are gradually corrected. Similarly, with the Charcot foot, a combination of equinus, shortening, translation, angular deformities and rotation of the foot will more often than not add up to a much larger deformity than originally perceived by the surgeon.

When surgically correcting the deformity present in a Charcot foot, there are some basic principles. First, one must recognize the Achilles is contracted and in equinus.

Magnitude Of Deformity In The Charcot Foot: How Much Correction Is Necessary?

These are the basic principles to keep in mind when surgically correcting deformities that are present in the Charcot foot. First, one must recognize the Achilles is contracted and in equinus (see photo at the left).[6] One must always surgically address the equinus deformity. Then the surgeon would need to correct and fuse the medial column in order to create a stable foot. The increased glycosylation will change the biology of the fibers in the Achilles, causing it to contract and the plantar ligaments of the foot to weaken, thus causing a breakdown of the midfoot.[7,8] The main lever arm of the foot will stress the talonavicular joint, naviculocuneiform joint and the cuneiforms to metatarsal joints. Radiographically, one will more often see a large break in the Meary's angle than a drop in the calcaneal inclination angle (see top photo on page 9). Lamm reports the normal Meary's angle is 4 degrees in a cavus position.[9] I see an average of 25

Radiographically, one will more often see a large break in the Meary's angle than a drop in the calcaneal inclination angle (top). The author has treated patients that had as large as 52.5 degrees of Meary's angle break (bottom).

Acute Versus Gradual Correction: Advantages And Disadvantages

Acute Correction	Gradual Correction
Best for small- to medium-sized deformities	Large deformities can be corrected
Better in the femur and humerus	Can use computer-assisted software to attain exact correction
Internal or external fixation	If surgeon is not satisfied with the position, he or she can run a new program
Lengthening is not typically possible	Works well in tibia/ankle region, where there is high risk of nerve issue with acute deformity
No ability to make adjustments	Works well if patient has poor soft tissue
	Requires external fixation and patient to adjust frame
	Slow correction gives skin and neurovasculature the ability to stretch

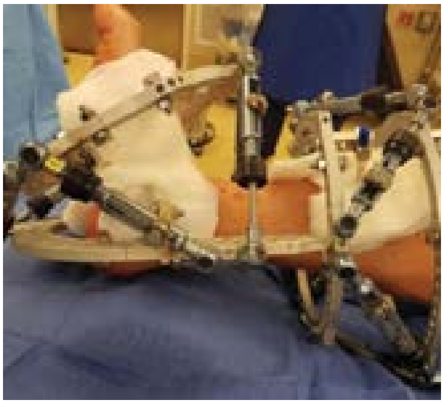
degree Meary's angle deformity, which puts the foot in a rocker bottom position, thus making the medial column break 29 degrees. I have treated patients that had as large as 52.5 degrees of Meary's break (see bottom image above).

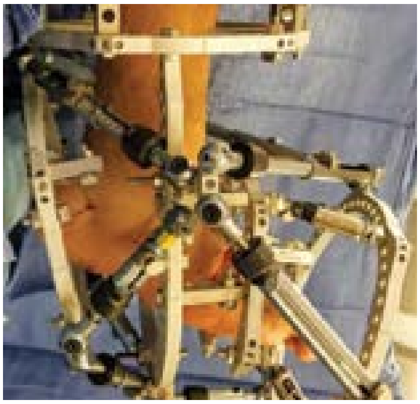
A way to simplify the correction goals in the Charcot foot is by trying to create a tripod stand with the foot. The calcaneus is one leg of the tripod and the other legs are located under the first and fifth metatarsal heads. In order to create that tripod, one must stabilize the medial column, which will subsequently drive the lateral column into a more optimal position.

After evaluating the equinus component and the medial column, the surgeon needs to consider any shortening of the foot. In cases in which the forefoot is subluxed onto the hindfoot, one needs to measure the amount of subluxation to see how many centimeters one will need to pull out to lengthen the foot. Usually by the time the surgeon gets the patient to the OR, the overlapping bones become stuck in the shortened position, which prohibits acute correction.

Transverse deformities can contribute heavily to the overall magnitude as an adducted or abducted foot can cause

The percutaneous Gigli saw osteotomy allows for a through-and-through midfoot, hindfoot or ankle osteotomy without having large skin incisions.



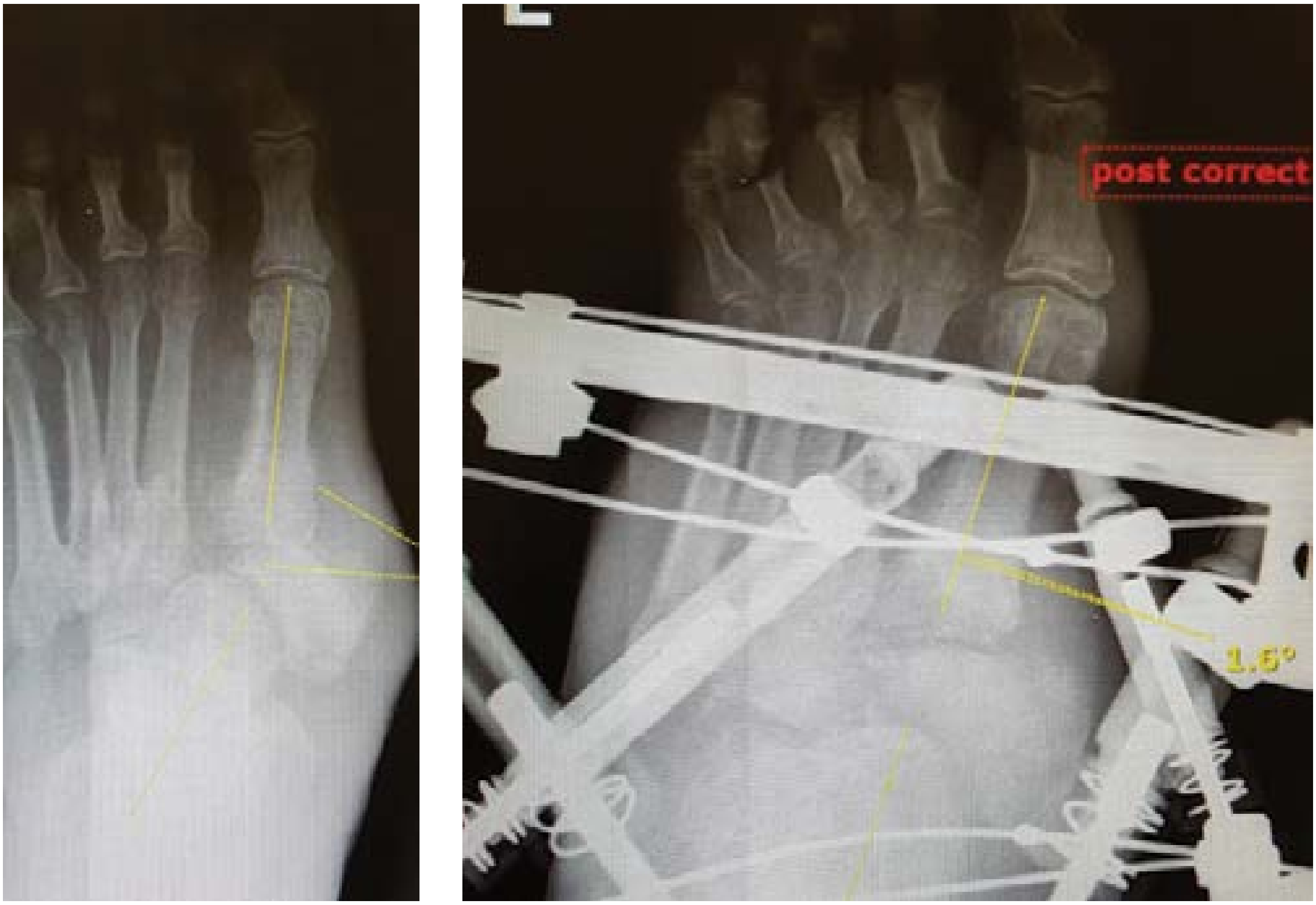


The butt frame (left) allows for the correction of forefoot deformities in any direction. The miter frame (middle) and the 6+6 construct (right) allow for simlutaneous correction of a combination of forefoot and hindfoot deformity.

foot shortening. When performing an adduction to rectus correction, one must be careful of the medial skin structures. The skin on the lateral aspect of the foot is the concern when correcting a large abductus deformity.

The next two deformities are more subtle but do not be fooled because they are still part of the overall magnitude of deformity. I will first discuss translation. With the goal of fusing the medial column, the surgeon must attain alignment in the dorsal/plantar view as well as in the lateral view. The surgeon must be able to bisect the first metatarsal all the way through the talus in both views, especially if one is to beam the foot. Otherwise, the beaming screw will miss. If one is plating and the first metatarsal is too medial, the plate will not fit. Aligning the mechanical axis of the foot will enable better fixation and function.

The other subtle deformity is a frontal plane rotation. Too often with an acute correction, the surgeon does a great job correcting the equinus, transverse, lateral and translational deformities. However, if one does not address the frontal plane, the patient will end up walking on the outside or inside of the foot, causing a



Here is pre-op AP view showing a 25 degree talo-first metatarsal angle with 2 cm medial translational shift of the first metatarsal (left) and a post-op AP view (right) showing reduction of the talo-first metatarsal angle to 1.6 degrees with reduction of translation.

new set of ulcers.

Grant and coworkers caution against the rote use of medial-based wedges.[10-12] These wedges have limited indications and may accentuate deformity in patients who have been selected inappropriately. Their results show that medial-based wedges in patients with even small degrees of hindfoot varus produced significant accentuation of hindfoot varus.

Forefoot to hindfoot position is critical when preparing for medial column fusion as any misalignment can create varus deformities and compromise one's fixation.

Being Aware Of The Structures At Risk

Ultimately, if performing an acute correction, the surgeon needs to add up all these deformities and calculate how much he or she is stretching the foot. In other terms, one will need to calculate how much to shorten the foot in order to avoid compromising any at-risk structures. If the surgeon corrects the deformity too quickly, the skin and vasculature are at risk for necrosis. While no two deformities are the same, in the Charcot foot, the structures typically at risk are the dorsal and medial skin, and the dorsal vasculature so a rocker to rectus correction will put the dorsalis pedis and dorsal skin at risk. An adductus to rectus correction will compromise the medial skin. A large equinus correction can risk the posterior tibial vasculature and a varus to valgus ankle correction compromises the medial skin and posterior tibial artery.

Weighing Acute Versus Gradual Correction

Once the surgeon has measured all the deformity parameters, the next step is to decide if acute or gradual correction is the proper method of correcting the Charcot foot. Using Herzenberg and Paley's planning techniques, the surgeon can apply either the law of concentric circles or the law of similar triangles to decide on the rate of correction.[13,14] One will also need to apply the formula described by Paley using the law of sines to see how much lengthening is obtained when derotating the forefoot out of a supinatus position. Without adding up all the deformity parameters before performing an acute correction, the surgeon is just guessing as to how much of a bone wedge to remove without risking the neurovascular structures and skin. This is similar to a surgeon measuring the intermetatarsal angle prior to a bunion surgery to decide where to perform the osteotomy. Therefore, it is incumbent upon the surgeon to do the same when performing a Charcot correction.

The beauty of using a hexapod computer-assisted external fixator is that one has more flexibility when correcting the multiplanar deformities that are present in the Charcot foot. The surgeon does not need to perform the aforementioned complex math equations because the computer software does that for you. Using a gradual approach can be a safer, more accurate and reproducible method in correcting the Charcot foot. You have the ability to slowly correct the deformities so the at-risk structures can adapt. The capability to run residual program corrections allows for the surgeon to address any new deformities that were unmasked during the correction. One can correct large deformities without having to perform shortening osteotomies.

External fixation can have minor drawbacks that have simple solutions. Proper education of the patient about why gradual correction is of the utmost importance. Patients who cannot perform the turns of the struts for various reasons can have the assistance of a home nursing organization to help. Weekly follow-ups with X-rays are essential to see if the program is going accordingly. A second procedure to remove the frame with the insertion of an internal fixation is part of the protocol.

Choosing The Right External Fixation

Now that the surgeon has calculated the magnitude and center of rotation of angulation (CORA) of the deformity, there are the decisions of where and how to perform the osteotomy.

I prefer to perform a percutaneous Gigli saw osteotomy (see top photo on page 10). This technique allows for a through-and-through midfoot, hindfoot or ankle osteotomy without having large skin incisions.[15,16]

Prior to performing the osteotomy, the surgeon needs to decide on the type of external fixator. With the Charcot foot, there are two constructs that will correct the usual deformities associated with Charcot. First is the butt frame (see bottom left photo on page 10), which is based upon a butt joint in carpentry. It allows for the correction of forefoot deformities in any direction. There are two other constructs that allow for simultaneous correction of a combination of forefoot and hindfoot deformity. These constructs are the miter frame (see bottom middle photo on page 10) and the 6+6 (see bottom right photo on page 10). The 6+6 construct is a variant of the Butt frame that places a ring anterior and posterior to the butt joint, creating two separate frames on one. The miter frame is based off the concept of a miter joint in carpentry in which there is a 45-degree angle connecting two straight items that are going at a 90-degree angle.

In the case of the Charcot foot, the posterior portion of the miter frame can address hindfoot and ankle deformities, and the distal portion can separately and simultaneously address forefoot deformities. It is not within the scope of this article to delve into the nuances of six axis computer-assisted deformity correction and all its intricacies. However, I do need to note a major point. The advantages of using gradual correction with a hexapod fixator is that one has the ability to make adjustments during the correction process, unlike an acute correction, which locks you into the original position of correction. With computer-assisted hexapod correction, if you unmask a deformity during the correction process, you have the ability to run residual programs that allow the surgeon to correct any deformity in any direction.

In Conclusion

Correcting the Charcot foot is not a simple surgery that involves randomly taking out wedges of bone to realign the foot. One must be aware that the Charcot foot is a much larger deformity than what we see on X-ray. It is imperative to measure all appropriate angles and ascertain the magnitude of the deformity prior to deciding upon what procedure to use. In my opinion, gradual deformity correction of the Charcot foot is the preferred method of obtaining correction in the foot with a large deformity. It is safe, reproducible and accurate. ■

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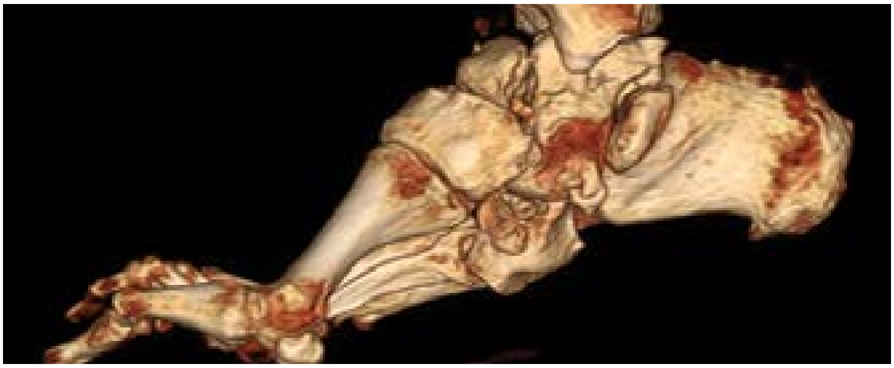
Advanced Concepts In The Beaming Of The Charcot Foot

Discussing the inherent challenges with the etiology of the Charcot foot, these authors advocate the use of Root biomechanical principles to facilitate a sound surgical plan and offer their recommendations for beaming in reconstructive surgery.

By William P. Grant, DPM, FACFAS, Bryan Barbato, BS, Lisa Grant-McDonald, DPM, Jeffrey Yates, BS, and Alexander Webb, BS

Charcot is a disease characterized by increased local bone resorption by osteoclasts. The receptor activator of nuclear factor-kappa-B ligand (RANKL) is an integral component in the regulation of osteoclast differentiation and activation. The RANKL induces the activation and differentiation of osteoclasts by binding to the osteoclasts' RANK.[1] Both RANK and RANKL are expressed constitutively. The RANKL overproduction is a characteristic of Charcot but it is not limited to Charcot. It also occurs in many bone diseases such as psoriatic arthritis, rheumatoid arthritis and osteoporosis.[2]



Here is a preoperative 3D CT showing a Charcot midfoot with a plantar ulcer beneath the cuboid. Note that the navicular sits superior to the talus with complete dislocation but no fracture. Also note the severe stacking of the metatarsals.

In a study involving three patient groups, Mabilleau and colleagues compared monocyte formation into osteoclasts and osteoclastic activity in vitro with and without the addition of RANKL.[3] The groups included patients with diabetic Charcot neuroarthropathy, healthy patients and patients with diabetes. Without the addition of RANKL, researchers noted a significant increase in osteoclast formation in the Charcot group in comparison with the healthy and control groups. There was also increased osteoclastic activity in the Charcot group in comparison with the others.

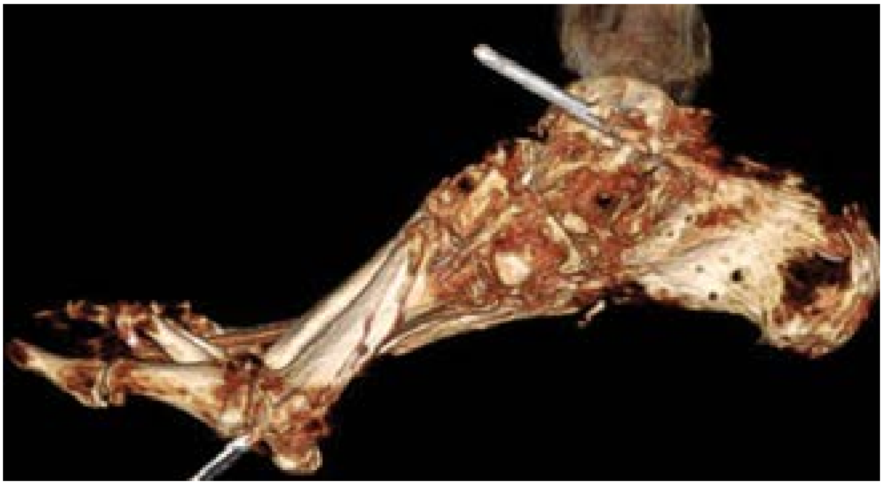
With the presence of RANKL, the study authors noted an increase in osteoclastic activity in all three of the groups.[3] However, osteoclastic activity was considerably more aggressive in the Charcot neuroarthropathy group and was four times greater than the osteoclastic activity in the healthy group. Osteoclasts in patients with Charcot neuroarthropathy differentiate to become highly active.[4]

A Closer Look At How AGE And RAGE Affect The Formation Of Charcot

The formation of advanced glycation end products (AGEs) is a common consequence of aging. Increased AGE production occurs in patients with prolonged elevated blood glucose levels, frequently termed hyperglycemia. Advanced glycation end products modify N-carboxymethyl-lysine of type I collagen (CML collagen).[5] The post-translational modification of the CML collagen occurs by non-enzymatic glycosylation, termed glycation. This primarily occurs in tissues with a slow turnover rate, exposing collagen proteins to the extracellular environment where non-enzymatic glycosylation takes place.[4]

Advanced glycation end products crosslink within and around collagen fibers, and compromise their functionality.[6] Type 1 collagen's main function is to resist tension and accounts for the rigidity in bone. The primary locations of type 1 collagen are skin, tendon, bone and dentin.[6] Collagen crosslinking within bone is known to affect bone stiffness and Young's modulus independent of the bones' mineralization and microarchitecture. This leads to weakening of bone strength without evidence of demineralization.[7]

Again note the dislocation of the intact talus and the 3D CT AP view demonstrating stacking of the metatarsals with the rolling down of the lateral side away from the viewer.



Here one can see preliminary correction with removal of the external fixator and the use of Steinmann pins due to the open wound plantarly. The surgeon has corrected the metatarsal stacking and there is now anatomical realignment of the foot with the talus and navicular articulating.

This 3D CT AP view confirms realignment of the talus and navicular as well as normal width of the foot with correction of the metatarsal stacking.

Advanced glycation end product accumulation recruits the increased formation of the pattern recognition receptor for AGE, known as RAGE, which expresses constitutively and causes increased downstream activation of RANKL when bound. According to Macaione and colleagues, increased RANKL activation causes osteoclastogenesis.[8] The soluble receptor (sRAGE) competes with RAGE to bind RANKL. The sRAGE also inhibits RAGE by binding to RAGE.[9]

Witzke and colleagues assessed the loss of RAGE defense as a cause of Charcot neuroarthropathy by focusing on three groups of patients.[7] The three groups included healthy control patients, patients with type 2 diabetes and patients with diabetic Charcot neuroarthropathy. Researchers recorded circulating levels of sRAGE and bone stiffness for each group. The study authors noted an 86 percent decrease in sRAGE values for patients with Charcot neuroarthropathy in comparison to the healthy control population. Bone stiffness was markedly reduced in the Charcot group. The study authors concluded that RAGE did in fact increase RANKL activation and RANKL is responsible for increased osteoclastic activity. Additionally, a reduction in bone stiffness with a concomitant increase in bone density may suggest a pathologic proliferation of cross-linked collagen.

Another potentially deleterious effect of reduction in circulating sRAGE is AGE-induced osteoblast apoptosis, which authors have implicated in alterations to bone repair in the face of elevated osteocalcin.[9] This may explain why Charcot fusion sites remain weak even after consolidation.

Key Insights On The Forces Acting On The Charcot Foot

For the foot and ankle surgeon, the most important part of these cell biology studies is the finding that bone stiffness was markedly reduced in patients with Charcot neuroarthropathy.[10] These findings correlate directly with studies that demonstrate a decreased Young's modulus of elasticity and tensile strength in the Achilles tendon in patients with Charcot neuroarthropathy.[11]

Carboxymethyl-lysine of type I collagen is a major constituent of bone, tendon and the ligaments that hold bones together. There is a combination of biochemical evidence and laboratory testing evidence that shows that AGE radically alters bone and tendon.[12] The best clinical treatment solution for this process would be a reversal of AGEs or a replacement of sRAGE, but these options are not feasible at this time.

This altered cellular biology that results in a diabetic Charcot foot requires an approach to surgical reconstruction that compensates for:

1) glycosylation of collagen resulting in failure of ligaments of the hindfoot and bone stiffness reduction; and
2) RANK-L-mediated and increased osteoclastic activity downregulating bone repair.

This demands a reconstruction plan that includes arthrodesis of the affected foot joints to negate abnormal ligaments from their normal role and selection of hardware strong enough to load share with the weakened bones.

As the foot moves throughout the gait cycle, the bones move relative to each other and Root and colleagues describe that the direction of forces acting upon the rearfoot or more proximal bone will react and angle with the direction of the forces acting on the forefoot or more distal bone.[13] As the joints in the foot move during the gait cycle, the ratio of compressive forces and rotational movement forces alternates. As the angle between joints becomes larger, the rotational movement forces are greater and compressive forces are lesser. During the period of great rotational movement forces, muscles and ligaments must function to resist excessive rotational motion at joints.

Root and coworkers theorized that when the foot is in a neutral or supinated position, the bone and joints of the medial column are at a lesser angle with each other, and are able to provide greater compressive forces in comparison to a pronated foot.[13] With the foot in a neutral or supinated position, there are reduced forces interacting at a joint.

Note the placement of a medial column beam for Charcot. One can perform beaming percutaneously.

The majority of the forces at that joint are developed from compression rather than rotational moment forces. However, if the angle between a joint increases the rotational moment forces, deformation is likely. It is the job of the muscles and ligaments to maintain sufficient tension force to resist any undesirable rotational motion at the joints in order to prevent disruption of joint integrity.

As muscles become overworked and ligaments become fatigued, this leaves the skeleton to resist the rotational forces on its own. Diabetes targets the intrinsic muscles of the foot and glycosylates tendon and ligament, creating a higher propensity for them to become fatigued and fail.

Peripheral neuropathy and polyneuropathy are common findings in the diabetic population. Neuropathy affects sensation and proprioception. Diminished sensation and proprioception inhibits one's ability to react to uneven

Here is an intraoperative radiograph showing the use of guide pins for placement of the beams within the first and second metatarsal segments to beam the medial column.

Here is a lateral view of Charcot reconstruction. All three metatarsal beams load share together because the subtalar joint is locked.

pavement, fatigued ligaments or any excessive or prolonged rotational movement forces that continually act upon the foot during the gait cycle.

Producing a foot which will be fixed in a neutral or slightly supinated position allows the bones and joints to function at a lesser angle with each other. This facilitates higher compressive forces and picks up some of the slack from the weakened intrinsic muscles, and fatigued ligaments and tendons. The foot will be capable of maintaining its own skeletal integrity.

The purpose of this article is to describe a biomechanical surgical approach based on Root biomechanics to reconstruct a Charcot foot. This results in a Charcot foot that does not rely on glycosylated soft tissues for stability but relies on the principles of proximal osseous stability.

What You Should Know About The Surgical Goals And Technique

The bones of the reconstructed Charcot foot should be neutral or slightly supinated. Glycosylation causes weakness in the ligaments that likely fail when the proximal stable bones and distal reactive bones are at increased angle during the propulsive stage of gait. Alternately, a neutral or slightly supinated foot position allows joint compression and synergy with arthrodesis hardware.

A neutral to slightly supinated foot has the following characteristics:

* A Meary's angle near 0 degrees
* Positive calcaneal inclination angle
* Slightly adducted forefoot
* Stable hindfoot

In light of the accumulating evidence that Charcot diabetic foot is most likely associated with AGE-RAGE glycosylation of collagen and ligamentous failure, arthrodesis of the displaced joints is the recommended surgical treatment. When performing arthrodesis, the positions of the foot should be as follows: adducted, a Meary's angle of 0 degrees, no supinatus and no stacking of metatarsals.

Since Charcot bone of the foot is intrinsically altered, its tensile strength, elasticity and porosity are abnormal. Therefore, any internal fixation the surgeon chooses should ideally function to supplement the weightbearing duties of attendant ligaments, tarsal and metatarsal bones. This introduces the concept of load sharing between implants that surgeons use for the Charcot diabetic foot.

Practically speaking, however, no current implants are designed as weight-bearing or load sharing for Charcot foot. This includes orthopedic screws and locking plates as well. Therefore, the responsibility to select the strongest fixation type and method currently falls to the surgeon.

Recommendations For Beaming As A Reconstruction Strategy

With this in mind, we can currently make the following recommendations:

1) Select the strongest hardware possible to load share since bone pathology does not favor normal arthrodesis. Bear in mind that stainless steel's load to failure is 240,000 PSI versus 180,000 PSI for titanium.
2) When it comes to realignment, stabilization and hopeful arthrodesis, ensure the foot is in a position of adductus with a positive Meary's angle and corrected cuboid height.
3) Use bent-wire external fixation in combination with internal fixation as it is synergistic with Steinmann pins or large diameter "beams."
4) Strategies to minimize failure of beaming hardware include using stainless steel instruments with the largest core diameter available.
5) Insert two beams into the medial column in the talus. These two beams share the load of the medial column with the pathologic ligaments and bones.
6) Lateral column stabilization arthrodesis fuses the metatarsal bases to the cuboid and cuboid to the calcaneus in an anatomic position. By rotating the lateral column metatarsal bases and cuboid superiorly, the surgeon can limit supinatus created by Charcot medial column failure. This decreases pressure on the lateral column.
7) Locking the most proximal foot joint, the subtalar joint, serves two purposes: It adheres to Root's principles of proximal osseous stability and permits load sharing of medial and lateral columns.
8) Use an external fixator in Charcot reconstruction with beaming. Authors have demonstrated that bent wire Ilizarov fixation has synergy of compression with screws that surgeons use as beams.[14]

In Summary

Beaming is a biologically-based biomechanical treatment for a metabolic disease. Its goal is to load share with metabolically altered ligaments, tendons and bone, and restore shape and function to the diseased diabetic foot. ■

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Key Principles On Frame Biomechanics And Application For Charcot Reconstruction

Recognizing the challenges of utilizing circular fixation in patients with diabetes and Charcot, this author discusses pertinent biomechanical factors and offers pearls on frame application to reduce complication risk.

By Byron Hutchinson, DPM, FACFAS

Surgeons have utilized circular fixation for Charcot reconstruction effectively for several years.[1-3] The diabetic patient poses unique challenges for the surgeon when considering the use of circular fixation. For the application to be successful, the surgeon needs to be familiar with certain aspects of frame biomechanics.

While a detailed discussion of frame biomechanics is beyond the scope of this article, Bronson and colleagues reviewed important aspects of frame stability and offered biomechanical analysis.[4] They emphasized having a strong understanding of the relative effect of the individual frame components and specific parameters of bone segment fixation on axial compression, torsional stiffness, anteroposterior and medial-lateral bending stability. Consideration of ring and wire diameter along with wire tension and wire angle, and their effects on bone segment stabilization is of critical importance as well. One needs to consider all of these variables when making recommendations for the use of circular external fixation in Charcot reconstruction.

The basic frame construct for Charcot deformity starts with the tibial block. The rings need to be close enough to the bone segment but wide enough to allow for swelling. For most patients with diabetes undergoing Charcot reconstruction, this requires funneling or coning of the tibial block (see above photo). This allows for the tibial block to be close to the bone segment and helps provide stability of the ring construct. In addition, it is important that the tibial block is high enough to provide additional stabilization to the block and to avoid vulnerable areas in the tibia. This tibial block is recommended for both ankle and midfoot Charcot reconstruction.

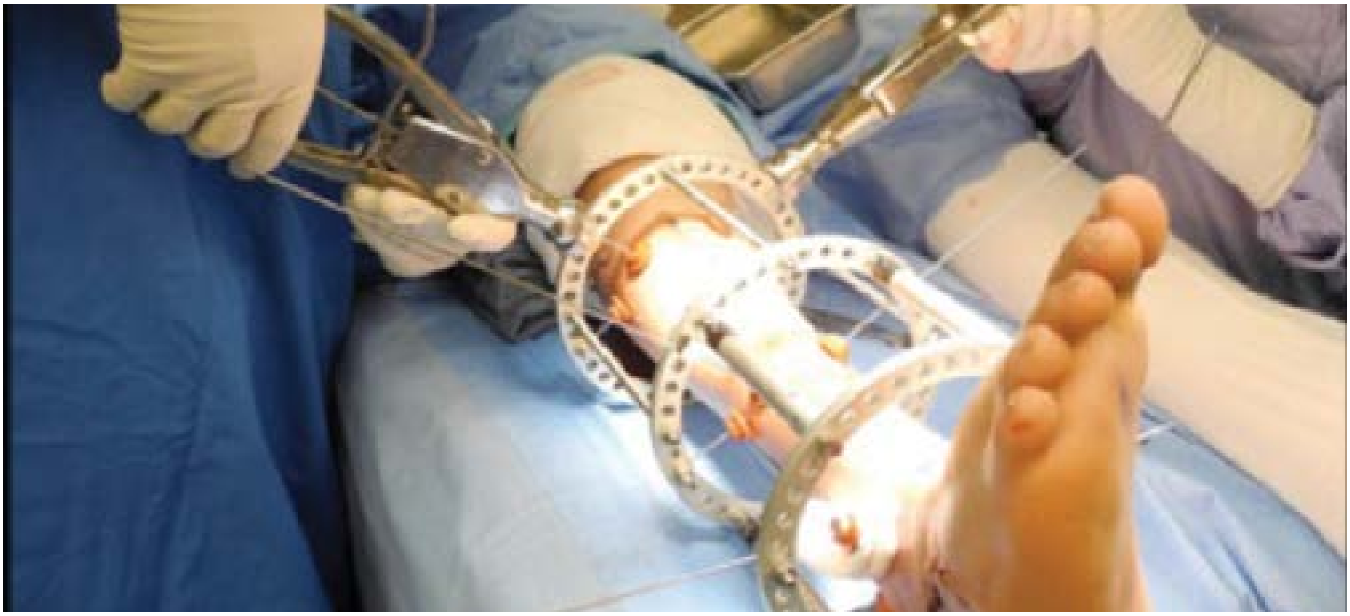
For most patients with diabetes undergoing Charcot reconstruction, this requires funneling or coning of the tibial block. This allows for the tibial block to be close to the bone segment and helps provide stability of the ring construct.

The basic fixation units are skinny wires and half pins. Understanding the relevant biomechanics and application of these fixation units is the most important aspect of a successful frame. A common mistake is bringing the wires down to the ring rather than building up to the wires with fixation elements. This places stress and tension on the wires resulting in wire failure or wire irritation/infection. Tensioned wires are self-stiffening and the more force applied, the greater resistance to force.[5]

The surgeon should ensure simultaneous tensioning of the skinny wires on the ring to maintain appropriate tension with one wire above the ring and one wire below the ring (see top photo on page 19). Simultaneous tensioning of the wires avoids the inherent problems with having to consider the angle of the wires on the ring with single tensioning. For example, any angle that is not 45 degrees will result in increased or decreased tension on the first wire when the second wire is tensioned.

Opposing olive wires will help to prevent translation and drop wires should be avoided. One can often use half pins along the tibial face and 4 mm half pins are the same as two tensioned wires at 90 kg as far as axial loading is concerned.[6] When it comes to patients with diabetes, more fixation units on a ring are recommended because of the frequency of pin site irritation or infection.

The double row footplate provides additional fixation options and has more stability than the single row footplate. Several options are available to close the footplate depending on the situation. Studies have shown that closure with two threaded rods is the most stable.[7] Placement of all the wires in the footplate is recommended before tensioning. Clifford and colleagues demonstrated a more effective tension sequence with simultaneous tensioning of the forefoot wires first followed by the calcaneal wires.[7] One may employ bent



The surgeon should ensure simultaneous tensioning of the skinny wires on the ring to maintain appropriate tension with one wire above the ring and one wire below the ring.

wire fixation along the footplate to work synergistically with the midfoot intramedullary beams (see photo at the right).

Limb salvage in diabetic patients undergoing Charcot reconstruction can be a considerable challenge for even the most experienced surgeon. Optimizing these patients is critical for a successful outcome and eliminating biomechanical failures in circular frame design will enhance the possibility for limb preservation. ■

This radiograph demonstrates the use of a bent wire fixation technique in a midfoot Charcot reconstruction.

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Current Insights On Charcot Ankle Reconstruction

When it comes to Charcot arthropathy of the ankle, this author emphasizes a strong awareness of the relevant pathologic and metabolic processes, assessment and optimization of comorbidities, and keys to optimal fixation.

By Byron Hutchinson, DPM, FACFAS

Charcot arthropathy of the ankle is a very complex, limb-threatening and life-altering deformity. Historically, surgeons avoided fusion because of a high incidence of nonunion or failure.[1] With improving methods of fusion and a better understanding of the neuropathic process in the ankle, limb salvage has become a much more viable option.

Surgical options in the ankle are typically reserved for the severe, unstable deformity when conservative care has failed.[2] The primary goals in ankle reconstruction are to achieve better alignment and stability of the ankle to allow for better bracing. In addition, those patients who are at high risk for ulceration or have had previous ulcerations can benefit from reconstruction.

It is paramount that the surgeon has a good understanding of the pathologic and metabolic processes at work in ankle Charcot. We know that Charcot is a non-infectious destruction of the joints and bone in patients with peripheral neuropathy. There is typically some trauma in the neuropathic patient that leads to two distinct pathways of destruction. Those with autonomic neuropathy and increased arteriovenous (AV) shunting develop subchondral collapse and fragmentation.

Those without autonomic neuropathy and no AV shunting develop neuropathic dislocation.[3] Interestingly, bone mineral density plays a role in the type of destruction. In 2004, Herbst and colleagues looked at dual-energy X-ray absorptiometry (DEXA) scans on affected limbs in 55 patients with diabetes to determine the

Here is a clinical view of active ankle Charcot.

Note the anteromedial incision for hindfoot fusion along with a tibiotalocalcaneal (TTC) fusion.

bone mineral density changes in midfoot and ankle Charcot.[4] They found that the fracture pattern predominated in the ankle and bone mineral density changes were pronounced in comparison to near normal values in the dislocation pattern. In 2011, LaFontaine and coworkers sought to analyze the histologic structure of Charcot bone in comparison to other patients with diabetes and healthy controls.[5] Their conclusion was that Charcot bone appeared to be woven bone. These studies and more have led many (including myself) to believe that diminished bone mineral density and a predominant fracture pattern is present in Charcot of the ankle.[6-8] In addition, woven bone has inferior integrity and these factors need to be taken into consideration during reconstruction in the Charcot ankle.

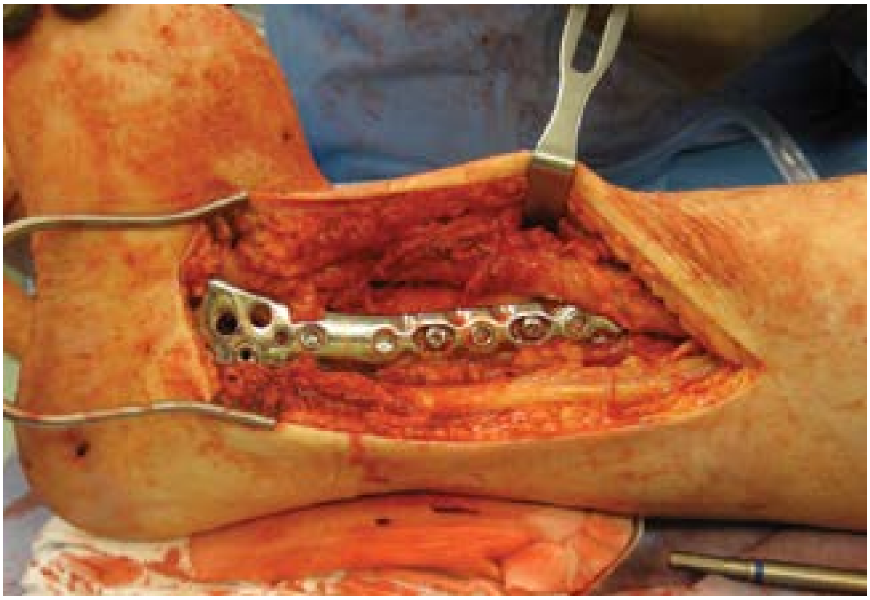
The basic surgical tenets are determining the planes of deformity and realignment. Control of infection is important as well as assessment of talar integrity and the viability of the soft tissue envelope.

The most favorable method for reconstruction of the ankle is not known but I favor a superconstruct involving a combination of internal and external fixation.[9] This superconstruct in the ankle takes into consideration the unique aforementioned metabolic situation when it comes to the ankle.

The primary goals of surgery are to provide bony stabilization and have an ankle that one can brace. In my experience, the best outcomes have been in those patients that commit to a Charcot restraint orthotic walker (CROW) boot for life. I have also found that the patients need to understand that this is limb salvage and in most circumstances, it is a "one and done" type of surgery. In addition, it is important to consider whether the surgery will improve their quality of life.

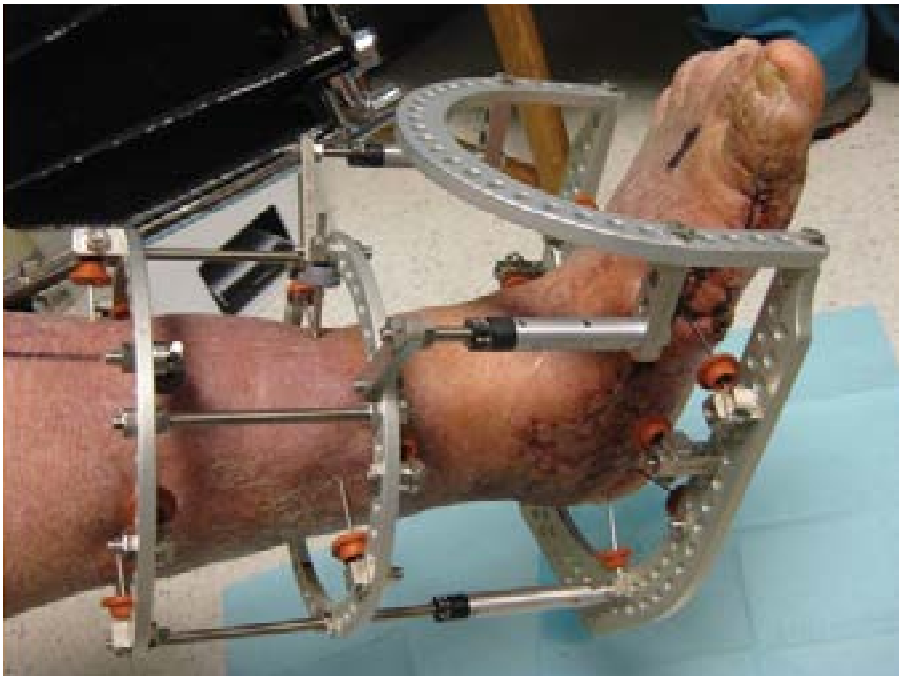
The vast majority of interventions I have performed have been in patients with inactive Charcot deformities in which they had a previous ulceration or were not able to wear a brace. In certain circumstances, this is ideal.

When patients have active Charcot, it is important for the surgeon to weigh the benefit of reconstruction versus the risk of limb loss in this setting. It has been my experience that patients with diabetes who present with a dislocation pathway

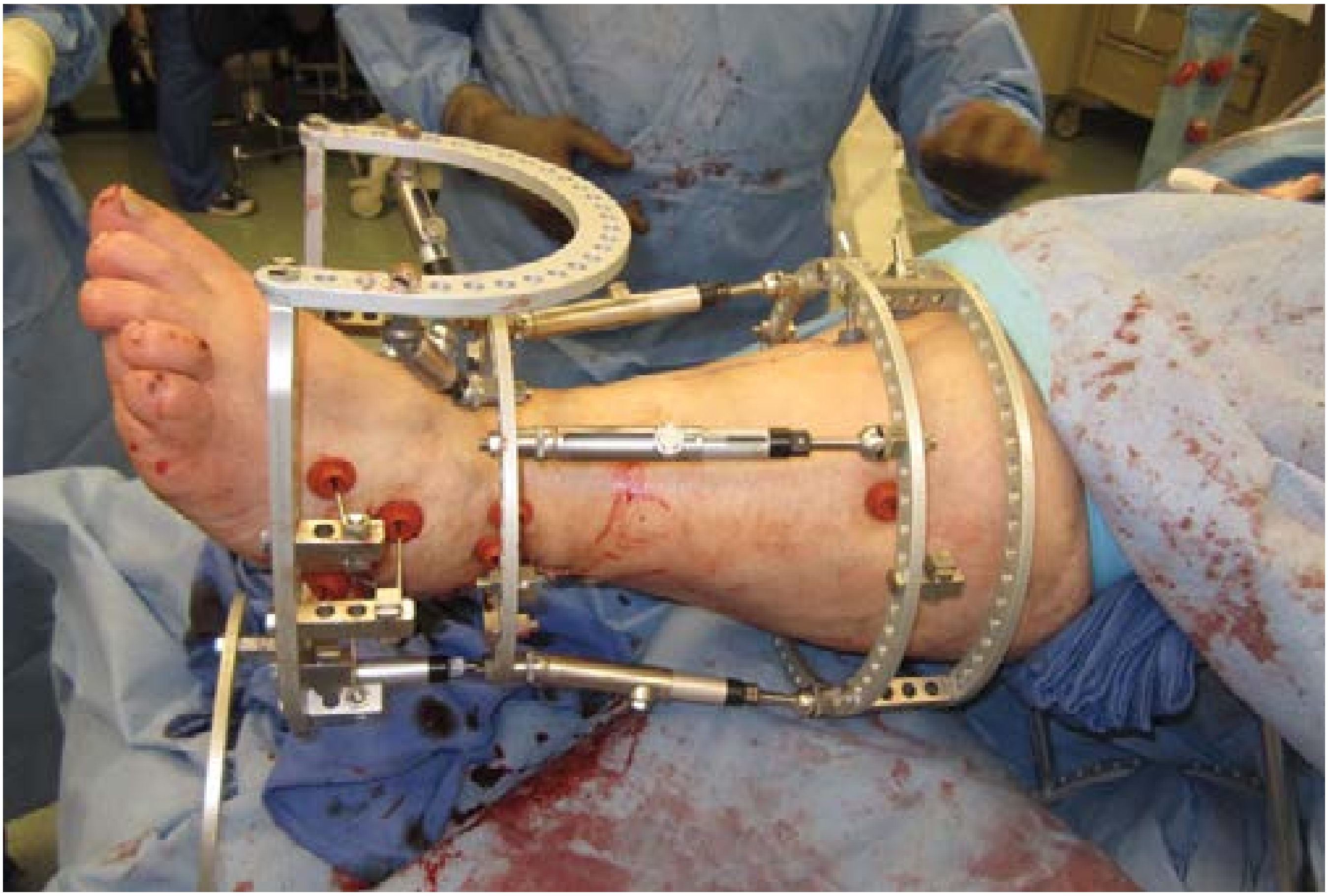


Here one can see the use of a distal femoral locking plate in a reconstruction case involving ankle Charcot.

The ideal external fixation construct for ankle Charcot is a circular fixator, which includes a tibial block and a foot plate. The author utilizes a double row foot plate and an extended tibial ring for more stability.



An extended tibial block avoids placing wires or half pins in the middle of the tibia where fractures can occur. In the left photo, note the mid-tibial fixation that can place the tibia at risk. In the right radiograph, there is a mid-tibial fracture due to fixation elements and premature weightbearing.



It is extremely important to funnel the frame based on the anatomy of the leg to keep the frame more stable.

and ankle deformity do much better with active Charcot reconstruction than those patients who have active fragmentation. If there is no deformity present, offloading is most appropriate.

Ensuring Preoperative Optimization Of Patients

Optimization of patients with Charcot deformity of the ankle is extremely important. This begins with evaluation and stabilization of the patient's comorbidities. One should obtain a metabolic bone profile prior to surgery.[10-12] The patient should have optimal glycemic control. Although there is no consensus on ideal HbA1c values, the closer one is to 8 percent, the more predictable the outcome. The surgeon should also consider end-stage renal disease. Dialysis patients generally do well if the surgery is timed around their dialysis schedule. In my opinion, renal transplant patients do not do well with reconstruction and are better served with a definitive amputation.

Psychosocial issues are also important to address. There are some recent studies to suggest that cognitive dysfunction occurs along with neuropathy.[13-15] The patient needs to have an appropriate support group and if that is not available, one has to consider a skilled nursing facility or home health at a minimum.

Pertinent Surgical Pearls

The surgical procedure centers around optimum alignment of the foot under the leg. Typically, one can do this through a lateral utility incision over the fibula and, at times, an ancillary anterior medial incision over the ankle joint, especially when midfoot correction is necessary. The vast majority of the Charcot ankles I see are in valgus as opposed to varus. Removing the fibula allows direct access to the ankle joint and facilitates removal of bone necessary to relocate the foot under the tibia.

The internal fixation platform that is most popular is an intermedullary (IM) nail.[16-18] When there is fairly good talar integrity and the deformity is varus or mild valgus, I prefer to use an IM nail. When there is a fracture dislocation pathway, the IM nail is preferable as well. When there is severe valgus or no talus, I recommend a lateral plate.[19]

> Patients with Charcot ankle deformity can be very challenging. With proper optimization, limb salvage is attainable.

The ideal external fixation construct for ankle Charcot is a circular fixator, which includes a tibial block and a foot plate. I utilize a double row foot plate and an extended tibial ring for more stability (see bottom photo on page 21). In addition, the extended tibial block avoids placing wires or half pins in the middle of the tibia where fractures can occur. It is also extremely important to funnel the frame based on the anatomy of the leg to keep the frame more stable (see bottom photo on page 22). The surgeon may use wires and/or half pins to complete the construct and should apply these using standard frame biomechanical principles.

It is important to have several fixation units on each ring with the understanding that some will have to be removed due to pin irritation or infection, which is higher in the diabetic population than the non-diabetic population. It is ideal to leave the frame on for 10 to 12 weeks.

In Conclusion

To summarize, patients with Charcot ankle deformity can be very challenging. With proper optimization, limb salvage is attainable. A combination of internal fixation and external fixation provides an excellent superconstruct to achieve successful limb salvage. ■

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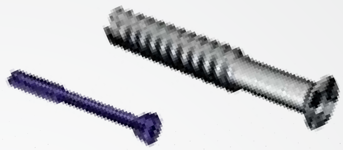

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