3rd International Course on
The Neuropathic
Osteoarthropathic Foot (Charcot)
Advanced Postgraduate Course

Rheine, Germany · 23-25 June 2016
Invitation

We are pleased to announce the 3rd International course on the Neuropathic Osteoarthropathic Foot (Charcot). The course will be held in beautiful Rheine, Germany, from 23-25 June 2016.

The course will combine theory with practical training sessions held in the specialised Interdisciplinary Diabetic Foot Centre at the Mathias-Spital.

The international course is based on the expertise gathered from 10 consecutive years of providing national courses on the Diabetic Foot. Based on an increasing interest and acknowledgement of the importance of proper treatment, we have decided to make the course available to an international audience. The course is in agreement with the International Consensus on the Diabetic Foot & Practical Guidelines on the Management and Prevention of the Diabetic Foot and is endorsed by the IWGDF (Int. Working Group of the Diabetic Foot).

The three day theoretical & practical course gives participants a thorough view of the different aspects of the diagnosis, treatment and management of the Charcot Foot. The course consists of theoretical sessions and practical sessions, where the main focus is on training the diagnostic and treatment skills necessary for the interdisciplinary treatment of Charcot patients. In the theoretical part, state of the art lectures and pro and contra presentations of disputed topics will be given by international specialists in the field.

We hope you will have a nice stay in Rheine.

Prof. Maximilian Spraul
Diabetologist, Mathias-Spital

Dr. Armin Koller
Orthopedic Surgeon, Mathias-Spital

Dr. Ludwig Schwering
Orthopedic Surgeon, Mathias-Spital

The course is supported by

General information


Venue, Practical part:
Mathias-Spital, Interdisciplinary Diabetic Foot Centre, Rheine, Germany

Venue, Theoretical part:
Mathias-Spital, University of Applied Sciences, Rheine, Germany

Form:
Lectures combined with hands-on workshops/training in clinic

Language: English

More information: www.charcotfootcourses.org

Social events
The course offers participants and faculty members plenty of opportunity to network and exchange knowledge during the social events. During the course, the following social events are included:

23 June, 19:30: Welcome dinner, Restaurant Casa Gonzales
Address: Tiefe Strasse 28 (located close to Hotel Lücke)

24 June, 20:00: Course dinner, Restaurant Farmacia
Address: Marktstrasse 2 (located close to Hotel Lücke)

25 June, 15:00: Excursion to Münster and dinner
at 18:00 at Pinkus Müller (local brewery).
Train to Münster from Rheine train station at.

www.charcotfootcourses.org

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<tr>
<th>Time</th>
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<td>17:00-17:15</td>
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<td>17:15-17:45</td>
<td>Pathogenesis of Charcot Osteoarthropathy, is there something new</td>
<td>Petrova</td>
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<td>17:45-19:00</td>
<td>Operative therapy of the active Charcot</td>
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<td>19:30-21:30</td>
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<td>09:00-09:30</td>
<td>X-rays and other radiological methods for the Charcot foot: pearls</td>
<td>Schwering</td>
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<td>09:30-10:00</td>
<td>Early diagnosis and conservative treatment of the Charcot Foot</td>
<td>Piaggesi</td>
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<td>10:00-10:30</td>
<td>Principles of deformity correction with external fixation</td>
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<td>11:00-12:30</td>
<td><strong>Practical Sessions 1</strong></td>
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<td>A: Early diagnosis and conservative treatment (Case presentation)</td>
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<td>C: Prosthetics and Orthotics in daily practice</td>
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<td>A: Ward round with case discussions</td>
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<td>C: Clinical orthopedic foot examination</td>
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<td>09:00-09:45</td>
<td>Rehabilitation and orthotic fitting</td>
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<td>09:45-10:15</td>
<td>Update on relevant studies concerning the Charcot foot</td>
<td>Spraul</td>
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<td>10:45-11:30</td>
<td>Biomechanics of the neuropathic and neuro-arthropathic foot</td>
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<td><strong>Practical Sessions 4</strong></td>
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<td>A: Handling of osteomyelitis in the Charcot Foot</td>
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<td>C: Conservative treatment and follow-up</td>
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<td>13:00-14:00</td>
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<td><strong>Summary and farewell</strong></td>
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The Charcot Foot in Diabetes

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LUIGI UCCIOLO, MD

The Charcot foot in diabetes poses many clinical challenges in its diagnosis and management. Despite the time that has passed since the first publication on pedal osteoarthropathy in 1883, we have much to learn about the pathophysiology, and little evidence exists on treatments of this disorder. The international task force was convened in January 2011 at the Salpêtrière Hospital in Paris, France, to review the literature and report on the definition, pathogenesis, diagnosis, and treatment of the diabetic Charcot foot. Recommendations in this report are solely the opinions of the authors and do not represent the official positions of the American Diabetes Association or the American Podiatric Medical Association.

DEFINITION—Charcot neuropathic osteoarthropathy (CN), commonly referred to as the Charcot foot, is a condition affecting the bones, joints, and soft tissues of the foot and ankle, characterized by inflammation in the earliest phase. The Charcot foot has been documented to occur as a consequence of various peripheral neuropathies, however, diabetic neuropathy has become the most common etiology. The interaction of several component factors (diabetes, sensory-motor neuropathy, autonomic neuropathy, trauma, and metabolic abnormalities of bone) results in an acute localized inflammatory condition that may lead to varying degrees and patterns of bone destruction, subluxation, dislocation, and deformity. The hallmark deformity associated with this condition is midfoot collapse, described as a ‘rocker-bottom’ foot (Fig. 1), although the condition appears in other joints and with other presentations. Pain or discomfort may be a feature of this disorder at the active (acute) stage, but the level of pain may be significantly diminished when compared with individuals with normal sensation and equivalent degrees of injury.

The set of signs and symptoms that occur together with CN qualifies this condition as a syndrome, the ‘Charcot foot syndrome.’

Definition and classification recommendations
- Nomenclature should be standardized to CN or the Charcot foot.
- Existing classifications do not provide prognostic value or direct treatment. Active or inactive should be used to describe an inflamed or stable CN, respectively. Acute and chronic can also be used in this regard, but there is no accepted measure that defines the transition point.

Pathogenesis—There is no singular cause for the development of the Charcot foot, but there are factors that predispose to its development, as well as a number of likely precipitating events. The current belief is that once the disease is triggered in a susceptible individual, it is mediated through a process of uncontrolled inflammation in the foot. This inflammation leads to osteolysis and is indirectly responsible for the progressive fracture and dislocation that characterizes its presentation (1). The evidence to support this hypothesis is largely circumstantial. A neurally mediated vascular reflex leading to increased peripheral blood flow and active bone resorption has been proposed as an etiological factor in the development of bone and joint destruction in neuropathic patients. However, the relationship between increased blood flow to bone and active bone resorption has not been conclusively defined.

Uncontrolled inflammation
When a bone is fractured, the release of proinflammatory cytokines including tumor necrosis factor-α and interleukin-1β leads to increased expression of the polypeptide receptor activator of nuclear factor-κB...
Predisposition

Neuropathy is a universal feature of the affected limb. Although it has been suggested that people with a Charcot foot may have particular patterns of sensory loss reflecting involvement of different fibers (5,6), this is not generally accepted. Nevertheless, three groups have shown that people who have had an acute Charcot foot exhibit retention of vasodilatory reflexes in contrast to diabetic individuals with distal symmetrical neuropathy without CN (7–9).

Despite these observations, it should be noted that the syndrome might also occur in patients with a spectrum of unrelated diseases complicated by nerve damage. These include distal neuropathies caused by toxins (ethanol, drug related) and infection (leprosy), as well as diseases of the spinal cord and nerve roots (tabes dorsalis, trauma, syringomyelia) and a number of other conditions (Parkinson’s disease, HIV, sarcoidosis, rheumatoid disease, and psoriasis). Although the neuroarthropathy is typically more proximal in those with disease of the spinal cord, the presentation may be otherwise indistinguishable.

Loss of protective sensation will increase the likelihood of trauma to the foot, while motor neuropathy could result in altered structure of the foot (with exaggeration of the plantar arch and clawing) and changed gait with resultant abnormal loading.

Finally, it is possible that peptides normally secreted from nerve terminals are also important in the underlying pathophysiology. Of these, calcitonin gene–related peptide (CGRP) is a likely candidate because it is known to antagonize the synthesis of RANKL. Hence, any reduction of CGRP through nerve damage will result in an increase in RANKL expression. It is of particular interest that CGRP has been reported to be necessary for the maintenance of the normal integrity of joint capsules, and it follows that any reduction in CGRP release by nerve terminals could facilitate joint dislocation (10).

Because it is not possible to identify those most likely to develop the Charcot syndrome, it is impossible to determine with any degree of confidence whether preexisting osteopenia is a significant predisposing factor. One group has, however, reported an apparent reduction in bone mineral density (BMD) of the femoral neck in the contralateral (unaffected) limb at the time of presentation.

The researchers also reported an association between BMD and the relative prevalence of fracture and of dislocation in the affected foot (11).

Diabetes may be associated with osteopenia, but the available evidence suggests that reduction of BMD is a feature of type 1 diabetes more so than type 2 diabetes (12). Any reduction of BMD in type 1 diabetes may relate to loss of islet peptides such as insulin and amylin (IAPP), both of which act as growth factors for bone. Despite this, the fracture risk in type 2 diabetes may be no less than in type 1 diabetes (12), and this would explain the fact that the presentation does not appear to differ between the two types of the disease. Any associated deficiency of vitamin D—with or without renal failure and secondary hyperparathyroidism—would increase the possibility of reduced BMD in diabetes. The use of thiazolidinediones could theoretically increase the likelihood of an acute Charcot foot through an effect on bone density, but this has not yet been reported. The use of corticosteroids as immunosuppressants in people with diabetes who have had a renal and/or pancreatic transplant (13) may explain the apparent high incidence of the Charcot foot in this group.

Although the Charcot syndrome may occur in a variety of conditions, diabetes is ostensibly the most common worldwide. Diabetes may predispose to its occurrence through a number of mechanisms. Apart from the presence of neuropathy and possible osteopenia, these include the effects of advanced glycation end products, reactive oxygen species, and oxidized lipids, which may all enhance the expression of RANKL in diabetes (10). The effect of local inflammation on this pathway would similarly compound the expression of RANKL. Furthermore, a single study has reported an apparent association between two OPG-related polymorphisms in people with a history of an acute Charcot foot in diabetes (14).

Many patients recall that the onset of the condition was precipitated by trauma that is often minor in nature (15). Other cases may be triggered by different causes of local inflammation, including previous ulceration, infection, or recent foot surgery. In this respect the occurrence of an acute Charcot foot as a complication of osteomyelitis is increasingly recognized in people with diabetes. Very occasionally, the onset of an acute Charcot foot may follow successful revascularization.
**DIAGNOSIS**—The initial manifestations of the Charcot foot are frequently mild in nature, but can become much more pronounced with unperceived repetitive trauma. Diagnostic clinical findings include components of neurological, vascular, musculoskeletal, and radiographic abnormalities. There have been no reported cases of CN developing in the absence of neuropathy. Accordingly, peripheral sensory neuropathy associated with reduced sensation of pain is the essential predisposing condition that permits the development of the arthropathy (16–19). Because of the very presence of insensitivity, a personal history concerning antecedent trauma is often unreliable (18,20,21). Typical clinical findings include a markedly swollen, warm, and often erythematic foot with only mild to modest pain or discomfort (16,18–20,22). Acute local inflammation is often the earliest sign of underlying bone and joint injury (23). This initial clinical picture resembles cellulitis, deep vein thrombosis, or acute gout and can be misdiagnosed as such. There is most often a temperature differential between the two feet of several degrees (20,24). The affected population typically has well preserved or even exaggerated arterial blood flow in the foot. Pedal pulses are characteristically bounding unless obscured by concurrent edema. Patients with chronic deformities, however, can develop subsequent limb-threatening ischemia. Musculoskeletal deformity can be very slight or grossly evident most often due to the chronicity of the problem and the anatomical site of involvement (16,17,19,25). The classic rocker-bottom foot, with or without planter ulceration, represents a severe chronic deformity typical for this condition (16,26,27). Radiographic and other imaging modalities can detect subtle changes consistent with active CN.

**Imaging of the Charcot foot**

Radiographs are the primary initial imaging method for evaluation of the foot in diabetic patients. Easily available and inexpensive, they provide information on bone structure, alignment, and mineralization. X-rays may be normal or show subtle fractures and dislocations or later show more overt fractures and subluxations. In later stages, the calcaneal inclination angle is reduced and the talo-first metatarsal angle is broken (Fig. 2). Medial calcification of the arteries is present in most Charcot feet and is a frequent secondary finding on radiographs (25).

However, radiographic changes of CN are typically delayed and have low sensitivity (28).

Magnetic resonance imaging (MRI) allows detection of subtle changes in the early stages of active CN when X-rays could still be normal. MRI primarily images protons in fat and water and can depict anatomy and pathology in both soft tissue and bone in great detail. Because of its unique capability of differentiating tissues with high detail, MRI has a high sensitivity and specificity for osteomyelitis and has become the test of choice for evaluation of the complicated foot in diabetic patients (29). Although not required for diagnosis when X-rays are diagnostic for Charcot bone and joint changes, MRI is very useful in making the diagnosis at its earliest onset before such changes become evident on plain films.

Nuclear medicine includes a number of exams based on the use of radioisotopic tracers. Three-phase bone scans, based on technetium-99m (99mTc), are highly sensitive for active bone pathology. However, diminished circulation can result in false-negative exams and, perhaps more importantly, uptake is not specific for osteoarthropathy. Labeled white blood cell scanning (using 111In or 99mTc) provides improved specificity for infection in the setting of neuropathic bone changes (30), but it can be difficult to differentiate soft tissue from bone. Therefore, this exam can be combined with a three-phase bone scan or sulfur colloid marrow exam when superimposed osteomyelitis is suspected (31). More recently, positron emission tomography scanning has been recognized as having potential for diagnosis of infection and differentiating the Charcot foot from osteomyelitis (32,33). However, this remains investigational at this time.

Evaluation of bone density may be useful in those with diabetes to assess onset of CN as well as fracture risk. BMD can be assessed using dual-energy X-ray absorptiometry or calcaneal ultrasound. BMD has been related to the pathological pattern of CN, whereby joint dislocation is more prevalent in those with normal mineralization versus fracture in those with diminished BMD (11).

Experts agree that radiographs are important as the first exam in virtually all settings (33,34). However, a negative result obviously should not offer any confidence regarding lack of disease. In a patient with low clinical suspicion of osteomyelitis and no sign of CN on radiographs, either three-phase bone scan or noncontrast MRI is very effective at excluding osseous disease. If the patient has an ulceration with a high likelihood of deep infection, MRI is the best diagnostic modality. Nonetheless, one test may not be adequate for full evaluation. In this setting where MRI diagnosis is indeterminate, a subsequent labeled white blood cell scan can provide more specificity and should be correlated with clinical findings. The decision of nuclear imaging versus MRI is largely based on personal preference, availability, and local experience. In general, if metal is present in the foot, nuclear medicine exams are preferred, whereas diffuse or regional ischemia makes MRI the preferred exam.

**Diagnostic recommendations for active CN**

- The diagnosis of active Charcot foot is primarily based on history and clinical findings but should be confirmed by imaging.
- Inflammation plays a key role in the pathophysiology of the Charcot foot and is the earliest clinical finding.
- The occurrence of acute foot/ankle fractures or dislocations in neuropathic individuals is considered active CN because of the inflammatory process of bone healing, even in the absence of deformity.
- X-rays should be the initial imaging performed, and one should look for subtle fractures or subluxations if no obvious pathology is visible.
- MRI or nuclear imaging can confirm clinical suspicions in the presence of normal-appearing radiographs.

**MEDICAL TREATMENT**—The medical treatment of CN is aimed at offloading the foot, treating bone disease, and
preventing further foot fractures (34). Because of the various etiologies of increased local bone resorption and/or secondary osteoporosis in patients with CN and limited randomized placebo-controlled trials in this area, treatment guidelines are largely based on professional opinion rather than the highest level of clinical evidence.

**Offloading**

Offloading at the acute active stage of the Charcot foot is the most important management strategy and could arrest the progression to deformity. Ideally, the foot should be immobilized in an irremovable total contact cast (TCC), which is initially replaced at 3 days, then checked each week. Edema reduction is often remarkable in the first few weeks of treatment. The cast should be changed frequently to avoid “pistoning” as the edema subsides. If possible, the patient should use crutches or wheelchair and should be encouraged to avoid weight bearing on the affected side. The casting is continued until the swelling has resolved and the temperature of the affected foot is within 2°C of the contralateral foot (35).

An alternative device for offloading the acute active stage of CN is a prefabricated removable walking cast or “instant TCC” technique, which transforms a removable cast walker to one that is less easily removed (36,37). It is important to take into consideration that TCC may actually have unfavorable consequences on the non-Charcot limb and induce unnatural stress patterns causing ulcerations and even fractures. Furthermore, patients with CN have increased instability and risk for falling and fracture as a result of multiple comorbidities including loss of proprioception and postural hypotension. Nonetheless, it should be noted that total immobility has disadvantages in itself with a loss of muscle tone, reduction in bone density, and loss of body fitness.

Duration and aggressiveness of offloading (nonweight bearing vs. weight bearing, nonremovable vs. removable device) are guided by clinical assessment of healing of CN based on edema, erythema, and skin temperature changes (34,35). Evidence of healing on X-rays or MRI strengthens the clinical decision to transition the patient into footwear. To prevent recurrence or ulceration on subsequent deformities, various devices are recommended after an acute or active episode has resolved, including prescriptive shoes, boots, or other weight-bearing braces. Frequent monitoring is required.

**Antiresorptive therapy**

Treatment by antiresorptive drugs has been proposed because bone turnover in patients with active CN is excessive. However, there is little evidence to support their use, but both oral and intravenous bisphosphonates (38) have been studied in the treatment of CN in small randomized, double-blind, controlled trials (39,40) or in retrospective controlled studies (41). Some patients cannot tolerate oral bisphosphonates but may benefit from intravenous therapy using pamidronate or zoledronic acid (42). Intranasal calcitonin is another antiresorptive agent that has been studied in CN. This treatment was associated with a significantly greater reduction in cross-linked carboxy-terminal telopeptide of type I collagen and bone-specific alkaline phosphatase than standard treatment in the control group that received only calcium supplementation and offloading. Calcitonin has a safer profile in renal failure when compared with bisphosphonate therapy (43–46). However, a single dose of intravenous bisphosphonate generally does not require renal adjustment. There is no conclusive evidence for using bisphosphonates in active Charcot foot, and our understanding is evolving as more trials are currently underway.

**Bone growth stimulation**

There is limited evidence for the use of external bone stimulation in CN. Ultrasonic bone stimulation was reported for the treatment of CN of the ankle and for the healing of fresh fractures. Direct current electrical bone growth stimulators have been used specifically in CN patients undergoing arthrodesis and clinically tested to promote healing of fractures in the acute phase of CN in small case series. Although these findings are promising, there have been no subsequent studies to validate this method, and its use has been supported only as an adjunct therapy during the postsurgical period (45–49).

**Recommendations for medical therapy**

- Offloading the foot and immobilization are the most important treatment recommendations in active CN and can prevent further destruction.
- There is little evidence to guide the use of available pharmacological therapies to promote the healing of CN.
- Protective weight bearing is required after an active episode, involving weight-bearing devices such as prescription shoes, boots, or braces.
- Lifetime surveillance is advised to monitor for signs of recurrent or new CN episodes as well as other diabetic foot complications.

**SURGICAL TREATMENT**—Surgical treatment of Charcot arthropathy of the foot and ankle is based primarily on expert opinion. Surgery has generally been advised for resecting infected bone (osteomyelitis), removing bony prominences that could not be accommodated with therapeutic footwear or custom orthoses, or correcting deformities that could not be successfully accommodated with therapeutic footwear, custom ankle foot orthoses, or a Charcot Restraint Orthotic Walker (50). This clinical approach is based on expert opinion and small, uncontrolled retrospective case series. There has been an increasing trend in the literature to advise earlier surgical correction of deformity and arthrodesis, based on the assumption that surgical stabilization would lead to an improved patient-perceived quality of life (51).

Several investigators have suggested that Achilles tendon lengthening combined with total contact casting has the potential to decrease the deforming forces at the midfoot and decrease the morbidity associated with CN (52–57). Exostectomy offers the potential to reduce pressure caused by bony prominences. This treatment is often combined with accommodative bracing and appears to obtain more favorable results in patients without associated ulcers (58–60).

Surgery has generally been avoided during the active inflammatory stage because of the perceived risk of wound infection or mechanical failure of fixation. Two recent case series would suggest potentially favorable outcomes with early correction of deformity combined with arthrodesis (61,62). Most case series have focused on reconstruction of the deformity by reduction and arthrodesis using standard methods of internal fixation. Because of the poor bone quality, expert opinion has advised an extended period of nonweight bearing after surgery to account for the poor bone healing and inherent weakness of the underlying osseous structures. Early surgical series showed improvement in restoring a plantigrade foot and preventing recurrence of ulceration, although nonunion, failure, and loss of initial correction were common (63–67). The concept of an internal fixation “superconstruct"
that extends internal fixation beyond the zone of fusion has evolved to address these issues (68). The combination of poor bone quality and a tenuous soft tissue envelope in a relatively immune-impaired population has led many surgeons to use a modification of the external fixation method of Ilizarov to correct deformity with a limited risk for surgical-associated morbidity (69–74).

**Charcot arthropathy of the ankle**
Given the common failures of nonsurgical management of CN of the ankle, the task force members agree that surgical management could be considered a primary treatment. Surgical correction of deformity at the level of the ankle is likely more necessary due to the poor tolerance of deformity in the coronal plane (i.e., ankle varus or valgus) and the resultant prominence of the malleoli and their vulnerability to pressure-induced ulceration. Several small uncontrolled series have recommended augmented internal fixation followed by prolonged periods of immobilization and nonweight bearing in neuropathic patients who sustain acute ankle fractures (75–77). Acute ankle fractures in patients with complicated diabetes are associated with significantly higher rates of noninfectious complications and need for surgical revision when compared with diabetic patients without other organ system comorbidities (78). Numerous techniques have been reported without comparative effectiveness (79–82). All of the surgical studies are retrospective in nature without a control group and are based on a limited number of patients. While a strong, stable construct is required, inconclusive data exist to recommend one form of fixation over another (i.e., internal, external, or combined) in the surgical reconstruction of the foot and ankle in patients who are not infected.

**Recommendations for surgical treatment**
- Surgical treatment is beneficial in CN cases refractory to offloading and immobilization or in the case of recalcitrant ulcers.
- The initial management of acute neurovascular fractures and dislocations should not differ from other fractures.
- Exostectomy is useful to relieve bony pressure that cannot be accommodated with orthotic and prosthetic means.
- Lengthening of the Achilles tendon or gastrocnemius tendon reduces forefoot pressure and improves the alignment of the ankle and hindfoot to the midfoot and forefoot.
- Arthrodesis can be useful in patients with instability, pain, or recurrent ulcerations that fail nonoperative treatment, despite a high rate of incomplete bony union.
- For severe CN of the ankle, surgical management could be considered a primary treatment.

**CONCLUSIONS**—The Charcot foot syndrome is a complex complication of diabetes and neuropathy. Its destructive effects on the foot and ankle begin with a cycle of uncontrolled inflammation. The classic rocker-bottom foot deformity is a late stage of the syndrome and can be avoided by early recognition and management. Offloading is the most important initial treatment recommendation. Surgery can be helpful in early stages involving acute fractures of the foot or ankle or in later stages when offloading is ineffective. An algorithm summarizing the approach to the Charcot foot can be seen in Fig. 3.

**Figure 3**—An algorithm depicting the basic approach to the Charcot foot. “Osteomyelitis can be difficult to distinguish from the Charcot foot. The reader is referred to the “Imaging of the Charcot foot” section of the article for techniques to improve specificity of various imaging modalities.

**Acknowledgments**—The American Diabetes Association/American Pediatric Medical Association Task Force meeting was supported by unrestricted educational grants from Small Bone Innovations, sanofi-aventis, and Integra LifeSciences. No other potential conflicts of interest relevant to this article were reported. The authors thank Cécile Swiatek, chief librarian, from the J-M. Charcot library at the Hospital Pitie-Salpetriere for her assistance with the site preparation for the task force and references in this article.

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German-Austrian consensus on operative treatment of Charcot neuroarthropathy: a Perspective by the Charcot task force of the German Association for Foot Surgery

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A number of published guidelines exist on the diabetic foot, yet the sections on Charcot neuroarthropathy (CN) focus mainly on diagnosis and conservative therapy. Surgical aspects, if ever present, are addressed very briefly and are very limited on surgical information and guidelines (1). For this reason, a group of German and Austrian foot surgeons who are well acquainted with the operative treatment of CN established a consensus statement despite a plethora of existing diverging opinions. The following proposal is far from scientific evidence, but may be the basis for an ongoing discussion and further research opportunity.

Etiology of Charcot neuroarthropathy

Charcot neuroarthropathy is characterized by a predominately painless destruction of pedal bones and joints in which the etiology is not entirely understood. A complex compound of neuropathy, repeated trauma, hypervascularization, and molecular-biological alterations of bone metabolism may result in dramatic deterioration of the foot skeleton. A distal symmetric polyneuropathy (PNP) is conditio sine qua non for the development of neuroarthropathy. Diabetes mellitus is the most frequent underlying disease, yet sequelae of long-term alcohol abuse or idiopathic cases should not be overlooked. Perhaps 80% of all patients suffering from PNP have long-standing diabetes mellitus. An additive effect of diabetic metabolism and alcohol or nicotine as neurotoxins has not yet been examined. Ischemic or idiopathic PNP are prevalent in a small percentage of patients.

Neuropathy may affect different efferent and afferent nerve fibers; first, sensory neuropathy interfering with the receptor-activated nuclear factor kappa-ligand/osteoprotegerin (RANK-L/OPG) system as a possible explanation for an unleashed inflammatory response to a minor trauma or repetitive stress (2). Secondly, autonomic neuropathy with dysfunctional vascular control and opened arteriovenous shunts as a possible reason for local osteoporosis and lastly, motor neuropathy with paresis of intrinsic foot musculature and consequent development of foot deformity (claw toes, high arched foot) as a reason for increased static and dynamic loading.

The diabetic foot syndrome comprises three clinical subgroups: peripheral vascular disease (PVD) in 25%, PNP in 25%, and a combination of PVD and PNP in 50%. CN, where PNP is always present (although not always noticeable on clinical examination), has an estimated incidence of 7% per year among diabetics with PNP, as recently published by a major German health insurer on the basis of data collected in 2007. In the German situation, this translates to about 5,000 cases emerging every year (3). Stuck et al. (4) reported an annual incidence of 1.2% in a cohort of diabetics with increasing incidence of CN in the presence of PNP or obesity. Coexistence of PVD may occur with long-standing CN
in up to 30% of cases. Nevertheless, the low incidence of CN in the general population is comparable to malignancies and makes the high frequency of misdiagnosis understandable. Sohn reported a 5-year mortality rate of 28.3% among patients with CN (5).

Classification of Charcot neuroarthropathy
Charcot neuroarthropathy is classified based on the topography of affected joints, course of the disease, and patterns of destruction. In this consensus, localization is classified according to the Sanders system (6). The simplicity and practicality of this system implies its limitations, when more than one joint line is involved or when the topographic pattern deviates from anatomical lines (e.g. Lisfranc, Chopart). The Sanders classification system does not allow for deduction of a specific operative procedure based on a given radiological CN pattern.

Another important classification was established by Eichenholtz in 1966 describing destruction as well as repair of joints and bone in the course of time (7). This clinical and radiographic staging system has been well accepted internationally, delineating three distinct stages: (1) destruction, (2) resolution, and (3) coalescence. A prodromal Stage 0 could represent a sensible modification in cases of bone bruise apparent on magnetic resonance imaging (MRI) without manifest changes on plain film radiographs. The denomination ‘Stage 0’ might underestimate a serious problem; therefore, another proposal is to subdivide Stage 1 into ‘1a’ with clinical signs of inflammation and bone bruise on MRI plus ‘1b’ with additional osseous destruction visible on conventional radiographs.

Ulcers often accompanying CN are best classified using the University of Texas Wound Classification System that describes ulcer depth and the presence of inflammation, ischemia, and/or PNP (8). Risk of amputation correlates well with the more severe stages (3D). Category E should be introduced in case of dialysis, as practical experience shows a high failure rate of conservative ulcer treatment when end stage renal disease is present. An ulcerated CN foot should be characterized by means of Sanders, Eichenholtz, and the University of Texas Wound Classification System.

Diagnosis of Charcot neuroarthropathy
Combination of medical history, clinical examination, and conventional radiography (anteroposterior, lateral, and mortise views) is sufficient for making the diagnosis of CN. Affected bones and the extent of bone bruise can be identified precisely with the help of MRI. Any suspicion of Eichenholtz Stage 0 or 1a, respectively, must include MRI or scintigraphy in addition to plain film radiographs. Clinical significance of bone bruise as incidental findings in patients with diabetic PNP remains unclear, in particular with regard to potential development of CN. Uncertainty also exists in terms of estimating safe loading capacity of Charcot feet with the help of MRI, as many of those feet with destructed joints will retain a life-long inflammatory activity due to degenerative changes. Last but not the least, distinction between CN and osteomyelitis remains difficult on the basis of radiological examination alone.

Specimen for microbiological testing should be taken from deep tissue, preferably from bone and through intact, non-contaminated skin under sterile conditions. Laboratory testing does not always facilitate a distinction between acute CN and osteomyelitis or abscess formation, as on the one hand acute CN is often accompanied by leukocytosis and elevated C-reactive protein. On the other hand, osteomyelitis may demonstrate only vague signs of inflammation due to ischemia or immunodeficiency (HbA1c > 11%).

As a complex correction of a deformed Charcot foot may turn into a catastrophe in the presence of relevant ischemia, the absence of palpable pulses must imply vascular examination ranging from Doppler sonography to invasive arteriography.

Therapy of Charcot neuroarthropathy – basic principles
Therapy of CN is often conservative. A deformed but plantigrade foot capable of full weight bearing in a shoe or orthosis and without increase of deformity is not a candidate for surgery. There are a variety of devices available for conservative treatment. Each device, such as total contact cast (TCC), prefabricated walker, Charcot Restraint Orthotic Walker (CROW), or individual ankle foot orthosis (AFO), has a different risk-benefit profile and has to be selected by the treating physician. Injuries due to ill-fitting orthoses or shoes may create an immense medical and financial burden.

An acute Charcot foot may call for in-patient treatment or off-loading by means of a wheelchair over a period of 6-8 weeks. After decrease in the acute inflammatory stage, total weight relief may be replaced by orthotic treatment with particular emphasis on rigid three-dimensional fixation of the foot and lower leg including elimination of tibial rotation. Physical load is gradually increased according to clinical parameters monitoring swelling, redness, and sensible heat. Resumption of walking as soon as possible protects against loss of bone mineral density, thereby reducing cadence and walking speed when using the orthosis. Partial weight bearing is not feasible in the presence of PNP. Thus, guidance of weight bearing takes place by limitation of walking time and speed. Knowledge on the field of rehabilitation with shoes and orthoses is extremely helpful.

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Principles of operative treatment of Charcot feet

Closed reduction and retention by means of casting is ineffective in cases of acute CN with joint dislocation and significant instability. This subtype of CN can only be managed by open reduction with internal or external fixation (ORIF or OREF).

From a biomechanical point of view, the two-column model of the foot has to be taken into account. Fusion of the lateral column should be considered, even if the problem is confined to the medial column only. As soon as conservative treatment signals an unfavorable outcome, reconstructive surgery should take place without waiting for Eichenholtz Stage 3, where deformity has become fixed and rigid. Ulcers are not necessarily an obstacle to surgery. An infected ulcer, however, should be treated first with debridement, moist dressings, and antibiotics. Bony prominences with high risk of ulcer occurrence or reoccurrence should be excised, preferably via a direct surgical approach or from the lateral or medial foot border. Vast soft tissue defects are treated in the scope of plastic surgery. Infected Charcot feet are the worst case scenario. To be precise, treatment is no longer targeted to neuroarthropathy but has to follow the rules of septic surgery. Even amputations or wide internal resections may be necessary.

The aim of surgery is to correct deformities in all three planes. The frontal and transverse planes are more important than the sagittal plane. Mild equinus position of the foot may even be useful to correct for a shortened limb due to loss of bone stock. On the other hand, CN of the tarsal bones may be in part a result of a shortened Achilles tendon. In this case and in the presence of a mobile ankle joint, tendoachilles lengthening (TAL) should be considered. Adequate technique, for example intramuscular lengthening, is important to avoid calcaneal foot position with the risk of heel ulcers. Preferably, osseous corrections are performed in a subtractive rather than in an additive fashion. Allogenic cancellous bone or synthetic bone substitutes cannot be recommended without reservation, although use of autologous graft is not stringently required.

To avoid disuse osteoporosis, total off-loading should be reduced to an unavoidable duration of 6–8 weeks. The use of circular frames may even permit early weight bearing with the appliance. Bone fusion can be evaluated by computed tomography or conventional radiographs. After internal fixation or after removal of an external fixation device, the foot has to be protected from bending and torque forces by means of an AFO that is generally worn over a period of 3–6 months. The device is designed for rigid fixation of the foot and full weight bearing, as patients with PNP cannot practice partial weight bearing.

Operative therapy of Charcot feet

Eichenholtz’s or Sanders’ classification does not enable a clear association of deformity patterns and operative techniques. Nevertheless, the Sanders classification is very common, therefore it is used for the following overview.

Sanders I

Type I affects the metatarsophalangeal (MTP) and the interphalangeal joints. The natural course of this type is different from the other four. The percentage of patients with PVD is significantly higher, whereas body mass index is not so much elevated. When the MTP joints are involved, bone changes are predominantly resorptive leading to the so-called candy stick deformity of the metatarsals. Reconstructive surgery in these cases is rarely indicated. Dislocation of the first MTP joint may require repositioning and fusion. For the most part, resections of bone in this type are performed due to severe destructions or superimposed infections.

Sanders II

Type II frequently affects the tarsometatarsal articulations. (Lisfranc). A rather common variation is periancillary involvement, and sometimes the neuroarthropathic changes are restricted to the medial or the lateral column. Diverging dislocations are seen as well as deviations of all metatarsals to the medial or lateral side. A frequent pattern of deformity with this type of CN is forefoot abduction together with a flattened medial arch and heel valgus. In case of Eichenholtz Stage 3 and stable tarsal joints, realignment is possible by means of two- or three-dimensional wedge resection. Pure medial fusion may be indicated if the lateral column is spared. Repositioning and achievement of stable fusion may be technically demanding in case of dislocation of all five metatarsals in all directions. Another common pattern is naviculo-cuneiform dislocation with plantar flexion of the talus with the navicular and dorsal dislocation of the first metatarsal with the medial cuneiform. Unresisted pull of the anterior tibial muscle may lead to progressive fragmentation and displacement making conservative treatment even under strict non-weight bearing conditions ineffective, so that early operative intervention may be indicated to restore stability of the medial column. Disagreement exists with respect to the optimal method of fixation, be it a frame, internal osteosynthesis, or a combination of both. There is a consensus that a particular stable fixation is necessary just as for Charcot surgery in general and different from traditional trauma surgery. As any operation in case of Eichenholtz Stage 3 may lead to an acute exacerbation of neuroarthropathy, postoperative immobilization is obligatory by means of a cast or an AFO over a period of several months.
Sanders III
By definition, Stage III involves the midtarsal (Chopart) joint line. Presentation in combination with type II is quite common. A typical deformity pattern for isolated type III is a rocker bottom foot with the cuboid being the lowest lying part of the foot skeleton. As the talonavicular joint holds a key role for biomechanics, coupling the movements of foot and lower leg, exact reduction and fixation are challenging as much as essential. Even if the talonavicular joint shows the most evident extent of dislocation, reduction and fusion of this joint alone is hardly ever sufficient. At the very least, inclusion of the subtalar joint is advisable to minimize rotational forces acting on the talus. In cases of doubt, triple arthrodesis is a guarantor for successful stabilization. Length compensation between medial and lateral column requires subtractive arthrodeses.

Sanders IV
In Sanders IV, the ankle joint and subtalar joint are impaired. Frontal plane deformities in the region of the hindfoot are hard to manage conservatively, particularly in cases of instability. Surgery aims at solid ankle fusion with broad contact area. Taloctomy may be a valuable option in the event of an extensive and rigid deformity to overcome soft tissue contracture. Tibio-calcaneal arthrodesis requires a months-long duration of orthotic after treatment with axial loading of the hindfoot. In terms of functionality, stable fibrous ankylosis is not necessarily inferior to complete bony fusion, as PNP allows pain-free walking in custom-made shoes.

Sanders V
Sanders V involves the calcaneus and constitutes the rarest type of CN. As long as the deformity is stagnant, conservative therapy is favorable, in particular in case of poor calcaneal bone quality with no support for screws or pins. If fragment distance of a calcaneal fracture is increasing due to pull of the Achilles tendon, treatment in a CROW or an AFO is ineffective or leads to a marked deformity. Again, surgery can be performed with a frame or with internal osteosynthesis, in particular with an intramedullary nail. If a nail has caused complications like septic or aseptic loosening with or without fracture, revision surgery can be done with external fixation. In case of impaired skeletal anchorage due to loss of bone substance, external fixation surgery may be considered as the primary treatment option.

Conclusion
This perspective is not a scientific review, nor the least common denominator within a group of diabetic foot surgeons. It is an attempt to develop a future-oriented consensus based on existing scientific literature as well as personal experience. As additional studies continue to expand the knowledge available for operative treatment of CN and its outcomes, more definitive evidence-based recommendations may be established.

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