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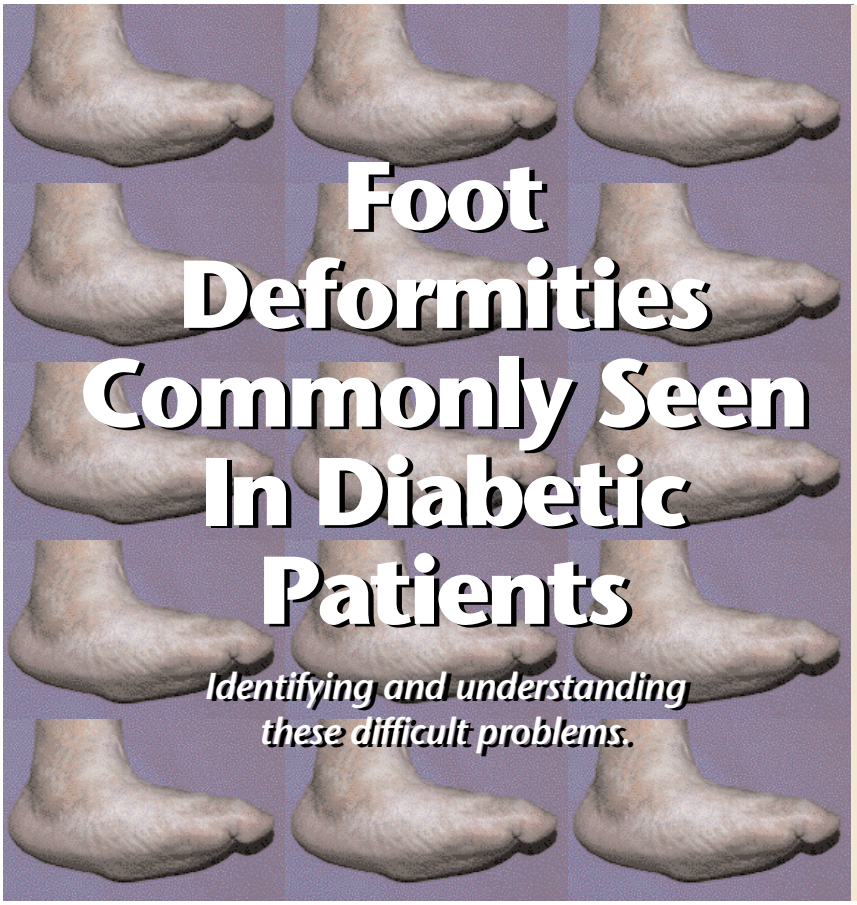

Foot Deformities Commonly Seen In Diabetic Patients

Identifying and understanding these difficult problems.

Objectives

After reading this continuing education article, the podiatric physician should be able to:

1) Be familiar with the forefoot deformities that are commonly seen in patients with diabetes mellitus.

2) Recognize and describe in detail skeletal changes of diabetic feet on plain radiography.

3) Understand the pathogenesis of Charcot osteoarthropathy and the series of events that result in the development of Charcot joint.

4) Know the characteristic anatomic patterns of bone and joint destruction which have been observed to occur in diabetics with charcot osteoarthropathy.

5) Be familiar with the anatomic location of deformities associated with patterns of bone and joint destruction in Charcot osteoarthropathy.

6) Confidently recognize radiographic changes of bone and joint destruction in Charcot osteoarthropathy.

7) Discuss an objective rationale of treatment based on acute versus chronic cases of Charcot joint.

8) Discuss immobilization options for the treatment of Charcot osteoarthropathy.

9) Know when surgical versus conservative treatment is indicated for Charcot joint.

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*Following this article, an answer sheet and full set of instructions are provided (p. 128).—**Editor***

By Jack Haddad, D.P.M.

In a population-based survey, more than 50 percent of patients with type I diabetes aged 15-50 years were found to have foot deformities. Among the majority of these deformities were collapsed arches and hammertoes.[1] Motor neuropathy was an in important factor in the etiology of these deformities because varying degrees of collapsed arches and hammertoes were significantly associated with increased sensory thresholds for perception, pain, and vibration. Furthermore, diabetic motor neuropathy results in weakness of the intrinsic muscles of the foot, thereby causing subtle changes in posture and gait.

Although the pathogenesis on foot deformity in diabetic patients is not well understood, the literature

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strongly suggests a combination of limited joint mobility, pressure resulting from motor neuropathy, and resorption of bone. Mueller et al.[2] have documented limited joint mobility in the feet of diabetic patients, which may contribute to increased peak plantar pressures by making the foot less flexible and less tolerant of changing proteins. Moreover, limited joint mobility of the ankle, subtalar, and metatarsophalangeal joints has been studied in cross sections of diabetic and non-diabetic patients by several authors.[2,3] Patients with a history of neuropathic ulceration exhibited less mobility than those without ulceration or non-diabetic controls. However, one must be advised that it is quite difficult to correlate limited joint mobility with the severe hypermobility that is frequently observed in Charcot neuroarthropathy.

According to a study by Campbell R. et al.,[4] abnormal collagen formation related to non-enzymatic connective tissue glycation may be associated with limited joint mobility, and occurs more frequently in diabetic than non-diabetic patients. Also, several publications reported on the non-enzymatic glycosylation of proteins, particularly hemoglobin and dermal collagen.[5,6]

Also, health professionals are now increasingly more aware of the mechanical aspects of foot deformities which might have an impact on the occurrence, potential healing, and recurrence of foot ulcers.

A study by Smith et al.[7] reported on the prevalence and rate of severity of radiographic foot deformities in the feet of 456 diabetic patients. The prevalence of Charcot changes was 1.4 percent (six subjects) and all had radiographic evidence of midfoot Charcot changes. Other more common deformities included were hallux valgus, alterations in height arch, lesser toe joint dislocations, and clawtoes.

A number of reports exist in the literature of isolated abnormal radiographic findings in diabetic patients. Williams et al.[8] have reported a significantly higher prevalence of periosteal reaction in the metatarsals of diabetic patients who did not have infection compared with non-diabetic control subjects. Periosteal reaction is sometimes regarded as evidence of response to an infection. In the absence of skin lesions, an alternative explanation to periosteal reaction is elevated mechanical stress. A strong association exists between increased bending stress in long bones and periosteal reaction.[9] This is a plausible explanation for periosteal reaction in patients with diabetes because it is now widely accepted that diabetic neuropathic patients have elevated loads under the metatarsal heads.[9]

It is widely believed that traumatic fractures in diabetic patients with neuroosteoarthropathy lead to a progressive Charcot process.

Generalized demineralization and focal osteolysis are skeletal changes that must be evaluated when interpreting plain radiographs. But, first, it is important to bear in mind that sufficient osseous vascular supply must be present in order for bone to undergo resorption.[10] Therefore, destructive bone resorption in diabetic patients can only be observed in patients with an adequate blood supply.

The diabetic foot may display diffuse demineralization in which all of the foot bones are affected, or a focal osteolysis may begin as a defect. The defect is located most often in the metatarsal heads and phalanges of the foot, sharply marginated and local, and measures 1 to 5mm in diameter. The defect may remain stable for years or progress rapidly to massive osteolysis. The osteolytic defect generally begins in the metaphysis and subsequently spreads into the epiphysis, spearing the diaphysis. At first, the end of the diaphysis may be ragged, but becomes pointed as the lesion progresses, forming a pencil-ing or candlestick-like configuration. The articular surface is usually the last to be resorbed.

It remains uncertain as to what causes these destructive bony changes. Researchers and clinicians are divided in their views on the etiology of resorptive bone changes occurring in diabetic patients. Multiple factors possibly relevant in the development of Charcot neuroarthropathy include metabolic perturbations, renal disease and transplantation, osteoporosis secondary to corticosteroid injections, decreased cartilage growth activity, non-enzymatic glycosylation of bone proteins, and imbalance of osteoclastic-osteoblastic activity.

In a study investigating markers of osteoblastic and osteoclastic activity in diabetic patients with osteoarthropathy, Gough et al.[11] discovered significantly higher levels of osteoclastic activity in patients with Charcot feet than those found in health subjects and diabetic controls.

These results augment other studies which report lower bone mineral density in lower limbs of Charcot patients than that found in control subjects. Forst et al.[10] found diminished bone mineral density in the limbs of type I diabetic patients as compared to controls.

Several case reports and experimental data exist in the literature which lends support for the hypoth-

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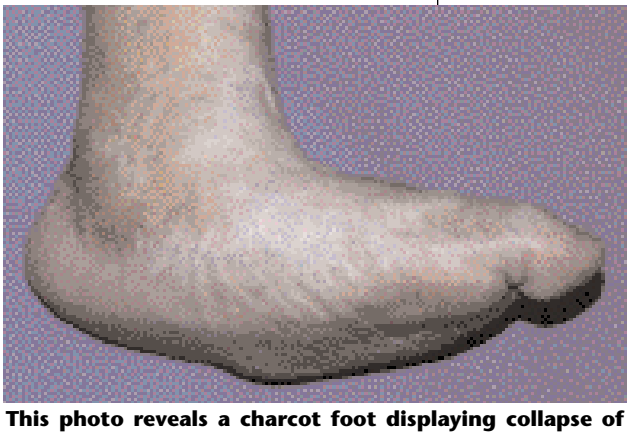

This photo reveals a charcot foot displaying collapse of the midfoot and a rockerbottom sole.

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esis that increased blood flow and active bone resorption are responsible for the development of Charcot neuroarthropathy. In three cases reported by Edelman et al.,[12] Charcot osteoarthropathy was developed within 2-5 years after the subjects had lower limb revascularization.

Clinicians often report that their patients have warm feet with bounding dorsalis and posterior tibial pulses. Archer et al.[13] noted an increased foot skin temperature in 22 diabetic patients with sensory neuropathy using mercury strain gauge plethysmography and doppler sonogram. Their findings reflected an increased blood flow that was five times greater than normal at 20 to 22 degrees C.

Repetitive moderate stress and repetitive impulse loading on an insensate foot causes tensile fatigue of cartilage and bone, resulting in soft tissue injury (ulceration). The earliest sign in cartilage damage is trabecular microfractures in the subchondral bone. This change in bone and joints and alterations in foot mechanics is another contributor to Charcot neuroarthropathy. Cavanagh et al.[14] examined patients with diabetic peripheral neuropathy and determined that foot deformities are a major determinant of increased peak plantar pressure and subsequent ulceration. To that end, a deformed Charcot foot places an individual at risk for ulceration. Patients often complain, "My arch is falling." Clinically, the medial longitudinal arch of the foot is observed to be depressed, as total collapse of the foot occurs over a short period of time.

In the presence of overlying soft tissue infection and ulceration, the differentiation of Charcot osteoarthropathy from osteomyelitis is often difficult.

It is widely believed that traumatic fractures in diabetic patients with neuroosteoarthropathy lead to a progressive Charcot process. In the series of 118 Charcot patients presented by Johnson,[15] conclusions were drawn that fractures were of major importance in initiating the destructive process in the majority of cases. He concludes that as long as the trauma of repetitive stress and weight bearing continues, resorption outpaces new bone formation.

There has been a causal association between corticosteroid or immunosuppressive treatment and the development of Charcot neuroarthropathy, though no longitudinal studies of a large diabetes population has been able to determine the true role of these agents in the etiology of neuroarthropathy. Clohisy and Thompson[16] reported on 18 patients with type I diabetes who have severe neuroarthropathy of the ankle and tarsus. Fourteen of these patients had received a renal transplant before a fracture was diagnosed, and none had a history of major trauma. Further research is warranted to determine whether bone weakened by corticosteroids is the underlying factor responsible for the development of destructive charcot neuroarthropathy in these patients.

Another relatively common radiographic finding in diabetic and obese patients is diffuse idiopathic skeletal hyperostosis (DISH).[17] Although the process is most commonly associated with spinal abnormalities, osseous excrescences of the foot and heel are present in most patients or may be seen in diabetic patients without DISH.

Charcot neuroarthropathy is a non-infective, destructive bone and joint fracture or dislocation associated with a peripheral neuropathy. There are widely varying estimates in the literature for the prevalence of Charcot neuroarthro-

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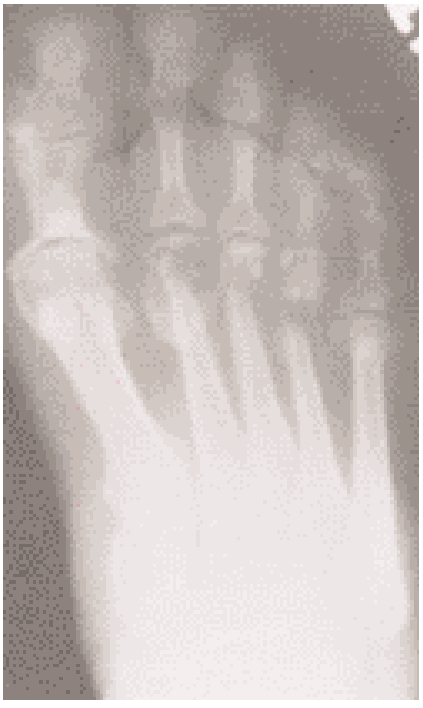
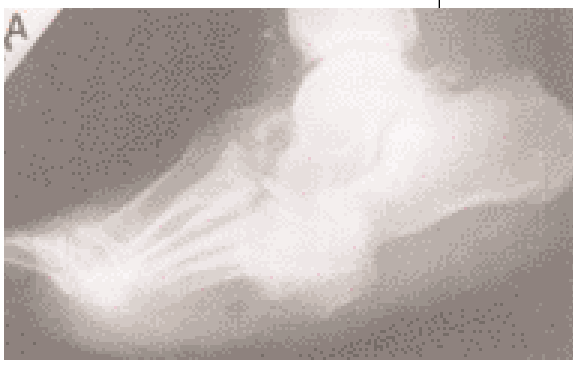


This is a 60 year-old diabetic patient with a charcot foot. Note the classic osteolytic bone changes involving the distal segment of the metatarsals and the ankle.



This charcot foot displays collapse of the tarsometatarsal , naviculocuneiform, talonavicular, and calcaneocuboid joints.

This photo demonstrates an evulsion fracture of the calcaneus of the posterior tubercle. The arrow is pointing in the direction of the pull of the Achilles tendon. Osteolytic changes are also appreciated at the naviculocuneiform joint.

pathy, a condition that is poorly understood and poses a formidable diagnostic and treatment challenge for all members of the health care team. Moreover, the condition is regarded as one of the most devastating foot complications of diabetes. Literature review has reported on the prevalence of Charcot neuroarthropathy associated with diabetes to from 0.08 percent to 7.5 percent.[18,19,20] It is very likely that there many more cases of Charcot neuroarthropathy that go unrecognized or misdiagnosed, as the condition is frequently an overlooked complication of diabetes.

The majority of patients diagnosed with Charcot neuroarthropathy are in their sixth and seventh decades, with an average age of 57 years of age.[21] At the time of diagnosis, the average duration of diabetes in a patient is approximately 15 years.[21]

Men and women are affected equally, and seventy-five percent of the cases reported have bilateral involvement.[21] Because Charcot is reported to be mainly bilateral, recognition of unilateral cases could lead to enhanced surveillance and therapeutic efforts to prevent progression to bilateral disease.

Information on the patterns of bone and joint destruction in Charcot neuroarthropathy has been compiled by Sanders and Frykberg[22] from a variety of English medical literature sources.

Pattern I is commonly characterized by involvement of the forefoot, the interphalangeal joints, phalanges, and metatarsophalangeal joints, or distal metatarsal joints. Of the affected joints (20 of 66) in the Sanders and Mrdjenovich[23] study, 30 percent was found to be pattern involvement. Of 116 affected limbs, Cofield et al.[24] reported 67 percent involvement in metatarsophalangeal and interphalangeal joints. Also, 91 percent of their patients with metatarsophalangeal joint involvement had underlying ulcers. Sinha et al.[25] reported metatarsophalangeal joint involvement in 26.8 percent (34 of 127) of all affected sites.

Pattern II involves the tarsometatarsal joint (Lisfranc s joint) and is often associated with plantar ulceration at the apex of the collapsed cuneiforms or cuboid. In charcot neuroarthropathy, 15 to 48 percent of the cases reported involved the tarso-metatarsophalangeal joint.[26]

Pattern III is characterized by involvement of the midtarsal joints and naviculocuneiform joints. There is usually dislocation or disintegration of the naviculocuneiform joints. Of the affected joints (21 of 66), Sanders and Mrdjenovich[23] reported 32 percent involvement of the naviculocuneiform, talonavicular, or calcaneocuboid joint.

Pattern IV involves the ankle or subtalar joint and usually accounts for only 3 to 10 percent of the Charcot cases.[23,24,25,26] With this pattern of Char-

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cot neuroarthropathy, even trivial trauma associated with an ankle sprain or a minor fracture may result in severe structural deformity and functional instability of the ankle.

Pattern V has been the least frequently reported pattern of bone destruction seen in Charcot neuroarthropathy.[24,25,26] It involves the calcaneus and is characterized by an avulsion fracture of the posterior tubercle of the calcaneus. Kathol et al.[27] reported on 21 diabetic patients with calcaneal fractures, of which 18 were non-traumatic and 14 were limited to the posterior third of the calcaneus.

In the presence of overlying soft tissue infection and ulceration, the differentiation of Charcot osteoarthropathy from osteomyelitis is often difficult. Charcot should be suspected when bone and joint changes are found in a diabetic patient with peripheral sensorimotor neuropathy, loss of protective sensation, absent deep tendon reflexes, diminished vibratory perception, and muscle weakness. Therefore, a complete history and physical coupled with radiographic findings should help narrow down the differential diagnosis. Also, radionucleotide and magnetic resonance (MRI) examinations, bone biopsy, bone culture, and histopathologic examination are specific diagnostic tools for distinguishing between Charcot neuroarthropathy and osteomyelitis in a diabetic patient.

During the initial stage of a Charcot process, plain radiographs may reveal mild inflammation of the soft tissue and or skeletal changes consistent with osteoarthritis or osteolytis. However, radiographic films should be repeated within two to three weeks to rule out fragmentation of bone, periosteal new bone formation, and stress fractures, especially in cases where there is suspicion of an injury and the initial plain films are negative.

Radiographic findings of a Charcot joint classically reveal extensive osseous destruction with little or no demineralization. There may be osseous fragments scattered throughout the soft tissue. Also, sclerotic bone is evident.

MRI is particularly helpful in differentiating neuroarthropathy from osteomyelitis. In examining patterns for Charcot s joints, Beltran et al.[28] noticed a low signal intensity on T1-T2 weighted images within the bone marrow space adjacent to the involved joint. MRI in cases of a Charcot is particularly helpful in visualizing an ulcer penetrating through the plantar aponeurosis.

In osteoarthropathy, diffuse and focal uptake of technetium has been reported in neuropathic feet in areas of active bone turnover and increased bony blood flow.[29]

Gallium-67 citrate which accumulates in sites of infection has also been reported to localize in non-infected neuropathic bone.[30]

In considering management strategies of a Charcot foot, the following should be sought carefully:

Lesko and Maurer suggested immobilization and protective weight bearing for acute Charcot with acceptable alignment.

1) acuteness of symptoms 2) anatomic pattern of bone and joint destruction, and 3) presence of infection. The aim of treatment should be to obtain stability of the foot with no excessive pressure on the skin from a bony prominence. Also, the key is to allow the acute stage of a Charcot process to convert to the reparative (quiescent) stage.

Lesko and Maurer[31] outlined an objective rationale of treatment based on the above considerations. They suggested immobilization and protective weight bearing for acute Charcot with acceptable alignment. The immobilization is necessary to prevent the destruction of bone and collapse of joints.

At least 3 months of non-weight bearing cast immobilization is required before the resumption of partial weight bearing in a therapeutic shoe or walking brace. Immobilization should continue until warmth of he skin subsides. Areas of increased warmth correspond to areas of inflammation. A hand-held infrared digital thermometer is a useful tool for the physician to monitor the inflammatory response in these patients. an increase in skin temperature greater than 2 degree C should be considered a significant finding indicating impending neuroarthropathy.[35]

Recent data suggests that non-weight bearing should initially be prescribed because ambulatory casting might not be as effective. A study by Shaw et al.[32] indicated that approximately one third of the total load applied to the casted extremity is transmitted to the leg via the walls of the cast.

Orthoses used together with therapeutic shoes may effectively decrease load on the foot. Saltzman et al.[33] examined the peak force transmitted to the foot in six diabetic patients with Charcot arthropathy. Their results revealed a lower peak force to the foot by 15 percent with the use of an added-depth shoe together with a Patellar tendon-bearing (PTB) orthosis.

The effectiveness of walking braces has been cited in the literature recently in reducing peak plantar pressure. Compared to both cast and shoe, greatest reductions were found in the forefoot while pressure-time integrals were actually increased in the heel region.[34] Landsman reported on the use of a custom-fabricated padded ankle foot orthosis (AFO) in patients with ulceration and midfoot Charcot. The AFO provided a reduction in peak pressure at the midfoot ulcer sites ranging from 70 to 92 percent, allowing the ulcer to heal in an average of nine weeks.

The custom fabricated Charcot restraint orthotic walker (CROW) is a bivalved, total-contact, full-foot enclosure AFO that consists of a poly-propylene shell and rocker sole. After an initial period of non-weight bearing treatment modality, a patient can benefit by having effective ankle and foot immobilization, and control of edema.

Surgical arthrodesis and reduction should be ascertained for Charcot cases that exhibit acute dislocа-

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tion with marked deformity, and bone fragmentation. In cases of chronic dislocation with severe deformity and bone fragmentation, surgical intervention is recommended only if soft tissue breakdown cannot be prevented by therapeutic footwear and bracing.

In the final analysis, data on the prevalence of structural foot deformities in patients with Charcot neuroarthropathy may assist researchers in studying the impact of these structural deformities on altered foot mechanics, pressure distribution, and incidence of diabetic foot ulceration.

Furthermore, although studies have been conducted to assess pressure at various parts of the diabetic and non-diabetic foot, further research is needed to quantify the pressure distribution as it relates to foot type or available foot and ankle range of motion during gait. ■

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EXAMINATION

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See answer sheet on page 129.

1) Which of the following is true regarding the immobilization of a Charcot foot?
   A) Prevent the destruction of bone and collapse of joints.
   B) Reverse the destruction of bone and collapse of joints.
   C) Only A
   D) None of the above.

2) Which imaging modality is most helpful in visualizing an ulcer penetrating through the plantar aponeurosis of a Charcot foot?
   A) CT scan
   B) MRI
   C) Both A and B
   D) Gallium-67 citrate

3) Which finding(s) leads most to the suspicion of Charcot osteoarthropathy in a diabetic patient?
   A) Glucose blood level of 300> than 1 year
   B) Radiographic changes of bone, peripheral sensorimotor neuropathy, absent deep tendon reflexes, and muscle weakness.
   C) Systemic infection
   D) None of the above.

4) Which of the following is a relatively common radiographic finding in diabetic patients?
   A) Diffuse idiopathic skeletal hyperostoses (DISH)
   B) Increased bone density
   C) Ewing's sarcoma
   D) All of the above

5) All of the following are true of patients diagnosed with osteoarthropathy except:
   A) Patients are usually in their sixth or seventh decade of life.
   B) Men and women are affected equally
   C) Unilateral involvement is most common
   D) Bilateral involvement is most common

6) Which is true concerning the foot joints in a patient with diabetes?
   A) Hypermobile joints
   B) Limited joint mobility
   C) None of the above
   D) All of the above

7) All of the following are common foot deformities seen in a diabetic patient except:
   A) Hammertoes
   B) Hallux valgus
   C) Collapsed arch
   D) Club foot

8) What skeletal changes could the diabetic foot display?
   A) Diffuse demineralization
   B) Focal osteolysis
   C) Both A and B
   D) None of the above

9) What must be present to initiate the destructive bone resorption process in a diabetic patient?
   A) Sufficient osseous vascular supply
   B) Decreased blood glucose level
   C) Absence of neuropathy
   D) All of the above

10) Which of the following is widely seen in a patient with osteoarthropathy of the foot?
   A) Fractures
   B) Increased joint laxity
   C) Increased bone density
   D) Bone tumors

11) Diabetic motor neuropathy results in which of the following?
   A) Weakness in intrinsic muscles of the foot
   B) Subtle changes in posture and gait
   C) Both A and B
   D) None of the above

12) Which of the following is true regarding periosteal bone reaction in the diabetic foot?
   A) Strongly associated with elevated mechanical stress in bone
   B) Sign of increased blood flow
   C) All of the above
   D) None of the above

13) What part of the bone is spared when an osteolytic defect develops in a diabetic foot?
   A) Metaphysis
   B) Diaphysis
   C) Epiphysis
   D) All of the above

14) What is the rationale on the use of walking braces for the treatment of a Charcot foot?
   A) Reduce peak plantar pressure
   B) Increase mobility of joints
   C) Both A and B
   D) None of the above

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EXAMINATION

(cont'd)

15) Which of the following aid in reducing the peak plantar pressure of the foot?
- A) Extra-depth shoe
- B) Patellar-tendon-bearing (PTB) orthoses
- C) Only B
- D) A and B

16) Which of the following should be sought carefully when considering management strategies of a Charcot joint?
- A) Acuteness of symptoms
- B) Presence of an infection
- C) Anatomic pattern of joint destruction
- D) All of the above

17) What are radiographic findings of a Charcot osteoarthropathy?
- A) Little or no demineralization of bone
- B) Sclerotic bone
- C) Osseous destruction
- D) All of the above

18) Which of the following aid in distinguishing between osteoarthropathy and osteomyelitis in a diabetic patient?
- A) MRI
- B) Bone culture
- C) Bone biopsy
- D) All of the above

19) What clinical manifestations are typical of a Charcot foot?
- A) Warm feet
- B) Bounding pedal pulses
- C) Absence of pedal pulses
- D) A and B

20) When Charcot osteoarthropathy involves the calcaneus of the foot, what type of calcaneal fracture is most commonly seen?
- A) Stress fracture
- B) Avulsion fracture
- C) None of the above
- D) All of the above

See answer sheet on page 129.

PM's CPME Program

Welcome to the innovative Continuing Education Program brought to you by *Podiatry Management Magazine*. Our journal has been approved as a sponsor of Continuing Medical Education by the Council on Podiatric Medical Education.

Now it's even easier and more convenient to enroll in PM's CE program!

You can now enroll at any time during the year and submit eligible exams at any time during your enrollment period.

PM enrollees are entitled to submit ten exams published during their consecutive, twelve–month enrollment period. Your enrollment period begins with the month payment is received. For example, if your payment is received on September 1, 2000, your enrollment is valid through August 31, 2001.

If you're not enrolled, you may also submit any exam(s) published in PM magazine within the past twelve months. **CME articles and examination questions from past issues of *Podiatry Management* can be found on the Internet at http://www.podiatrymgt.com/cme.** All lessons are approved for 1.5 hours of CE credit. Please read the testing, grading and payment instructions to decide which method of participation is best for you.

Please call (631) 563-1604 if you have any questions. A personal operator will be happy to assist you.

Each of the 10 lessons will count as 1.5 credits; thus a maximum of 15 CME credits may be earned during any 12-month period.

The* Podiatry Management Magazine *CPME program is approved by the Council on Podiatric Education in all states where credits in instructional media are accepted. This article is approved for 1.5 Continuing Education Hours (or 0.15 CEU's) for each examination successfully completed.

***PM's* CME program is valid in all states except Kentucky, Pennsylvania, and Texas.**

Enrollment/Testing Information and Answer Sheet

Note: If you are mailing your answer sheet, you must complete all info. on the front and back of this page and mail with your check to: ***Podiatry Management*, P.O. Box 490, East Islip, NY 11730.** Credit cards may be used only if you are faxing or phoning in your test answers.

TESTING, GRADING AND PAYMENT INSTRUCTIONS

(1) Each participant achieving a passing grade of 70% or higher on any examination will receive an official computer form stating the number of CE credits earned. This form should be safeguarded and may be used as documentation of credits earned.

(2) Participants receiving a failing grade on any exam will be notified and permitted to take one re-examination at no extra cost.

(3) All answers should be recorded on the answer form below. For each question, decide which choice is the best answer, and circle the letter representing your choice.

(4) Complete all other information on the front and back of this page.

(5) Choose one out of the 3 options for testgrading: mail-in, fax, or phone. To select the type of service that best suits your needs, please read the following section, "Test Grading Options".

TEST GRADING OPTIONS

Mail-In Grading

To receive your CME certificate, complete all information and mail with your check to:

Podiatry Management
P.O. Box 490, East Islip, NY 11730

There is **no charge** for the mail-in service if you have already enrolled in the annual exam CPME program, and we receive this exam during your current enrollment period. If you are not enrolled, please send $15.00 per exam, or $99 to cover all 10 exams (thus saving $51 over the cost of 10 individual exam fees).

Facsimile Grading

To receive your CPME certificate, complete all information and fax 24 hours a day to 1-631-563-1907. Your CPME certificate will be dated and mailed within 48 hours. This service is available for $2.50 per exam if you are currently enrolled in the annual 10-exam CPME program (and this exam falls within your enrollment period), and can be charged to your Visa, MasterCard, or American Express.

If you are *not* enrolled in the annual 10-exam CPME program, the fee is $17.50 per exam.

Phone-In Grading

You may also complete your exam by using the toll-free service. Call 1-800-232-4422 from 10 a.m. to 5 p.m. EST, Monday through Friday. Your CPME certificate will be dated the same day you call and mailed within 48 hours. There is a $2.50 charge for this service if you are currently enrolled in the annual 10-exam CPME program (and this exam falls within your enrollment period), and this fee can be charged to your Visa, Mastercard, or American Express. If you are not currently enrolled, the fee is $17.50 per exam. When you call, please have ready:

1. Program number (Month and Year)
2. The answers to the test
3. Your social security number
4. Credit card information

In the event you require additional CPME information, please contact PMS, Inc., at **1-631-563-1604**.

ENROLLMENT FORM & ANSWER SHEET

Please print clearly...Certificate will be issued from information below.

Name _____ Soc. Sec. #_____
Please Print: FIRST MI LAST

Address_____

City_____ State_____ Zip_____

Charge to: _____Visa _____ MasterCard _____ American Express

Card #_____ Exp. Date_____

Note: Credit card payment may be used for fax or phone-in grading only.

Signature_____ Soc. Sec.#_____ Daytime Phone_____

State License(s)_____ Is this a new address? Yes_____ No_____

Check one:

_____ I am currently enrolled. (If faxing or phoning in your answer form please note that $2.50 will be charged to your credit card.)

_____ I am not enrolled. Enclosed is a $15.00 check payable to Podiatry Management Magazine for each exam submitted. (plus $2.50 for each exam if submitting by fax or phone).

_____ I am not enrolled and I wish to enroll for 10 courses at $99.00 (thus saving me $51 over the cost of 10 individual exam fees). I understand there will be an additional fee of $2.50 for any exam I wish to submit via fax or phone.

Over, please

ENROLLMENT FORM & ANSWER SHEET (cont'd)

EXAM #1/02
Foot Deformities
(Haddad)

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
9.	A	B	C	D	19.	A	B	C	D
10.	A	B	C	D	20.	A	B	C	D

LESSON EVALUATION

Please indicate the date you completed this exam

_____

How much time did it take you to complete the lesson?

_____ hours _____minutes

How well did this lesson achieve its educational objectives?

_____Very well _____Well

_____Somewhat _____Not at all

What overall grade would you assign this lesson?

A B C D

Degree_____

Additional comments and suggestions for future exams:

_____

_____

_____

_____

_____

_____