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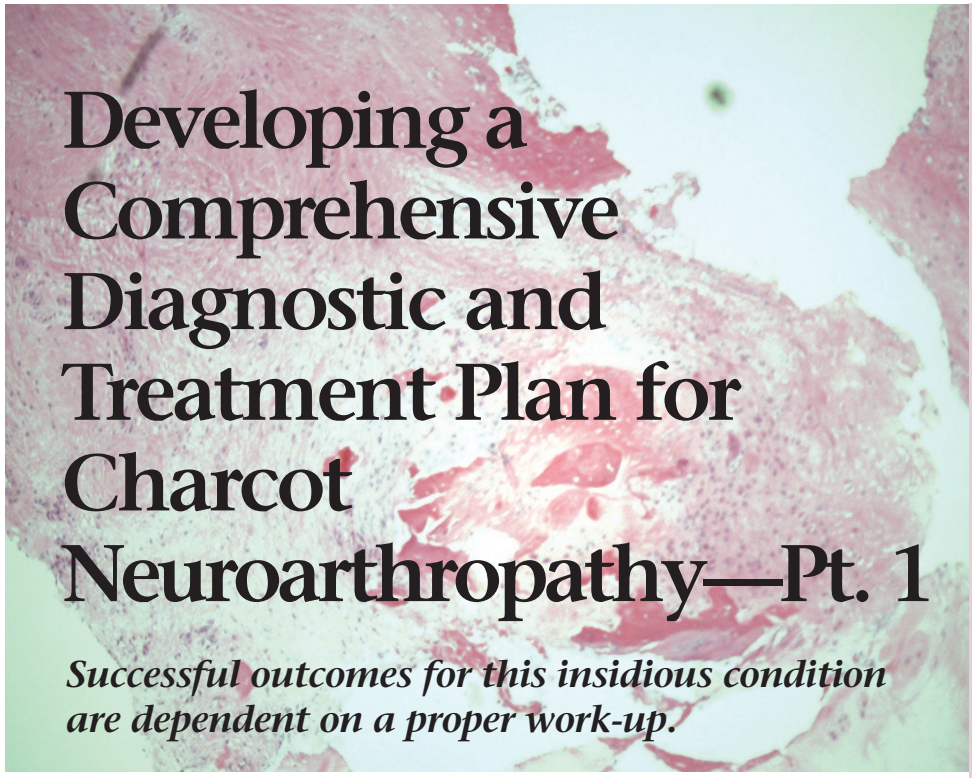

Developing a Comprehensive Diagnostic and Treatment Plan for Charcot Neuroarthropathy—Pt. 1

Successful outcomes for this insidious condition are dependent on a proper work-up.

GOALS and OBJECTIVES

1) The practitioner will be able to discuss the main theoretical causes of neuroarthropathy.

2) The practitioner will be able to list the diagnostic options for neuroarthropathy and identify the gold standard option and implement them in clinical practice.

3) The practitioner will be able to classify a case of neuroarthropathy by both disease stage and anatomic location based on radiographic and clinical examination.

4) The practitioner will be able to formulate a conservative plan for treatment of a case of neuroarthropathy and identify the gold standard off-loading technique.

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*Following this article, an answer sheet and full set of instructions are provided (p. <None>).—**Editor***

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What is Charcot foot?

Jean Martin Charcot first published a description of neuropathic arthropathy in 1868, although William Musgrave first noted arthropathy in syphilitic patients in 1703.[1-3] Charcot neuroarthropathy begins with a neuropathic foot that typically suffers from some type of trauma, sometimes very minor or unremembered. This incident is followed by an acute inflammatory stage with progressive fragmentation of bone and joints, disorganization, and finally collapse of the foot and ankle if weight bearing continues during this stage. The collapse is accentuated by the continued pull of the conjoined triceps tendon. The final stage is character-

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ized by slow resolution of the inflammation with permanent, and sometimes bizarre, deformities remaining that occurred during the prior phase. These can lead to a non-functional and many times chronically ulcerated foot. Synonyms for the disorder are numerous, including Charcot's foot, Charcot's joint, Charcot's fracture, neuropathic osteoarthropathy, and neuroarthropathy, to name a few.

The incidence of neurarthropathy in the *insensate* diabetic population varies in the literature but at specialty centers where the index of suspicion is most accurate, rates can be as high as 13%.[4] Once neuroarthropathy is diagnosed, the incidence of contralateral involvement goes up to 30% either due to some inherent predilection for the process or due to increased pressures on the initially uninvolved extremity. A retrospective analysis comparing mortality and major amputation between Charcot patients and simple diabetic foot ulcer patients was published in 2004. This small study did not show a significant difference in mortality between the two groups, although the amputation rate trend was higher in the Charcot group.[5]

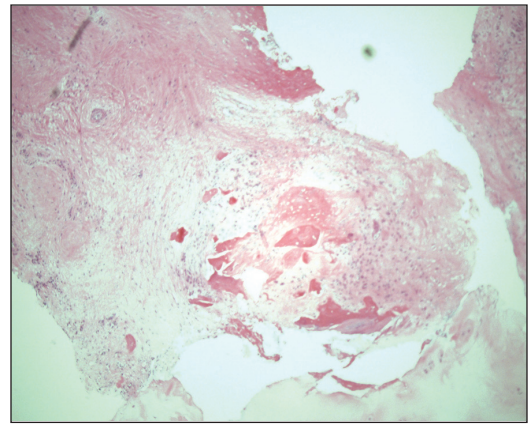

Figure 1: Slide Showing Shards of Bone Ground into the Synovium

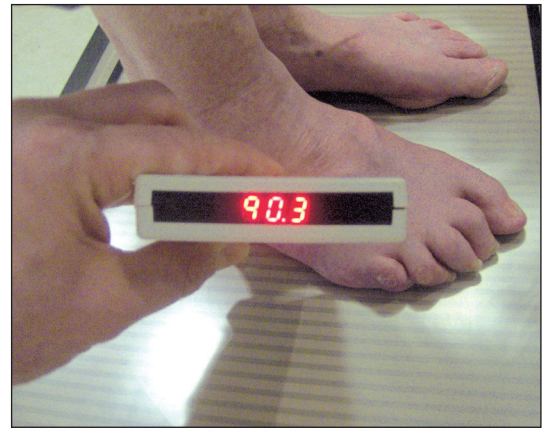

Figure 2: Infrared Dermal Thermometry

What causes Charcot foot?

The two classically opposed theories are the German neurotraumatic theory and the French neurovascular theory. The Germans (via Virchow and Volkmann) believed that the insensitive joints are subject to repetitive microtrauma and finally deterioration. The French (via Charcot) believed that deficiencies in the trophic centers of the spine led to a vasodilatory "washing out" of the bony substance of the extremity. Both theories have been bolstered by animal experiments.[6-7] More recently, clinicians have accepted a confluence of these two theories. Additionally, researchers have investigated the possibility of a pre-existing "diabetic osteoporosis" in setting the stage for neuroarthropathy.[8-13] Jeffcoate has also implicated the possibility of a cycle of pro-inflammatory cytokine release that allows the vicious cycle of inflammation and osteopenia to continue. (14) Lastly, important work has also added the theory of the glycosylated diabetic Achilles tendon to the understanding of the overall picture of the Charcot foot. [15-16]

How is Charcot diagnosed?

Diagnosing an early Charcot's neuro-arthropathic fracture necessitates a high index of suspicion. A neuropathic patient at the earliest stage (Stage 0) will demonstrate subtle signs and symptoms such as edema, pain or very subtle radiographic changes which can be easily mistaken for infection. (Table 1) After quickly ruling out a short list of differential diagnoses such as cellulitis, gout, and etc., (Table 2) one should immediately assume that a neuropathic arthropathy is present and prophylactically immobilize and off-load the extremity while awaiting definitive testing. Both triple-phase bone technitium scans and magnetic resonance imaging are useful to show activity out of proportion to the subtle clinical signs of inflammation to assist in diagnosis.[17-18] All diabetic foot wounds should be carefully explored with a blunt sterile probe.[19] A presumptive diagnosis of osteomyelitis can be made when skeletal structures are exposed, which is bolstered by an elevated erythrocyte sedimentation rate and C-reactive protein.[20-21]

A follow-up bone biopsy and culture will allow appropriate antibiotic treatment of the causative organism and is the gold standard in subtle cases. In cases when the possibility of osteomyelitis versus neuroarthropathy exists, then the synovium should be evaluated as well. (Figure 1) Shards of bone

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TABLE 1

Early Signs and Symptoms of Charcot Neuroarthropathy

Dull, Deep, Unilateral Pain Despite Neuropathy

Crepitus

Sudden Change in Foot Shape

Unilateral Edema

Unilateral Erythema

Unilateral Warmth

Small Fleck Fractures on Plain Radiograph

ground into the synovium is indicative of Charcot. Horowicz wrote the definitive paper on this—and every clinician treating Charcot should be familiar with his paper.[22] It has been our experience that while many authors reference this original paper, this gold standard diagnostic test is underutilized in actual clinical practice. Patients who are diagnosed with both osteomyelitis and Charcot have serial erythrocyte sedimentation rates drawn to monitor the treatment of the bone infection and are followed concomitantly with our infectious disease specialists. Additionally, patients diagnosed with neuroarthropathy should be fully evaluated for the underlying cause of sensorium loss. We've included a list of possible causes of neuroarthropathy in Table 3, although the list is not exhaustive.

Once Diagnosed, How is Charcot Foot Monitored?

Historically, radiographs were monitored as the means to assess the slow consolidation of neuroarthropathy. A more objective and the current standard for serial monitoring is the quantification of inflammatory activity in a neuroarthropathic joint through the use of dermal infrared thermometry (Figure 2) .On a week-to-week basis, temperatures are compared between the affected and unaffected foot (the control) to judge the efficacy of treatment and readiness of the foot for surgery or shoeing. A temperature change of more than two degrees Celsius compared to the surrounding skin or contralateral site has been shown to be a positive indicator of an underlying pathologic condition of the plantar foot.[23-25]

Skin temperatures of the affected foot and contralateral foot are measured after allowing the skin temperature to equilibrate to room temperature for ten minutes after removing cast, brace or shoes. Measurements with a hand-held infrared dermal thermometric probe are taken over the medial and lateral arch, medial and lateral malleoli, the dorsum of the foot and the tibial crest, with care to avoid direct sunlight on the extremities which can raise surface temperatures falsely. This technique has been well described in the literature.[26-32]

Markers of Bone Metabolism

Researchers are on the hunt for markers of bone resorption and formation in serum and urine that would allow clinicians to identify Charcot neuroarthropathy at an earlier stage.[33] Numerous markers of bone metabolism have been evaluated. Selby, et al. measured urinary deoxypridinoline (bone resorption marker) and bone specific alkaline phosphate (bone formation marker) in patients with acute Charcot neuroarthropathy and non-Charcot patients with diabetes. The authors found an increase in both markers in Charcot patients, indicating an ongoing remodeling process of bone resorption and formation.[34] Ulianova, et al. found, however, that while both processes increase in neuroarthropathy, only resorption increases in osteomyelitis, while other authors found that bone turnover markers are not use-

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TABLE 2

Differential Diagnosis

Acute Traumatic Fracture or Dislocation	Superficial Thrombophlebitis
Stress Fracture	Cellulitis
Bone Tumor	Necrotizing Fasciitis
Gout	Abscess
Pseudogout	Osteomyelitis
Degenerative Joint Disease	Septic Arthritis
Plantar Fibroma	Inflammatory Arthritis
Deep Vein Thrombosis	Reflex Sympathetic Dystrophy

TABLE 3

Neuropathies Associated with Charcot Joints

Diabetes	Hemochromatosis
Alcoholism	Antiretroviral Therapy
Spina bifida	Lyme Disease
Myelomeningocele	Hansen's Disease
Syringomyelia	Amyloidosis
Syphilis	Steroid Use
Pernicious Anemia	Spinal Cord Compression
Charcot Marie Tooth Syndrome	Multiple Sclerosis

ful in discrimination between the two entities.[35-36]

We have recently presented data from our own facility that calls into question the usefulness of the markers listed in Table 4.[37] We attempted to correlate abnormal values of the four markers with abnormal infrared pedal temperatures. While trends were present for some of the markers, the correlation was not statistically significant. This, in conjunction with the previously mentioned study showing changes in bone markers in the presence of osteomyelitis, have lead us to consider markers "not ready for prime time." They are currently utilized at our facility as a second-line diagnostic for decision-making in borderline cases, unusual cases or bilateral cases that make thermometry problematic. We plan on correlation of additional markers with thermometry readings in the future.

Figure 3: Technique for Long Leg Reconstructive Plain Radiographs

Radiology

All patients who enter our program undergo a plain radiograph series. In addition to standard views, we obtain the long leg calcaneal axial and hindfoot alignment views on all patients. The usefulness of these views in surgical planning is indispensable and our radiology department was very helpful in modifying their techniques when we brought the articles to them that outlined the techniques (Figures 3 & 4).[38-39] In addition, at St. Luke's, we are able to take advantage of the hospital's relationship with General Electric technology to obtain some of the highest resolution computerized tomograms available. We are lucky in that our hospital has a special relationship with General Electric and the latest in CT scanners and digital imaging are available to our patients. Three-dimensional modeling gives our surgeons the ability to visualize these complex, multi-planar corrections prior to making the incisions. (Figures 5a & b)

Pressure Mapping and Force Measurement

A computerized gait analysis platform is maintained at the center and all patients are evaluated with an initial barefoot static and dynamic image. Any patient who undergoes a surgical intervention is re-evaluated when healed to evaluate the efficacy of the procedure. In-shoe images can also be obtained to assist our pedorthist in shoe and brace modifications (Figure 6).

Classification Systems

Neuroarthropathy can be classified according to both the stage of disease process and the anatomical location. We have not considered the classic Eichenholz and the later Sella and Barrette radiographic staging systems useful in clinical practice due to the lack of correlation with clinical findings and therefore do not use them.[40-41] We use a four-stage system which is a combination of the Armstrong and Lavery pragmatic acute-to-chronic system with the addition of the "pre-Charcot" stage discussed by Yu, et al.[42-43]

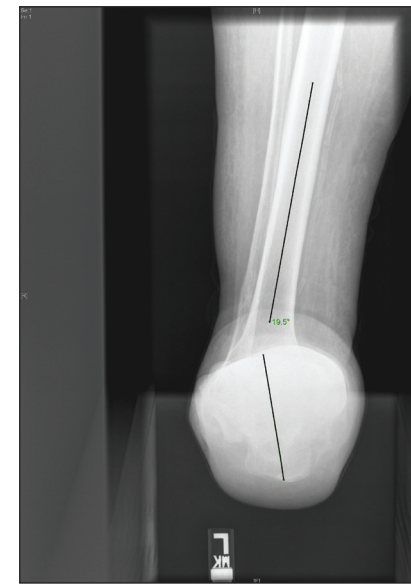

Figure 4: Long Leg Calcaneal Axial View

Our system includes a post-surgical stage to identify those patients who have post-surgical inflammation but are surgically stabilized. (Table 5) The inactive Charcot patients are split into two distinctive groups: those with pathology such as pain, significant deformity; and the non-pathologic group who are ready for shoeing. We believe our system captures those pre-clinical cases of neuroarthropathy that can be "nipped in the bud" and also allows for clear cut decision-making regarding treatment in those pa-

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TABLE 4

Common Bone Markers

Tartrate-resitant acid phosphotase is a glycosylated monomeric metalloenzyme that is used as a bone resorption marker.

Osteocalcin is a protein secreted by osteoblast that is used as a biochemical marker for bone formation.

Bone alkaline phosphate is a hydrolase enzyme responsible for removing phosphate groups from many types of molecules and is used as a biochemical marker for bone formation.

Urinary excretion of deoxypridinoline crosslinks is a biochemical marker for bone resorption.

Neuroarthropy—Pt. 1...

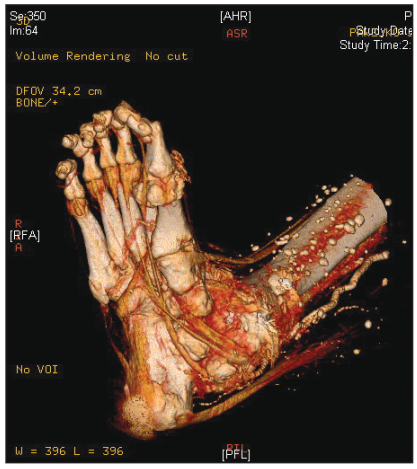

Figure 5a: Three Dimensional CT Scan with Extrinsic Tendon Reconstruction

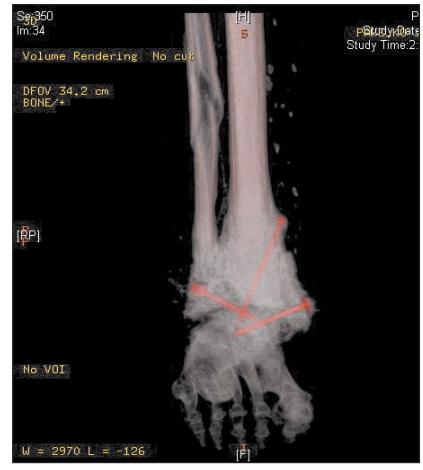

Figure 5b: Three Dimensional CT Scan with Hardware Highlighting

tients with full-blown cases.

Patients enrolled in the program are classified according to both disease stage and anatomic location. There are many anatomic systems available to practitioners.[44-46] Our anatomic classification system is an expanded version of the Sanders system that we have modified to capture cases that we feel need to be treated very differently. In addition to the five classic locations described by Sanders, et al., we separate cases of classic neuroarthropathy from fresh fractures. (Table 6) We feel that this is important because, although these fractures may be treated with a similar protocol as in non-neuropathic fractures initially, they more often than not can trigger on full-blown neuroarthropathy.[47-48] We also identify distal absorptive osteopathy that we see in the phalanges. Lastly, we differentiate between medial and lateral midtarsus disease as the treatment differs for each.

Conservative Treatment Options:

Immobilization and Off-Loading

As long as patients do not have a deep infection requiring immediate debridement, all patients initially begin conservative treatment with the goal of ulceration healing and conversion of the Charcot process from the active phase to the inactive phase, while maintaining the bony architecture of the foot and ankle. The keystone of treatment, just as when treating diabetic foot ulcerations, is immobilization and off-loading. This can be accomplished using a wheelchair, which can create problems with compliance in homes without adequate wheelchair access. Crutches provide another option, but are not easily used by older or obese patents.

We have had success with a product called a Roll-a-bout (Roll-A-Bout Corporation, Frederica, DE, USA) There are different models that can accommodate patients up to 400 pounds and as tall as 6'10". The gold standard, however, is correct application of the classic rigid

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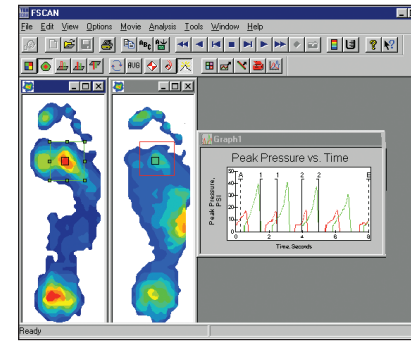

Figure 6: Example of Plantar Pressure Mapping to Assess Orthotic Un-Loading of Ulceration

TABLE 5

St. Luke's Charcot Staging System

Stage 0—Pre-Charcot

Stage 1a—Active Charcot

Stage 1b—Post-Surgical Charcot

Stage 2a—Non-Pathologic, Inactive Charcot

Stage 2b—Pathologic, Inactive Charcot

TABLE 6

Modified Sanders Anatomic Classification System

Level I Forefoot (IPJ, MPJ)
- *A. Absorptive Distal Osteopathy*
- *B. Insidious*
- *C. Traumatic—Metatarsals*

Level II Lisfranc's Joint

Level III Midtarsal Joints
- *A. Medial Column*
- *B. Lateral Column*

Level IV Ankle and Subtalar Joints
- *A. Insidious*
- *B. Traumatic Ankle Fractures*

Level V Calcaneal Insufficiency Fracture

** Modifications to the Sanders System in Italics*

total contact cast as described by Brand.[49-51] (Figure 7) While clinicians have made several modifications to the original technique over the years, such as use of synthetic casting material rather than plaster, the basic device remains the same.[52-53] The cast reduces edema, applies external stabilization of fracture fragments, prevents further injury, enforces compliance; and most importantly, negates the damaging effect of the triceps surae while the foot is in the weakened status of active-phase Charcot. Safe application of a total contact cast requires simple training in either doctoral or post-doctoral programs, or attendance at a casting lab, or proctoring by a specialist familiar with the technique.

Figure 7: Rigid Total Contact Cast

We are amazed at the amount of dogma surrounding the technique. The literature has many references by clinicians who note constraints such as application time, material costs, and risks inherent in the technique. It has been our experience that clinicians make the majority of these statements with either limited clinical contact with patients, or have never actually applied a total contact cast. This single example of "diabetic foot dogma" has limited availability of the best treatment for patients, and possibly cost thousands of diabetic amputations. To this day, however, we see patients with tight Achilles tendons and inflamed, edematous Charcot feet being ambulated in diabetic shoes, Unna boots, or non-custom walking boots. This virtually guarantees gross deformity and failure.

At our program, a total contact cast is applied in approximately 15 minutes. The cost of materials is well under the third-party reimbursement. The efficient application of total contact casts has been discussed by other authors as well.[54] Patients with open wounds receive debridement and application of a slow-release antimicrobial dressing at each cast change. Of course, there will be patients who cannot tolerate the cast for reasons such as active infection, overwhelming drainage, obesity, and claustrophobia, necessitating alternative off-loading devices and techniques. (Table 7). Once the foot is stable, the patient transitions into a removable cast walker or Charcot Restraint Orthotic Walker (CROW) (Figures 8 & 9).[55] Patients progress into extra-depth footgear with custom inserts after one month as long as dermal thermometric measurements remain within two degrees Celsius. Patients requiring tri-plane control will be prescribed ankle foot orthoses to be used within the footgear or a custom-molded high-top shoe with extended shanks, rigid counters, and supra-malleolar bracing. (Figure 10)

A temperature change of more than two degrees Celsius compared to the surrounding skin or contralateral site has been shown to be a positive indicator of an underlying pathologic condition of the plantar foot.

Adjunctive Medical Treatments

In addition to mandatory off-loading, many times we will initiate treatment in patients with an extreme amount of inflammation with a short course of rest, ice, elevation, compression (Jones boot, Unna's boot, or pneumatic compression), and bed-rest. Once formal casting begins, we will always evaluate each patient for adjunctive therapy to shorten the disease process. After evaluating the bone markers and the patient's renal status, augmentation treatment with bisphosphonates (oral or intravenous), salmon calcitonin therapy, or non-invasive bone stimulation will be selected in an effort to attenuate the bone destruction. Overall, the research behind these adjunctive treatments is far from concrete and many questions remain. Our position, however, has been that due to the severe morbidity associated with neuroarthropathy, adjunctive treatment should be offered as long as the risks are low.

Bisphosphonates

Bisphosphonates are potent inhibitors of osteoclast activation. There have been trials showing significant reduction in symptoms and bone turnover markers compared to control groups. They have also been shown to normalize skin tem-

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TABLE 7

Alternative to the Rigid Total Contact Cast

Non-Removable Cast Brace ("Instant Total Contact Cast")

Removable Cast Brace

Patellar Tendon Bearing Brace ("PTB")

Charcot Restraining Orthotic Walker ("CROW")

Crutches

Wheelchair

Bedrest

Knee-Bearing Scooter ("Roll-A-Bout")

Hands- free crutch device ("iWALKFree")

Figure 8: Charcot Restraining Orthotic Walker

perature differential between affected and non-affected feet.[56-60] However, it has been shown that bisphosphonates decrease bone remodeling and are contraindicated in patients with renal insufficiency.[61]

Due to recent questions regarding bisphosphonate-associated osteonecrosis in patients undergoing dental surgery, we have taken the approach that any patient who may possibly need bone surgery on the affected foot will be treated with salmon calcitonin preferentially at the present time.[62-63] Due to the slow clearance of these drugs from bone, these drugs are not given if surgery is being contemplated. The dental literature is being followed closely. We still utilize bone oral and intravenous bisphosphonates in those patients with limited deformity in the acute phase of Charcot when the goal is to arrest the process without any reconstruction.

Both triple-phase bone technitium scans and magnetic resonance imaging are useful to show activity out of proportion to the subtle clinical signs of inflammation to assist in diagnosis.

We initially used oral bisphosphonates such as Fosamax, Boniva, and Actonel exclusively in the acute phase of CN. We have used IV Pamidronate sparingly. We now require a full dental examination prior to considering bisphosphonate therapy. The majority of our patients are now prescribed calcitonin-salmon spray daily, alternating nostrils each day. Researchers have shown this treatment to be beneficial in driving down the markers of bone metabolism.[64] Calcitonin-salmon nasal spray has been used to increase vertebral bone mass in post-menopausal osteoporosis and to decrease the incidence of vertebral fractures.

Figure 9: Cast Walker Immobilization with External Bone Growth Stimulator

The treatment adverse reactions are mild to moderate in severity (mostly local nasal complaints). We've noted an anecdotal decrease in pain in the involved extremity when utilizing this therapy. It can be used at any time of the day without regard to food restrictions or supplementation. The previous research with calcitonin-salmon spray did not, however, drive down the skin temperature faster than the control, and questions remain. We hope to add to the data being gathered on this adjunct.

Electrical Bone Stimulation

Harshorne reported the use of electrical energy to directly stimulate bone healing in 1841.[65] Yasuda, et al.[66] studied electrical fields and bone formation in the early 1950's. They were able to demonstrate the development of subperiosteal callus in bones under mechanical stress. The callus was formed as a result of the electrical potentials induced by the mechanical stress (piezoelectricity). Electronegative potentials are generated in areas of compression and electropositive potentials are generated in areas of tension. They were then able to show that passing 10 µA of continuous current along the bone could result in similar callus formation. Increased osteoblastic (bone formation) activity would be seen on the concave side of the bone, which has an electronegative potential. This is why the cathode negatively-charged electrode is placed at the site of nonunion or at the fracture site.[67] The amount of current is dose specific; currents less than 5 µA do not cause bone formation, currents of 5 to 20 µA produce progressively increasing amounts of bone formation, currents over 20 µA produce necrosis.[68-69]

Current can be delivered to the bone by either direct current or by intermittent pulsed electromagnetic fields. Direct current devices are surgically implanted where as intermittent pulsed electromagnetic field devices are noninvasive and deliver current by means of two opposing coils of wires mounted on a cast or skin. The coils face each other at 180 degrees.[68-69]

Research has shown some benefit in the use of stimulation of bone healing through electrostimulation, magnetic fields, and low intensity ultrasound when treating neuroarthropathy.[70-74] We typically utilize this adjunct when traumatic fractures that morph into neuroarthropathy, long bone fractures, or high risk fusions exist in the clinical picture. We utilize pulsed electromagnetic field stimulators (EBI Medical Inc, Parsippany, NJ, USA) to speed up bone formation. Direct current devices have been used with success in a few surgical patients with high risk tibiocalcaneal fusions implanted directly into the fusion site.

Patient Education, Lifestyle and Disease Modification

One of the main reasons that we have excellent salvage rates and functional extremities is due to the

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"whole picture" approach that we take with these patients. Applying a total contact cast and prescribing a bisphosphonate in a patient who is blind, with poor dentition, in chronic renal failure, has glycosylated hemoglobin of 10, and must drive himself to and from doctors' visits is doomed to failure and complications. At the initial enrollment of these patients, we look at many factors that will enhance the treatment of the neuropathic joint as well as decrease overall morbidity in the patient (Table 8). We have developed patient education sheets on Charcot and casting, as well. While the cast application is simple and straightforward, these last components of our conservative treatment program are certainly the most challenging and utilize the most time and resources.

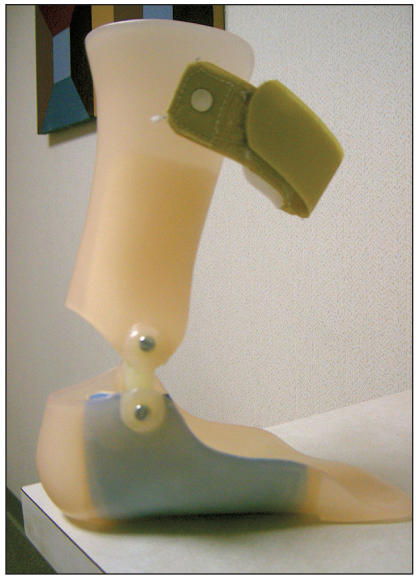

Figure 10: Molded Foot and Ankle Orthosis

Prevention of Recurrence?

A patient with two consecutive visits with equal and symmetrical foot temperatures is considered to be in the inactive phase. Patients are then evaluated for chronic pain, instability, equinus or bony deformity that would preclude safe and comfortable ambulation with shoes. If any of these exist, then surgery is contemplated. From the outset, we know that any patient with a history of neuroarthropathy has high risk for re-activation of the process in the same foot as well as a 30% chance of developing similar problems in the contra-lateral foot. We educate the patient on this as well as review the signs and symptoms of the process. We also educate the patient on risk factors particular to their neuro-arthropathy. (Table 9) Lastly, we make sure that we make an appointment for the patient to go to his/her podiatrist for regular high-risk foot care. ■

Editor's Note: *Part 2 will appear next month.*

TABLE 8

Patient Education

- Charcot Neuro-arthropathy disease process
- Treatment options
- The Necessity of Off-loading
- Compliance with treatment plan
- Diagnostics
- Blood glucose control (HBgA1c)
- Hand controls for car
- Residential Modifications (Ramps, Grab Bars, etc.)
- Weight Management and Conditioning
- Dilated Eye Examinations
- Dental Examination if Bisphosphonates Are Planned
- Smoking Cessation
- Pain Management
- Depression Management

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Continued on page 160

TABLE 9

Risk Factors for Neuroarthropathy

Clinical Signs of Peripheral Neuropathy:[75]
- Insensate to Monofilament
- Decreased Vibratory Sensation
- Decreased Deep Tendon Reflexes

Patient History of:
- Retinopathy
- Nephropathy
- Previous Foot Ulcer
- Neuro-arthropathy

Activities / Events including: (112)
- Use of Ladders or Digging Tools (i.e., Shovels)
- Obesity
- Lifting Heavy Objects
- Sudden Change in Activity Level
- Impact Sports/Activities (Jogging, Stair-Climber, Dance)
- Osteopenia
- Foot or Ankle Surgery of any kind
- Traumatic Fractures of the Foot or Ankle
- Minor Trauma (sprains, contusions, etc.)

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Dr. Bernstein is board certified by the American Board of Podiatric Surgery and is a Fellow of the American College of Foot and Ankle Surgeons. He graduated from Temple University School of Podiatric Medicine and completed both a residency in foot surgery and a fellowship in limb salvage surgery with Dr. Stanley Kalish in Atlanta, Georgia. He currently practices in the Lehigh Valley and is program director of the Charcot and Reconstructive Foot Program at St. Luke's Hospital and Health Network, Quakertown Campus. Dr. Bernstein participates in mission trips to impoverished regions to perform pediatric deformity surgery on a yearly basis.

John Motko is a registered nurse who works at the Wound Management Center St. Luke's Health Network, Quakertown Campus. He has a BS in Nursing from Moravian College/ St. Luke's School of Nursing. He is certified in wound care from both the American Academy of Wound Management and the Wound, Ostomy and Continence Nurses Society. He is also a Certified Hyperbaric Registered Nurse. He has over seven years of clinical experience in caring for patients with chronic non-healing wounds and Charcot neuroarthropathy.

EXAMINATION

Continuing Medical Education

See answer sheet on page 163.

1) Urinary excretion of deoxypyridinoline crosslinks is a biochemical marker for bone formation.
   a) True
   b) False
   c) only when patient is diabetic
   d) only when patient is a non-diabetic

2) Infrared dermal thermometry should be completed:
   a) immediately after the cast, shoe or brace is re moved to ensure accurate skin temperatures.
   b) 24 hours after cast removal
   c) 10-20 minutes after cast removal
   d) directly through the casting material

3) When obtaining infrared dermal temperatures to track the activity of a neuropathic fracture, one should:
   a) Compare the affected foot temperatures to other areas on the affected foot
   b) Compare the affected foot temperatures to corre sponding areas on the unaf fected foot
   c) Compare the affected foot temperatures to the patient's oral temperature
   d) Compare the affected temperatures to a non-Charcot patient's temperatures

4) Stage 0 Charcot Neuroarthropathy is typically characterized by the following:
   A) Marked Deformity
   B) Edema and Warmth
   C) Massive Disorganization of Osseous Structures on Plain Radiograph
   D) Fever, elevated white count, high erythrocyte sedimentation rate

5) Achilles tendon contracture is associated with Charcot Neuroarthropathy:
   A) Occasionally
   B) Only in insulin-dependent patients
   C) Often
   D) Never

6) Acceptable means of immobilization of an acute Charcot foot are:
   A) Custom diabetic shoes
   B) Total Contact Cast
   C) Unna Boot, Post-Operative Shoe and Partial Weightbear ing with Cane
   D) Extra-Depth Shoes

7) The german theory of neuroarthropathy links Charcot with:
   A) Repetitive microtrauma
   B) Vasodilation
   C) Pro-inflammatory cytokines
   D) Nephropathy

8) What is the gold standard test for diagnosis of neuroarthropathy?
   A) bone culture
   B) sedimentation rate
   C) Synovial biopsy
   D) Serum bone markers

9) The following are possible underlying causes of neroarthropathy:
   A) alcoholic neuropathy
   B) Psoriatic arthritis
   C) Peripheral vascular disease
   D) Raynaud's phenomenon

10) Bisphosphonate therapy should not be offered when the patient is suffering from:
   A) Dental pathology
   B) herpes zoster
   C) peripheral vascular disease
   D) xerosis of the skin

11) The Sanders classification system involves:
   A) disease stage
   B) anatomic location
   C) diabetes control
   D) ulcer depth

12) The Eichenholz classification system involves:
   A) disease stage
   B) anatomic location
   C) diabetes control
   D) ulcer depth

13) The Eichenholz classification system utilizes:
   A) patient pain profiling
   B) radiographic presentation
   C) infrared dermal thermometry
   D) all of the above

14) A prerequisite for neuroarthropathy is:
   A) trauma
   B) obesity
   C) poor glucose control
   D) peripheral neuropathy

Continued on page 162

Continuing Medical Education

EXAMINATION

(cont'd)

15) Patients with Charcot neuroarthropathy have blood flow that is:
- A) Always excessive compared to normal baseline
- B) Varies based on comorbidities, family history and age
- C) Normal
- D) Generally decreased compared to normal baseline

16) Contralateral neuroarthropathy occurs:
- A) 5% of the time
- B) 30% of the time
- C) 80% of the time
- D) Never

17) The differential diagnosis of Charcot should include:
- A) cellulitis
- B) necrobiosis lipoidica diabeticorum
- C) diabetic dermapathy
- D) diabetic bullosis

18) Contracture of the Achilles tendon exists in Charcot patients:
- A) Never
- B) Rarely
- C) Some of the time
- D) Most of the time

19) Patient should not transition from total contact cast to shoegear until:
- A) Infrared temperatures are within 2 degrees Celcius bilaterally
- B) Bone markers are normalized
- C) Three months have elapsed from first cast
- D) The patient promises not to walk too much

20) Patients should be transitioned from total contact cast to shoegear after temperatures are equal and symmetrical:
- A) Immediately
- B) Gradually while temporizing with a transitional device such as a CROW or cast boot
- C) Never
- D) When glucose is under control

See answer sheet on page 163.

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Developing a Comprehensive Diagnostic and Treatment Plan for Charcot Neuroarthropathy–Pt. 1 (Bernstein and Motko)

Circle:

1.	A	B	C	D	11.	A	B	C	D
2.	A	B	C	D	12.	A	B	C	D
3.	A	B	C	D	13.	A	B	C	D
4.	A	B	C	D	14.	A	B	C	D
5.	A	B	C	D	15.	A	B	C	D
6.	A	B	C	D	16.	A	B	C	D
7.	A	B	C	D	17.	A	B	C	D
8.	A	B	C	D	18.	A	B	C	D
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