CHAPTER **30**

VASOSPASTIC DISORDERS, ISCHEMIC DIGITS, AND THE USE OF EPINEPHRINE IN DIGITAL SURGERY

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INTRODUCTION

The postoperative course following digital surgery can be complicated by the presence of ischemic or vasospastic processes. When these conditions exist, postoperative ischemic changes may be further exacerbated with the use of epinephrine. It is our hope that this review will provide increased awareness of various vasospastic disorders and ischemic presentation, as well as an understanding of the role of epinephrine use in digital surgery. The ability to quickly recognize and treat potential postoperative complications is vital to patient safety. Therefore, it is imperative that the podiatric physician be well versed in the etiology, physiology, and treatments of these disorders.

RAYNAUD'S PHENOMENON

First described in 1862 by Maurice Raynaud, the Raynaud phenomenon (RP) represents an exaggerated vascular response to either cold temperature or emotional stress, compared with normal physiologic vascular response to these same factors. Normal vessel reactivity to these stimulants is extremely complex, but generally involves shunting of blood flow from extremities to preserve core body temperature and maintain heat. In part, regulation is monitored through reflexive sympathetic nervous system release of norepinephrine.[1] Vessel reactivity is further affected by vascular smooth muscle, which undergoes phasic activity and variable contractility, depending on the anatomic location and physiologic stimulants.[2] Similarly, nitric oxide and prostacyclin derived from endothelial cells play a function in normal vascular response, preventing both aggregation and adhesion of platelets. Endothelin-1 is a counter-regulatory endothelial derived vasoconstrictor, which affects vascular response in conjunction with prostacyclin.[3] The peripheral nervous system affects vasodilation in normal vasculature through release of substance P and vasoactive intestinal peptide (VIP), with counter-regulation through the release of somatostatin and neuropeptide Y providing vasoconstriction.[4] The normal physiologic responses are thought to impact RP through exaggeration of one or more of these described methods.

A sharply demarcated color change along digital skin is a common clinical description of RP, which arises due to an abnormal vasoconstriction of digital and cutaneous arterioles, as well as arteriovenous shunts, from either local defects or extrinsic factors. While most commonly reported to the upper extremity, lower extremity attacks are frequent and predicted to be underreported. Episodes are described by sudden onset of cold fingers or toes, beginning in a single digit and extending symmetrically, with skin changes including pallor or cyanosis. A localized defect of the vascular system represents the primary form of RP, with no evidence of further associated disorder or disease. This primary form may be referred to as Raynaud's disease, and is usually symmetric and bilateral when occurring. Primary RP is typically seen with onset at 15-30 years of age, more commonly in women, and with positive family history. Any gangrenous changes are limited to distal digit skin and normal nail fold capillaries are seen. Secondary disease forms include presence of RP with a related illness, such as systemic lupus erythematosus (SLE), scleroderma, or systemic sclerosis (SSc). Most commonly, attacks occur when shifting from warmer to cooler environments.

Secondary RP, which is more common in men, is seen with later age onset (>30 years), increased severity, asymmetry, and association with other diseases. Some patients exhibit other vascular symptoms including migraines or pulmonary hypertension. Again, triggers are noted to include cold room or high stress or anxiety levels. For either form of RP, the preferred diagnostic measure is cold water challenge with measurement of digital pressure, digital blood flow, and skin temperature. These measurements may aid in the initial ability to discern between primary RP, secondary RP, and normal individuals.[1]

The mechanism of pathogenesis in RP is hypothesized to be due to a localized vascular defect; however, the

specific pathophysiology is thought to differ between RP types. In primary RP, current evidence supports an increase in α-2 adrenergic response in digital and cutaneous vasculature, while the secondary form is influenced by environment, drugs, and disease. Secondary RP is mainly influenced by an underlying vascular disorder affecting reactivity of terminal vessels.[5] As an example, intimal fibrosis and endothelial dysfunction cause an increase in platelet adhesion early in SSc, which is thought to play a role in the increased incidence of RP in this population. It is important to remember that vascular reactivity is not always associated with endothelial function in these patients, making the described pathophysiologic mechanism only a hypothesis. The primary form of RP may be influenced by increased expression or sensitivity of α-2 receptors, increased efficiency of α-2 receptors, or decreased α-1 receptor activity. Subtypes of α-2 receptors exhibit different cold sensitivity profiles, and studies support the α-2C receptor as the primary receptor for thermoregulatory control.[6] Interestingly, a theoretical unifying explanation exists for temperature induced RP in primary and secondary forms. It is thought that the increased α-2 adrenergic response is correlated to increased protein tyrosine kinase activity, causing greater cooling in RP individuals when compared with control subjects.[7,8] At this time, there is only initial research into this hypothetical link. Separately being studied is the possibility of a genetic link. Preliminary genetic research has identified five loci (chromosome X, 6, 7, 9, 17) as possible disease links, but more research is needed to establish any definitive link.[9]

Treatment of RP begins with avoidance of sudden cold exposure and minimizing emotionally stressful situations. Whole body warmth is an important concept, rather than heavily covering only the extremities. The avoidance of tobacco and medications such as decongestants, amphetamines, or diet pills are also important due to their vasoconstrictive properties. The most common pharmacologic treatment includes use of calcium channel blockers, which have been shown to reduce frequency and severity of attacks.[10] Direct vasodilators such as nitroglycerin, hydralazine, nitroprusside, or topical nitric-oxide are also used as adjunctive treatment measures for RP. Unfortunately, sympatholytic agents such as prazosin or methydopa are unable to target the specific α-2C receptor and have been unsubstantiated in clinical trial effectiveness.[11] Anticoagulant and antithrombotic therapy is utilized if ulceration or thrombosis has occurred. Aspirin, heparin, and clopidogrel are most commonly utilized for this function. Many other medications have been studied in clinical trials including sildenafil, estrogen, prostaglandins, calcitonin, ACE-inhibitors, and angiotensinogen receptor blockers (ARBs), with mixed results reported.[12-15] It should be noted that no specific perioperative recommendations for management of RP have been published, other than standard conservative treatment such as maintaining body warmth.

OTHER VASOSPASTIC DISORDERS

Various other vasospastic disorders must be considered as differential diagnoses to RP. However, it must be remembered that these disorders may be independent, associated with RP, or associated with other vascular disorders. While less research is available, and many recommendations overlap with RP, each will briefly be described below.

Livedo reticularis is described as a mottling skin change, common on the arms and legs during a cold response, and may be seen with RP. This condition is benign and reversible with skin re-warming. Livedo reticularis is also associated with vasculitis and occlusive vascular disease.[16] It is differentiated from RP by the lack of a sharply demarcated skin change and lack of delay in vascular flow. No further treatment is recommended for this condition, however if desired, the patient may be referred to a dermatologist for cosmetic treatment options.

Acrocyanosis is a persistent, painless, symmetric cyanosis of the extremities secondary to vasospasm of arterioles and capillaries in cold environments. Persistent cyanosis with concomitant edema, difficult reversal of discoloration, presence of extremity sweating or diaphoresis, and lack of ulceration or trophic changes differentiate this disease from RP. It is also not generally associated with any occlusive vascular disease. Treatment is usually unnecessary and ineffective in this disorder.[17]

Pernio is defined as a chronic skin inflammatory disease after cold exposure which may be pruritic and painful with erythematous lesions. The pathophysiology is due to an abnormal vascular response to cold or humid environments secondary to microvascular damage decreasing the ability of normal phasic vascular responses. These cutaneous lesions slowly resolve over 2 to 3 weeks, but may form ulcerations on the extremity affected. Treatments include ultraviolet light, Calcium channel blockers, and cold avoidance similar to that of RP.[18]

Additional differentials may include arteriosclerosis, thromboangiitis obliterans, drug-induced vasospasm, cryoglobulinemia, vascular injury, or frostbite. Each of these differential diagnoses involves external stimuli, which should be elicited in routine history taking, physical

examination, and laboratory testing. Cholesterol emboli or the "blue toe" must also be included in a differential diagnosis but will be discussed in detail in a following section.

ISCHEMIC DIGIT

Postoperative ischemia can result from vasospasm secondary to mechanical manipulation during surgery or to a disruption in flow via emboli, laceration or thrombus.[19] Clinical evaluation of an ischemic limb or digit will characteristically exhibit pain, pulselessness, pallor, paresthesia, and paralysis. The digit will initially present cool and pale with a delay in subpapillary venous plexus refill time. However, the digit may appear cyanotic and eventually gangrenous the longer it remains untreated.

Additionally, signs of numbness or paresthesia may present due to early nerve dysfunction.[20] There may also be a gradual increase in the level of pain experienced, and without treatment, a progressive decrease in pain as sensory loss advances. However, postoperative evaluation of pain and quality of paresthesia may not provide an accurate representation, due to the common usage of local anesthesia before or after the procedure.

CHOLESTEROL EMOBLI

Cholesterol emboli, or "blue toe syndrome," are due to embolic occlusions of distal arteries from proximal arterial sources and are normally sudden in nature. Digits present as cool, painful, and cyanotic in the presence of a strong pulse and warm foot. The lateral and plantar aspects of the foot are frequently involved with scattered areas of petechiae noted.[21] Calf pain and muscle tenderness, along with fever, elevated eosinophils and erythrocyte sedimentation rate can be seen as a reaction to inflammation to the proposed emboli.[21] Recognition of an atherothrombotic emboli requires diagnostic evaluation to identify the source, as an unstable atherosclerotic lesion may be present elsewhere.[20]

VENOUS CONGESTION

Venous congestion occurs when there is overfilling and distention of the veins with blood as a result of mechanical obstruction. This can commonly occur in digital surgery with constrictive bandaging or when adequate elevation is not achieved postoperatively. A digit with venous congestion will present cyanotic, but will be warm to touch and subpapillary venous plexus refill time will be within normal limits. This represents the clinical differentiation between the venous congested digit and the ischemic digit. Simply loosening the bandage and elevating the foot allows the digit to return to normal color. However, in an ischemic digit, this will provide no benefit and will actually worsen the ischemic process.

DIAGNOSTIC EXAMINATION

In the presence of an ischemic digit, non-invasive vascular studies (NIVS) should be immediately performed. NIVS, including ankle-brachial index (ABI), and pulse volume recordings (PVR) are inexpensive and non-invasive tests to measure systolic blood pressures to determine the level and extent of vascular obstruction. Toe pressures of less than 60 percent of the ankle pressure indicate digital artery occlusive disease. On PVR evaluation, a normal waveform will present as a systolic upstroke followed by a downstroke with a dicrotic notch, as would be seen with venous congestion. With an ischemic digit, the waveform will present as a delayed upstroke or flat systolic peak with diminished amplitude on PVR. In addition to ABI and PVR, waveforms and digital pressures by way of duplex doppler ultrasonography (DDU) can also be evaluated to assess if there is adequate perfusion.[22] Abnormalities in vessel walls would show a disturbance in the normal laminar flow on ultrasonography and can be associated with stenosis.

Arteriography provides the most information in an acute setting, in that it provides detailed anatomy and in the presence of occlusive disease, and will allow the physician to distinguish between thrombosis and embolism.[20] After NIVS, arteriography should be considered on the second day of treatment, if no improvement has been observed. This test however, is invasive, and in a severely limb threatening presentation, immediate surgical revascularization may be the preferred option.

TREATMENT

As a general guideline for treatment of an ischemic process, the limb should be placed in dependency, bandages loosened, and adjustment or removal of a pin should be performed immediately. The use of an extremity warming device, such as a Bair hugger, should also be employed at the onset of this process. One should then consider pharmacologic therapy, first utilizing Nifedipine 10 to 60 mg with Aspirin 81 mg daily. If no improvement is noted within a short time span, proceed to temporary chemical sympathectomy by local infiltration of plain lidocaine. Combine the use of transdermal nitroglycerin if no improvement is noted post-injection

and finally short-term heparin drip may be utilized for 24 to 72 hours. The Podiatry Institute faculty employs the above mentioned treatments simultaneously, and this protocol has been recognized as an acceptable treatment by vascular surgery specialists. A number of agents can also be used through intraarterial catheter-directed thrombolysis.[23] Short term dosing of anticoagulants should also be prescribed for the patient upon discharge. A vascular surgery consult should also be employed at the onset of any ischemic process.

PHYSIOLOGY OF EPINEPHRINE

Epinephrine, also referred to as adrenaline, is a hormone and neurotransmitter. It is a sympathomimetic monoamine derived from the amino acids phenylalanine and tyrosine. The Latin roots ad-+renes and the Greek roots epi-+nephros both literally mean "on the kidney," referring to the adrenal gland, which sits atop the kidneys and secretes epinephrine. Epinephrine was first isolated and identified in 1895 by Napoleon Cybulski, a Polish physiologist, but it wasn't until 1904 that epinephrine was artificially synthesized by Friedrich Stolz.[24]

Epinephrine is a "fight or flight" hormone, and plays a central role in the short-term stress reaction. It is released from the adrenal glands when danger threatens or in an emergency, most commonly referred to as an "adrenaline rush." Such triggers may be threatening, exciting, or environmental stressor conditions such as high noise levels, bright light, and high ambient temperature. It increases heart rate and stroke volume, dilates the pupils, and constricts arterioles in the skin and gastrointestinal tract while dilating arterioles in skeletal muscles. It elevates the blood sugar level by increasing catabolism of glycogen to glucose in the liver, and at the same time begins the breakdown of lipids in fat cells. Like some other stress hormones, epinephrine has a suppressive effect on the immune system.[25]

Epinephrine's actions are mediated through adrenergic receptors. Epinephrine is a non-selective agonist of all adrenergic receptors. It activates α1, α2, α1, and α2 receptors to different extents. It binds to α1 receptors of liver cells, which activate inositol-phospholipid signaling pathway, signaling the phosphorylation of glycogen synthase and phosphorylase kinase. It inactivates and activates each respectively, leading to the activation of glycogen phosphorylase, which catalizes breakdown of glycogen so as to release glucose to the bloodstream. Simultaneously, protein phosphatase-1 (PP1) is inactivated, stopping reversal of all previous phosphorylations. Epinephrine also activates α-adrenergic receptors of the liver and muscle cells, thereby activating the adenylate cyclase signaling pathway, which will in turn increase glycogenolysis. α-2 receptors are found primarily in skeletal muscle blood vessels where they trigger vasodilation. However, α-adrenergic receptors are found in most smooth muscles and splanchnic vessels, and epinephrine triggers vasoconstriction in those vessels. It is this action on α-adrenergic receptors that we take advantage of with the addition of epinephrine to local anesthesia to achieve surgical hemostasis.[25]

LOCAL ANESTHESIA AND EPINEPHRINE

Local anesthetics produce anesthesia by inhibiting excitation of nerve endings or by blocking conduction in peripheral nerves. This is achieved by anesthetics reversibly binding to, and inactivating sodium channels. Sodium influx through these channels is necessary for the depolarization of nerve cell membranes and subsequent propagation of impulses along the course of the nerve. When a nerve loses depolarization and capacity to propagate an impulse, the individual loses sensation in the area supplied by the nerve. Local anesthetics exist in ionized and non-ionized forms, the proportions of which vary with the pH of the environment. The non-ionized portion is the form that is capable of diffusing across nerve membranes and blocking sodium channels. Anesthetics with presence of greater non-ionized portions have a faster onset of action.

Local anesthetics differ in respect to the pH at which the ionized and non-ionized forms are present at equilibrium but this pH is generally in the range of 7.6-8.9. The more closely the equilibrium pH for a given anesthetic approximates the physiologic pH of tissues, 7.35-7.45, the more rapid the onset of action. A decrease in pH shifts equilibrium toward the ionized form, delaying onset of action. This explains why local anesthetics are slower in onset of action and less effective in the presence of inflammation, which creates a more acidic environment with lower pH. Contrastingly, the addition of sodium bicarbonate is used clinically to increase the pH of local anesthetic solutions thereby enhancing onset of action. All local anesthetics, with the exception of cocaine, are vasodilators. Vasodilatation occurs via direct relaxation of peripheral arteriolar smooth muscle fibers. Greater vasodilator activity of a local anesthetic leads to faster absorption and, thus, shorter duration of action. To counteract this vasodilatation, epinephrine is often included in local anesthetic solutions.

Addition of epinephrine to the local anesthetic solution may improve safety and allow administration of lower doses of local anesthetic. Since local anesthetics are vasodilators,

they tend to be absorbed into the bloodstream from the operative field because of vasodilatation of peripheral arterioles. Epinephrine induces vasoconstriction, delaying absorption of the local anesthetic for longer duration of action at the site of injection. By delaying absorption, epinephrine also increases the safe dose of local anesthetic that may be administered. Addition of epinephrine also improves hemostasis of the operative field, which may decrease duration of the operation and thus obviate the need for prolonged local anesthetic effect. This can help avoid the need for subsequent injection if local anesthetic begins to wear off as surgery proceeds.[26]

Epinephrine has been demonstrated to cause effective vasoconstriction at concentrations as small as 1:1,000,000, although most surgeons use a concentration of 1:100,000 to 1:400,000, which is available in commercial preparations. Lower concentrations of epinephrine take longer to achieve maximal effect. Thus, when using dilute concentrations of epinephrine, the surgeon should wait longer between injection and incision than when using solutions containing higher concentrations of epinephrine.[26] When lidocaine is used alone, it produces 1 to 2 hours of vasodilatation, and marcaine alone produces 3-5 hours of vasodilatation. However, addition of epinephrine causes vasoconstriction for 1 hour with maximum hemostasis achieved at 5 minutes.[27]

In addition to anatomic dissection, the use of epinephrine has long been used as the sole method of hemostasis in foot surgery. Ankle, calf, and thigh tourniquets have also been employed, but there are distinct disadvantages to their use when compared with epinephrine. Tourniquets cause immediate discomfort at the site of inflation, which necessitates the need for general or spinal anesthesia if used longer than 45 minutes. Tourniquets also completely disrupt the blood supply to tissues distal to it, often causing muscle damage as demonstrated by the increase in serum myoglobulin after use. It should be noted that the use of epinephrine only disrupts about 80% of blood supply to infiltrated tissues. Tourniquet use also has the potential for nerve damage and increasing the risk for development of venous thromboembolism.[27]

Epinephrine obviously should be used with caution in patients with known vasospastic disorders. However its use in digital surgery has been widely debated. Podiatry Institute faculty routinely employ epinephrine in digital surgery, and with close monitoring and judicious patient selection, complications have been minimal at best. With that said, there are many studies that dispel the myth inhibiting the use of epinephrine in digital surgery. Krunic et al found that a literature review failed to provide evidence to support the dogma that the use of epinephrine with local anesthesia caused digital necrosis. They actually concluded that local with epinephrine provided better and longer pain control during digital procedures.[28] Radovic et al also reviewed over 150 cases and found that patients receiving local anesthetics containing epinephrine revealed no complications in the foot and ankle.[29]

There are several studies that support the use of epinephrine in digital surgery, especially when there is trauma involved and there is an obvious need to obtain hemostasis. The discouragement of its use is an old myth, and many current authors and scholars would agree that in the absence of underlying vascular compromise, epinephrine, 1:200,000 to 1:100,000, is safe to use in digital blocks along with local anesthetics.

CONCLUSION

Ischemia and venous congestion are possible postoperative complications following digital surgery. Vasospastic disorders have been reviewed in detail in order to provide a basis for preoperative evaluation and management, as well as their role in increasing the risk of these postoperative complications. Additionally, ischemic digits and venous congestion have been reviewed as postoperative complications, with current diagnostic and treatment protocols discussed. The use of epinephrine has also been implicated as a causative factor for postoperative ischemia, however, studies have failed to produce adequate evidence that this correlation exists. Thorough history and physical examination and proper patient selection greatly reduce the incidence of postoperative ischemia. Additionally, timely recognition and treatment of ischemic complications with the above treatment protocol provide enhanced postoperative prognosis.

REFERENCES

1. Maricq HR, Jennings JR, Valter I, et al. Evaluation of treatment efficacy of Raynaud phenomenon by digital blood pressure response to cooling. Raynaud's Treatment Study Investigators. *Vasc Med* 2000;5:135.
2. Maricq HR, Weinrich MC, Valter I, et al. Digital vascular responses to cooling in subjects with cold sensitivity, primary Raynaud's phenomenon, or scleroderma spectrum disorders. *J Rheumatol* 1996;23:2068.
3. LeRoy EC, Medsger TA Jr. Raynaud's phenomenon: a proposal for classification. *Clin Exp Rheumatol* 1992;10:485.
4. Flavahan NA, Vanhoutte PM. Effect of cooling on alpha-1 and alpha-2 adrenergic responses in canine saphenous and femoral veins. *J Pharmacol Exp Ther* 1986;238:139.
5. Spencer-Green G, Morgan GJ, Brown L, FitzGerald O. Hypothenar hammer syndrome: an occupational cause of Raynaud's phenomenon. *J Rheumatol* 1987;14:1048.

6. Carpentier PH, Maricq HR. Microvasculature in systemic sclerosis. *Rheum Dis Clin North Am* 1990;16:75.
7. Cutolo M, Grassi W, Cerinic MM. Raynaud's phenomenon and the role of capillaroscopy. *Arthritis Rheum* 2003;48:3023.
8. Anderson ME, Allen PD, Moore T, et al. Computerized nailfold video capillaroscopy—a new tool for assessment of Raynaud's phenomenon. *J Rheumatol* 2005;32:841.
9. Susol E, MacGregor AJ, Barrett JH, et al. A two-stage, genome-wide screen for susceptibility loci in primary Raynaud's phenomenon. *Arthritis Rheum* 2000;43:1641.
10. Thompson AE, Shea B, Welch V, et al. Calcium-channel blockers for Raynaud's phenomenon in systemic sclerosis. *Arthritis Rheum* 2001; 44:1841.
11. Russell LI, Lessard,JA. Prazosin treatment of Raynaud's phenomenon: A double-blind single crossover study. *J Rheumatol* 1985;12:94.
12. McFadyen IJ, Housley E, MacPherson AI. Intraarterial reserpine administration in Raynaud syndrome. *Arch Intern Med* 1973;132:526.
13. Langevitz P, Buskila D, Lee P, et al. Treatment of refractory ischemic skin ulcers in patients with Raynaud's phenomenon with PGE, infusions. *J Rheumatol* 1989;16:1433.
14. Mohrland JS, Porter JM, Smith EA, et al. A multiclinic, placebo-controlled, double-blind study of Prostaglandin Raynaud's syndrome. *Ann Rheum Dis* 1985;44:754.
15. Clifford PC, Martin MF, Sheddon EJ, et al. Treatment of vasospastic disease with prostaglandin E1. *Br Med J* 1980;281:1031.
16. Champion RH. Livedo Reticularis: A review. *Br J Derm* 2006;77:167.
17. Monticone G, Colonna L, Palermi G, et al. Quantitative nailfold capillary microscopy findings in patients with acrocyanosis compared with patients having systemic sclerosis and control subjects. *J Am Acad Dermatol* 2000;42:787-90.
18. Su WP, Perniciaro C, Rogers RS, et al. Chilblain lupus erythematosus (Lupus pernio) : clinical review of the Mayo clinic experience and proposal of diagnostic criteria. *Cutis* 1994;54:395-9.
19. Malay DS. In: The P.I. Manual. 1999. Podiatry Institute Publishing, Decatur, GA.
20. Mitchell ME, Mohler ER, Carpenter JP. Acute arterial occlusion of the lower extremities (acute limb ischemia). UpToDate Online 16.3.
21. Loscalzo J, Creager MA, Dzau VJ. Arterial obstructive diseases of the extremities. In: Vascular Medicine, 1st ed. Boston; 1992. p. 857-8.
22. Olin JW, Kaufman JA, Bluemke DA, et al. Atherosclerotic vascular disease converence: Writing Group IV: Imaging. Circulation 2004;109;2626.
23. Results of a prospective randomized trail evaluating surgery versus thrombolysis for ischemia of the lower extremity. The STILE trial. *Ann Surg* 1994;220:251-66.
24. Boron WF, Boulpaep EL. Medical physiology: a cellular and molecular approach. Philadelphia: Elsevier/Saunders; 2005
25. Voet D. Biochemistry, 3rd ed. 2004
26. McLeod IK, Gallagher DJ, Seagle MB. Local aAnesthetics. URL: http://emedicine.medscape.com/article/873879-overview.
27. Noss JS, Johnson RWS, Merrill T, et al. Hemostasis. In: McGlamrys Comprehensive Textbook of Foot and Ankle Surgery. 3rd ed. Philadelphia. Lippincott Williams and Wilkins; 2001 p. 2065-74
28. Krunic A, Wang L, Soltani S. Digital Anesthesia with Epinephrine: An old myth Revisited. *J Am Acad Derm* 2004;51:755-9.
29. Radovic P, Smith RG, Shumway. Revisiting epinephrine in foot surgery. *J Am Podiatry Med Assoc* 2003;93:157-60.