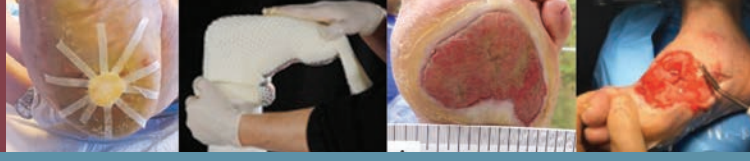

Total Contact Casting the Charcot Foot

Here's a closer look at why this is the "gold standard" for treating this condition.

BY ERIC JAAKOLA, DPM AND RYAN MOSS, DPM

History and Etiology

The earliest record of neuropathic arthropathy includes Sir William Musgrave in 1703 contributing this disease process to a complication of venereal disease. Later on, the term "Charcot Foot" was formed after the accounts of Jean-Martin Charcot in the 1880s, as he noted neuropathic arthropathy among his patients with tertiary syphilis.[14] This was the most common etiology of the disease process but was swiftly overtaken with the increasing prevalence of diabetic peripheral neuropathy in the U.S.

The prevalence of Charcot's neuro-arthropathy (CN) has been debated in literature, ranging from 0.08% to 7.5%. In regard to diabetic patients with peripheral neuropathy, this percentage rises to 29% in some specialty clinics.[11] While this disease is most often seen unilaterally, the prevalence of bilateral CN is not uncommon, with studies reporting up to a third of the cases. This is a debilitating disease process with a high risk of progression into severe deformity that can be limb-threatening.

Currently, the accepted clinical treatment for acute CN is "prompt, uncompromising reduction in weight-bearing stress, frequent monitoring, and gradual progression to unprotected weight-bearing in prescription footwear."[1] While this may be done with different methods, total-contact casting has been proven to be extremely effective in achieving this goal.

Recent studies as well as consensus documents favor total-contact casting for CN in terms of its efficacy in preventing further destruction of the foot structure from weight-bearing forces throughout the disease process. For the majority of foot and ankle neuropathic fractures and dislocations, the gold standard for treatment is non-surgical in nature, with the utilization of total-contact casting followed by appropriate bracing and footwear.[2]

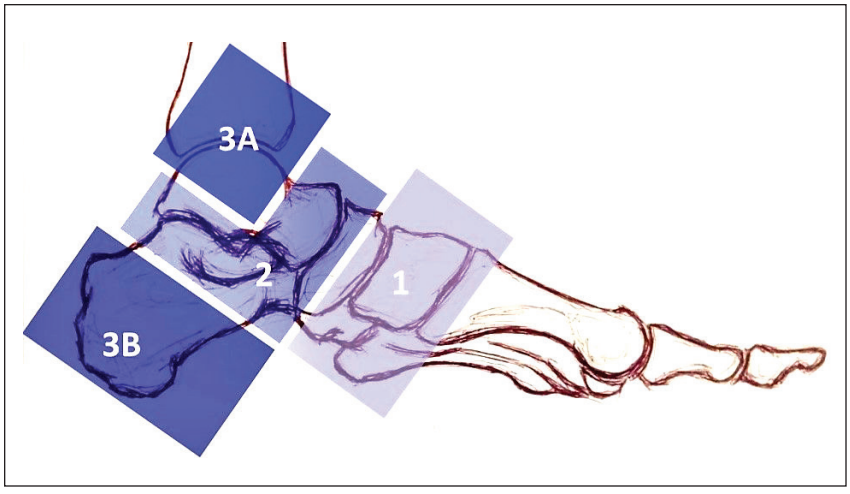

Figure 1: Brodsky Classification

In 2011, the ADA Consensus Statement described appropriate treatment as 'off-loading at the acute active stage of the Charcot foot as 'the most important management strategy' which could arrest the progression of the deformity. "Ideally, the foot should be immobilized in an irremovable total contact cast."[3] In 2014, Snyder, et al. agreed with the 2011 Report that recommends off-loading and immobilization—with total-contact casting "at the acute/active stage in order to prevent progression to deformity, and potentially, amputation."[3]

Classification

Charcot's arthropathy has been classified in a number of ways with different systems. Brodsky's Classification organizes the disease process in terms of location. In 1991, Sanders introduced a similar but expanded classification system (Figures 1 and 2).

The Eichenholtz classification system divides into phases of the disease process:

Stage 0: later added, describes the Inflammation phase, character-

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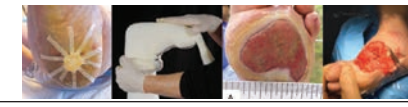

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ized by clinical signs of edema, erythema, and increased warmth but without radiographic changes.

Stage 1: Development phase holds the same clinical features but also with radiographic changes including demineralization of bone, subchondral lucency, joint subluxation/deformity, and peri-articular bony fragmentation.

Stage 2: Coalescence phase, demonstrates a decrease in temperature, edema, and erythema, with radiographic findings include coalescence of the bony debris, remineralization of the bone and callus formation.

Stage 3: Remodeling phase, demonstrates bone turnover and quality being normalized. Bony deformities that may have occurred in prior stages will persist in this phase, whether they are stable or unstable.

Pathogenesis of Charcot's Neuroarthropathy

There are two more common theories to date regarding the cause of CN; the first is neuro-traumatic, where unperceived repetitive trauma propagates osseous destruction that is undetected due to peripheral neuropathy. The second is neurovascular, where autonomic neuropathy causes sympathetic dysregulation, leading to vasodilation and increased blood flow into the bone, creating a mineral washout with subsequent bone destruction.[18]

As we still do not have a firm understanding regarding the specifics of the pathophysiology involved in the disease process, many clinicians have come to the conclusive assumption that it is some combination of the two theories.

It is known that there is an increase in osteoclastic activity in CN, as noted with increased levels of osteoclast markers (pyridinoline cross-linked carboxy-terminal telopeptide) and decreased markers for osteoblast precursors (carboxy-terminal propeptide) found in Type 1 collagen. This indicates a higher concentration of osteoclasts than osteoblasts. There is also a rise in alkaline phosphatase in patients with acute CN, which indicates increased bone turnover.[15]

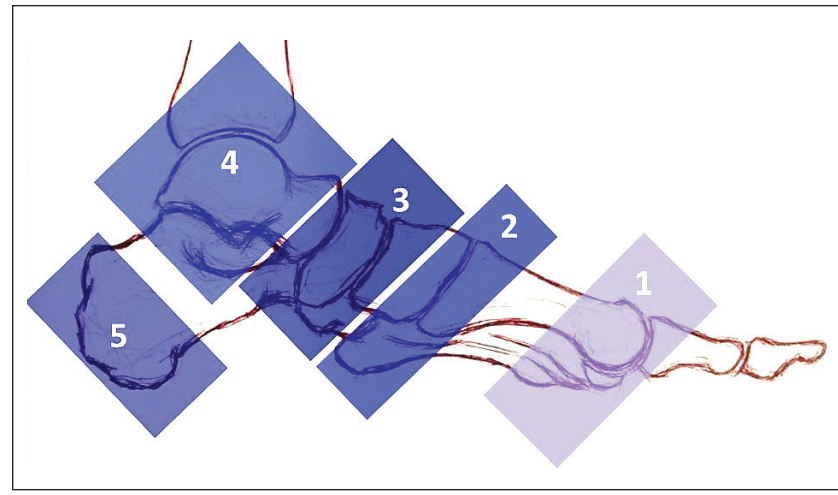

Figure 2: Sanders Classification

RANKL

Receptor activator of nuclear factor kappa-B ligand (RANKL)has been studied extensively in its relation to osteoclast activity in CN. It plays a significant role in osteoclastogenesis, but recent evidence supports the propagation of CN through a RANKL-independent pathway. Mabilleau, et al.[16] demonstrated a significant increase in osteoclast resorption with RANKL; however, when the RANKL antagonist osteoprotegrin (OPG) was added to the RANKL, the diabetic control group demonstrated a greater reduction in resorption than in the CN group. This clearly implied there is a separate, independent pathway of bony destruction present in CN.

Inflammatory cytokines that lead to bone resorption have been identified in CN bone specimens, including antibodies IL-1, IL-6, IL-8, TNF-alpha, and H&E. These cytokines initiate osteoclast recruitment and proliferation. There is also a decrease in anti-inflammatory cytokines IL-4 and IL-10.

Additionally, it has been noted that active osteoclasts also express these inflammatory cytokines during an active Charcot event, creating a positive feedback persistence in the inflammation process. This process is characteristically similar to what is seen in CN. IL-1 and TNF-alpha, which have the ability to induce monocyte apoptosis, but a resistance to this signal is seen in monocytes during active CN.[4]

An algorithmic approach to the evaluation and management of CN can aid in choosing the appropriate treatment choices which can ultimately reduce treatment duration and risk of complications.

The findings of a RANKL-independent pathway of osteoclast activity suggests that the increased levels of pro-inflammatory cytokines TNF-alpha, IL-6, and IL-8 are a cause of bony destruction, and that inflammation is the final common pathway in CN pathogenesis. These findings could suggest a potential treatment in anti-TNF-alpha therapy.[17]

Management of Charcot

An algorithmic approach to the evaluation and management of CN can aid in choosing the appropriate treatment choices, which can ultimately re-

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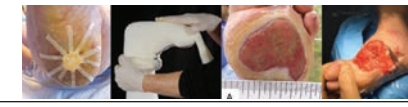

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duce treatment duration and risk of complications (Figure 3). Initial clinical suspicion for CN may indicate the need for imaging modalities to identify the process and also determine the severity of the physical manifestations. Radiographic imaging should be performed initially to determine any fractures, dislocations, or fragmentation at the joints. Radiographs may also demonstrate subchondral lucency, indicating the hyperemia within the bone. One of the most difficult points of diagnosing Charcot arthropathy is distinguishing the disease from osteomyelitis, as many clinical signs and imaging features are similar. Further imaging modalities may be necessary, such as magnetic resonance or nuclear imaging.

Once a diagnosis of Charcot arthropathy is confirmed, off-loading is the immediate treatment goal. This is done to prevent further destruction to the bony structure of the foot. In addition to being an effective off-loading technique, total-contact casting is not removable, and thus the patient

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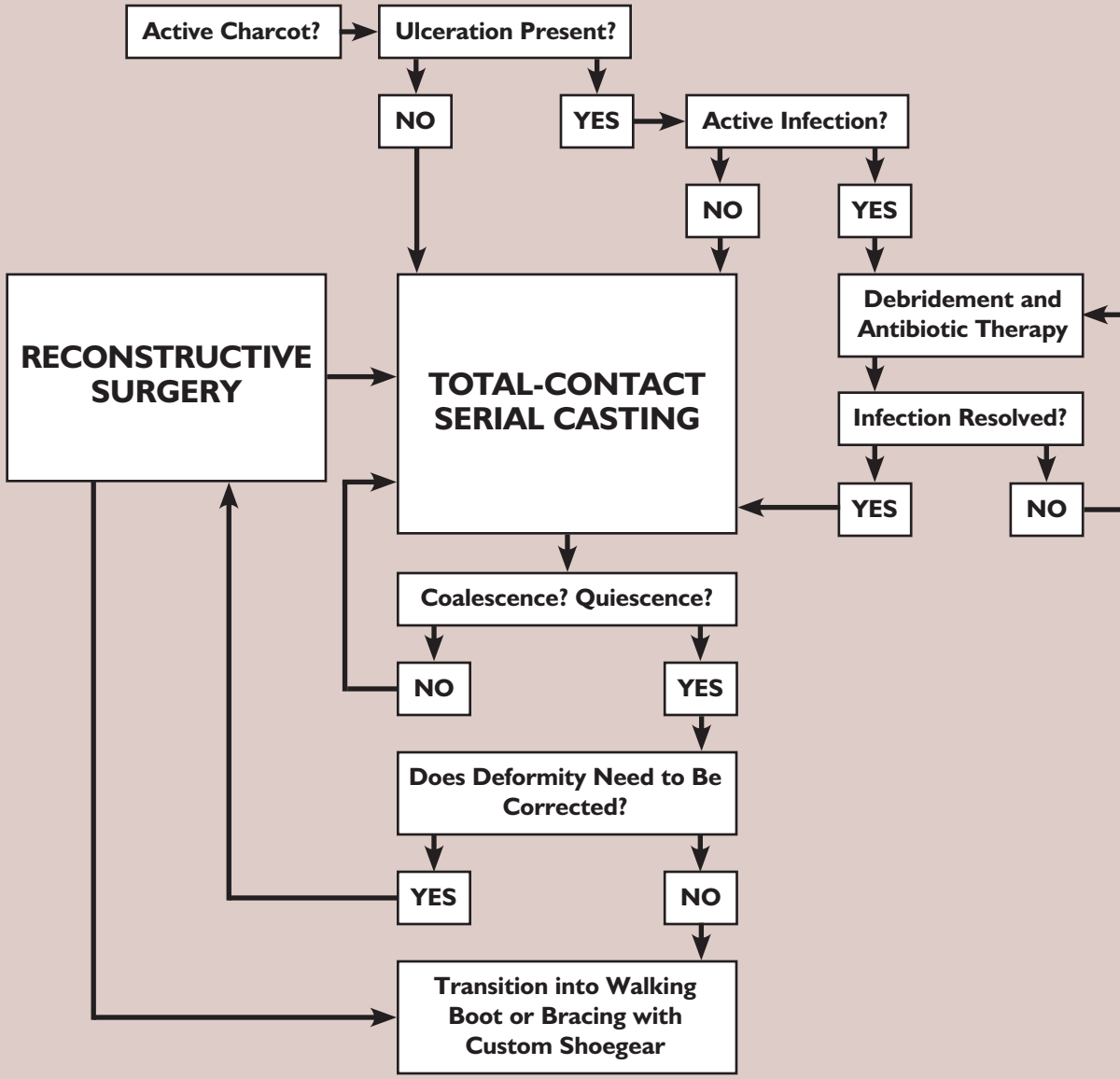
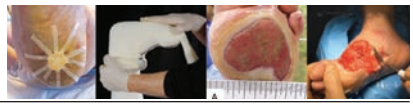
FIGURE 3:
Algorithm for Active CN Management





Casting *(from page 106)*

essentially remains compliant with the off-loading for the duration of the casting treatment. Casting continues throughout the active phase of the disease, changing the cast weekly if ulcerations are present or otherwise up to three weeks at a time, according to patient tolerance and clinical judgment. Once coalescence and consolidation occurs, the patient is transitioned into a removable CAM walker. Following transition, the patient may be treated conservatively with prescription custom footwear and close follow-up, or surgical management may be considered for reconstructive purposes.

Total-Contact Casting

"The utilization of a TCC reduces the mechanical forces, inflammation, and edema; redistributes the plantar pressure; limits bone and joint destruction; and can consolidate the progression of deformity. Its overall aim is to maintain a plantigrade foot, which can then allow weight-bearing in a shoe or brace."[5]

The purpose of total-contact casting is to maintain a plantigrade foot for the duration of treatment. In the case of CN, casting maintains the foot in its current position and structural orientation with the aim to prevent further disruption such as dislocation or fracturing. The casting method used applies the cast in such a way that it redistributes the focalized high plantar pressures to a larger surface area.

In addition, TCC can also aid in reducing the inflammation and edema. In this manner, total-contact casting is effective in reducing the risk of worsening the deformity, which may prevent the patient from having a plantigrade foot that can be accommodated in a shoe or brace after quiescence.

Indications for Total-Contact Casting

Total-contact casting may also be used in conjunction with plantar ulcerations in order to promote faster healing. Multiple randomized control studies show that TCC results in consistent healing of active diabetic foot ulcerations from 36 to 52 days for 85% to 95% of patients.

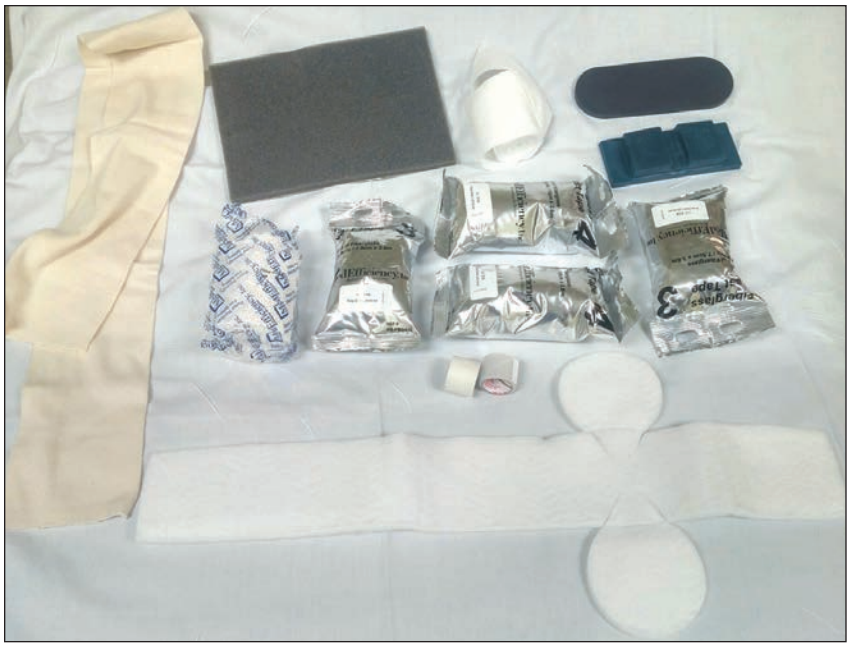


Figure 4: TCC Materials

A meta-analysis performed by Ulbrecht investigating the time to heal ulcerations utilizing total-contact cast off-loading showed an average time of 43 days in 88% of patients.[2]

The patient may be treated for plantar ulcerations with a TCC but only if there is no active infection or deep probing wound with potential for abscess formation; typically, a Wagner grade I or II ulceration. The patient must also have adequate blood supply to the limb in order to allow healing, typically an ABI equal to or greater than 0.7. The patient must also be educated on the lifestyle change that being casted entails. Be aware of any possible allergy to casting materials. Also, claustrophobia must be discussed with the patient, as this condition may make casting too stressful for the patient to endure. Compliance with cast care instructions is imperative as well; keeping the cast clean, dry, and intact. A wet cast must be removed immediately as moisture within the cast may cause skin breakdown or worsening of existing wounds.

Several kits are available for total-contact casting, each with variability in technique.

Casting Method

The goal of cast application is to apply a close contact cast to the leg, with the limb in as close to neutral positioning as possible in order to hold the limb within the cast equally at all aspects of the cast, not just the plantar aspect. This reduces the pressure on the weight-bearing plantar area of the foot, redistributing this pressure to the surface area region of the leg.

Several kits are available for total-contact casting, each with variability in technique. Each kit affords the ability to properly immobilize and offload the Charcot foot. It should be noted that the "EZ" Total Contact Cast produced by Derma-Sciences carries the same efficacy as the traditional cast. (Figures 6 and 7)

Traditional TCC placement includes a bias-cut stockinette sleeve, tape, Sci Foam for protecting the digits, 1/4 inch felt anterior tibial crest pad and malleolar pads, one cast

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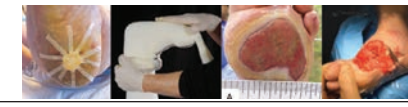

Casting *(from page 107)*

padding roll, one plaster roll, four fiberglass rolls, weight-bearing platform, and peg. A diagram of these materials is seen in Figure 4.

A bias cut stockinette sleeve is placed on the limb, overlapping the toes and knee. If possible it is important that the ankle is neutral for the duration of the casting. The toes are then covered with Sci Foam. The 1/4 inch felt material is then used for protection over the bony prominent regions of the leg. Each malleoli is covered, and the tibial crest is covered with one long strip. This protects these regions from the cast saw upon removal. The leg is then wrapped with cast padding. Overlap each pass by 50% to prevent over/under-padding in areas. The idea of total-contact casting is that the cast is form-fit to the leg, which is done so by having minimal padding. Additional padding will create space for the foot to move within the cast and cause complications, so this must be avoided.

The first layer of cast material is a 4" plaster roll applied circumferentially. This layer is wrapped in close contact to the cast padding, snug but not tight. It is wrapped proximally to just past the apex of the calf muscle belly. A 4" fiberglass roll is then wrapped in similar manner and tension, with 25% overlap.

Next, two 4" fiberglass rolls are pulled out dry, and folded to the appropriate length for a posterior splint, with a slit cut in order to place the plat-

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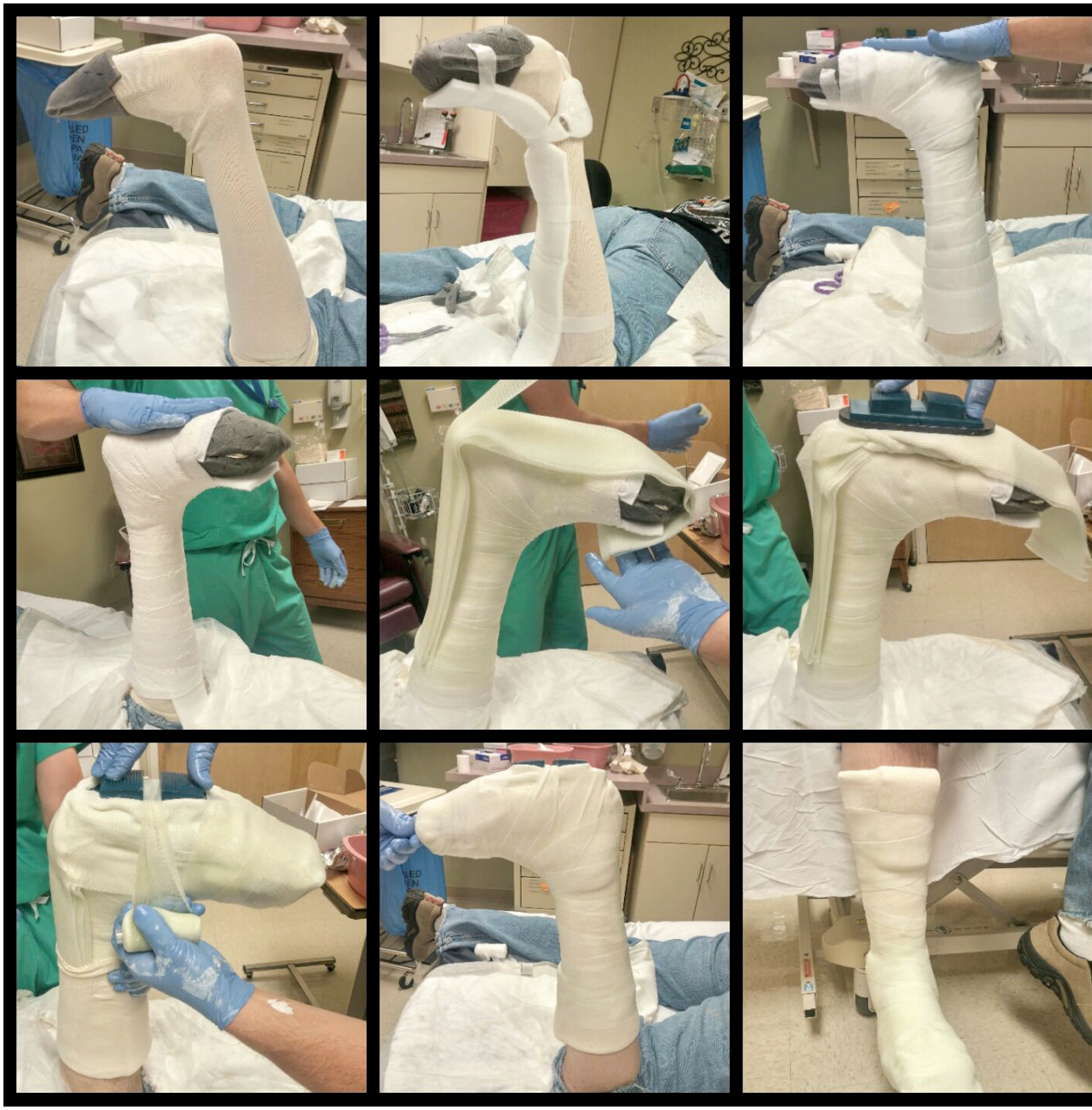
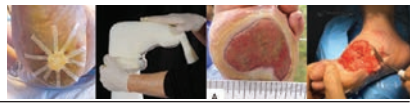


Figure 5: Casting process



Casting *(from page 108)*

form and walking peg plantarly. Some of the layers can then be bunched up together as necessary to build up the contour beneath the platform, whether it be filling in the medial arch in a cavus foot, or adding to the heel to compensate for equinus. The ideal placement of the platform and peg is to have it 90 degrees to the longitudinal axis of the leg, and in centered alignment with the frontal plane as well. The inner space between the two peg squares should line up with the anterior tibial crest in the lateral view.

The stockinette is then pulled down over the casting material and a final roll of 4" fiberglass is wet and rolled to cover and hold the peg and posterior splint material together (Figure 5).

Figure 6: Rolling on the TCC-EZ Cast

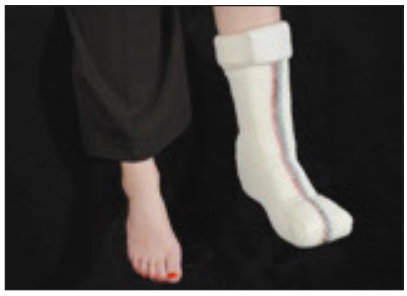


Figure 7: Finished TCC-EZ Cast

Complications

Total-contact casting is not without complications. Due to the nature of the cast, with minimal padding and close contact to the contour of the limb, any inadvertent divots or concavities made in the cast during application will harden and can potentially cause skin breakdown. Increased focal pressure can cause a pressure sore or focal ischemia which is painful and risks a new ulceration. Poor contact in an area will allow motion to occur within the cast, producing friction and possible blistering and ulceration, forcing the remaining areas of proper contact to distribute the pressure.

Complications such as these can be avoided with proper cast application on the appropriate patient, along with changing the cast often. Guyton et al. studied the iatrogenic complications from total-contact casting and found an overall complication rate of 5.52% per cast. "A frequently changed total-contact cast is a safe modality for the off-loading and immobilization of the neuropathic foot, albeit with an expected constant rate of minor, reversible complications."[9]

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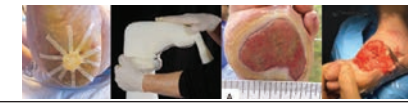

Casting *(from page 109)*

Post-Casting Management

The conversion from active to coalescence is based on clinical judgment and radiographic evaluation. Dermal thermometry using an infrared thermometric probe is also useful and well-described in literature.[10] These modalities should dictate the appropriate casting regimen and progression to prescription footwear.[1]

Regarding surgical reconstruction: "In general, surgery in the Charcot's arthropathy patient should be considered prophylactic in nature. If undertaken, the goal of any procedure should be to enable the patient to successfully return to accommodative footwear."[1] When considering the need for surgical management of Charcot's arthropathy, generally surgical intervention occurs following coalescence. Typically, there are two groups of surgical candidates. The first group includes those with a relatively stable foot with an excisable bony prominence treated with exostectomy. The second group includes those patients with unstable deformities that necessitate reconstruction. In these patients, the goal is to reconstruct a stable, plantigrade foot.

Conclusion

Charcot neuroarthropathy is a debilitating disease process with a complex pathogenesis that is still not fully understood. With the steadily rising population of diabetes today, the prevalence of CN will only continue to increase. As further investigation and research is poured into fully understanding the pathogenesis and searching for therapies, it is crucial that these patients and their manifestations are managed well.

Total-contact casting is a versatile and effective method for off-loading patients while still allowing them to ambulate.

The use of TCC reduces the patient's risk for worsened deformity which can progress to a limb-threatening deformity. The prompt diagnosis and initiation of treatment with total-contact casting for immobilization and protection throughout the active phases of this disease process is key to giving the patient the best possible outcome after quiescence. **PM**

The use of TCC reduces the patient's risk for worsened deformity which can progress to a limb-threatening deformity.

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