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 predominantly 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 condition 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.
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 recurrence 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 perinavicular 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. Talectomy 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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