Mechanisms of Vascular Disease:

A Reference Book for Vascular Specialists

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Edited by Robert Fitridge and Matthew Thompson

Completely Updated Edition 2011

Barr Smith Press

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BARR SMITH PRESS
An imprint of
The University of Adelaide Press

Published in Adelaide by

The University of Adelaide, Barr Smith Press
Barr Smith Library
The University of Adelaide
South Australia 5005
press@adelaide.edu.au
www.adelaide.edu.au/press

The University of Adelaide Press publishes peer-reviewed scholarly works by staff via Open Access online editions and print editions.

The Barr Smith Press is an imprint of the University of Adelaide Press, reserved for scholarly works which are not available in Open Access, as well as titles of interest to the University and its associates. The Barr Smith Press logo features a woodcut of the original Barr Smith Library entrance.

This CIP cataloguing for this work is as follows;

Mechanisms of vascular disease : a reference book for vascular surgeons / Robert Fitridge, Matthew Thompson, [editors].

1. Blood vessels, Diseases.
2. Blood vessels, Surgery.

I. Fitridge, Robert
II. Thompson, M. M.

For the full Cataloguing-in-Publication data please contact National Library of Australia:
cip@nla.gov.au

ISBN (paperback) 978-0-9871718-2-5

Book design: Midland Typesetters
Cover design: Emma Spoehr, based on a diagram by Dave Heinrich of the Medical Illustration and Media Unit, Flinders Medical Centre
Paperback edition printed by Griffin Press, South Australia

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Acknowledgements

The Editors gratefully acknowledge the outstanding contributions of each Author involved in this reference book. We would also like to acknowledge the invaluable efforts of Ms Sheona Page who has worked tirelessly on this project. We would also like to thank Prue Cowled PhD and Ms Cayley Wright for their assistance.

Abbreviation List

a1-PI	a1-protease inhibitor
5-HT	5-Hydroxytryptamine/Serotonin
AAA	Abdominal aortic aneurysm
AAS	Acute aortic syndrome
AAV	Adeno-associated viruses
ACE	Angiotensin converting enzyme
ACS	Acute coronary syndrome
ACS	Abdominal compartment syndrome
ACTH	Adrenocorticotropic hormone
ADAMTS	A disintegrin and metalloproteinase with thrombospondin motifs
ADP	Adenosine diphosphate
AIDS	Acquired immune deficiency syndrome
ALI	Acute lung injury
AMP	Adenosine monophosphate
AMPA	α-amino-3 hydroxy-5-methylisoxazole
ANA	Anti-nuclear antibody
ANCA	Anti-neutrophil cytoplasmic antibody
AOD	Aortic occlusive disease
AP1	Activated protein 1
APC	Activated protein C
APC	Antigen presenting cell
APLAS	Antiphospholipid antibody syndrome
ApoAI	Apolipoprotein AI
ApoE	Apolipoprotein E
APS	Antiphospholipid antibody syndrome
APTT	Activated partial thromboplastin time

ARDS	Acute respiratory distress syndrome
AT	Antithrombin
ATP	Adenosine triphosphate
AVP	Ambulatory venous thrombosis
β2-GPI	β2-glycoprotein Ib
bFGF	Basic fibroblast growth factor
BKCa	Large conductance calcium activated potassium channel
BMPs	Bone morphogenetic proteins
BMS	Bare metal stent
CAD	Coronary artery disease
CaM	Calmodulin
CAM	Cell adhesion molecule
cAMP	Cyclic adenosine monophosphate
CCK	Cholecystokinin
cGMP	Cyclic guanine monophosphate
CD	Cluster of differentiation
CD40L	Cluster of differentiation 40 ligand
CEA	Carotid endarterectomy
CETP	Cholesteryl ester transfer protein
CFD	Computational fluid dynamics
CG	Cationized gelatin
CGRP	Calcitonic gene regulated peptide
CHD	Coronary heart disease
CI	Confidence interval
CIMT	Carotid intimal-media thickness
c-JNK	c-Jun N-terminal kinase
CK-MB	Creatinine kinase (Myocardial specific)
CNCP	Chronic noncancer pain
cNOS	Constitutive nitric oxygen synthase enzyme
COX-1	Cyclooxygenase-1
COX-2	Cyclooxygenase-2
CROW	Charcot restraint orthotic walker
CRRT	Continuous renal replacement therapy

CRP	C-reactive protein
CRPS	Complex regional pain syndromes
CT	Computational tomography
CTA	Computed tomographic angiography
CTD	Connective tissue disorders
CTGF	Connective tissue growth factor
CYP	Cytochrome P450
CVD	Cardiovascular disease
CVI	Chronic venous insufficiency
DAG	Diacylglycerol
DES	Drug-eluting stent
DRG	Dorsal root ganglion
DNA	Deoxyribonucleic acid
DSA	Digital subtraction arteriography
DTS	Dense tubular system
DVT	Deep vein thrombosis
EC	Endothelial cell
ECM	Extracellular matrix
EDCF	Endothelium-derived contracting factor
EDH	Endothelium-dependent hyperpolarisation
EDS	Ehlers-Danlos syndrome
EET	Epoxyeicosatrienoic acids
ELAM-1	Endothelial-leukocyte adhesion molecule-1
ELG	Endoluminal grafts
ELISA	Enzyme linked immunosorbent assay
E_K	Equilibrium potential
E_M	Membrane potential
eNOS	Endothelial nitric oxide synthase enzyme
EPC	Endothelial progenitor cells
EPCR	Endothelial protein C receptor
ePTFE	Expanded polytetrafluoroethylene
ERK	Extracellular signal-regulated kinase
ESR	Erythrocyte sedimentation rate

ET	Essential thrombocytosis
ET-1	Endothelin 1
EVAR	Endovascular aortic aneurysm repair
EVLA	Endovenous LASER ablation
FDA	Food and drug administration
FDPs	Fibrin degradation products (soluble)
FGF	Fibroblast growth factor
FGF-2	Fibroblast growth factor 2
FMN	Flavin mononucleotide
FVL	Factor V Leiden
GABA	Gamma-aminobutyric acid
GABA B	Gamma-aminobutyric acid subtype B
G-CSF	Granulocyte colony stimulating factor
GMCSF	Granulocyte-macrophage colony stimulating factor
GP	Glycoprotein
GPCR	G-protein coupled receptor
GSV	Great saphenous vein
HDL	High density lipoprotein
HDL-C	High density lipoprotein cholesterol
HIF	Hypoxia inducible factor
HIT	Heparin induced thrombocytopenia
HIV	Human immunodeficiency virus
HLA	Human leukocyte antigen
HMG Co-A	Hydroxymethylglutaryl coenzyme-A
HMW	High molecular weight
HPETE	Hydroperoxyeicosatetraenoic acid
HETE	Hydroxyeicosatetraenoic acids
HR	Hazard ratio
hsCRP	High-sensitive C-reactive protein
HSP	Heat shock protein
HUV	Human umbilical vein
IAH	Intra-abdominal hypertension

IAP	Intra-abdominal pressure
IAPP	Intra-abdominal perfusion pressure
ICAM-1	Inter-cellular adhesion molecule-1
ICAM-2	Inter-cellular adhesion molecule-2
ICP	Intra-compartmental pressure
ICU	Intensive care unit
IFN	Interferon
IGF-1	Insulin-like growth factor-1
IHD	Ischemic heart disease
IL	Interleukin
IL-1	Interleukin-1
IL-1α	Interleukin-1 alpha
IL1-β	Interleukin-1 beta
IL-6	Interleukin-6
IL-8	Interleukin-8
ILT	Intraluminal thrombus
IKCa	Intermediate conductance calcium-activated potassium channels
IMH	Intramural haematoma
IMP	Inosine monophosphate
iNOS	Inducible nitric oxide synthase enzyme
IP(3)	1,4,5-inositol triphosphate
IRI	Ischemia reperfusion injury
IVIG	Intravenous pooled immunoglobulin
IVUS	Intravascular ultrasound
KGF	Keratinocyte growth factor
KGF-2	Keratinocyte growth factor-2
LAP	Latency associated peptide
LCS	Limb compartment syndrome
LDL	Low density lipoprotein
LDS	Loeys-Dietz syndrome
LLC	Large latent complex
LEC	Lymphatic endothelial cells

LFA-1	Lymphocyte function-associated antigen-1
LO	Lipoxygenase
LOX	Lysyl oxidase
LOPS	Loss of protective sensation
LPA	Lysophosphatidic acid
LPS	Lipopolysaccharide
LTA	Lipoteichoic acid
LTGFBP	Latent TGF binding protein
MAC-1	Macrophage-1 antigen
MAPK	Mitogen activated protein kinase
MCP-1	Monocyte chemoattractant protein-1
M-CSF	Macrophage-colony stimulating factor
MFS	Marfan syndrome
MHC	Major histocompatibility
MI	Myocardial infarction
MIP-1	Macrophage inflammatory protein-1
MLC_{20}	Myosin light $chain_{20}$
MLCK	Myosin light chain kinase
MLCP	Myosin light chain phosphatase
MMP	Matrix metalloproteinase
MODS	Multiple organ dysfunction syndrome
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
mRNA	Messenger RNA
MRSA	Methicillin resistant *Staphylococcus aureus*
MRSE	Methicillin resistant *Staphylococcus epidermidis*
MRTA	Magnetic resonance tomographic angiography
MTHFR	Methylenetetrahydrofolate reductase
MT-MMP	Membrane-type MMP
MVPS	Mitral valve prolapse syndrome
NADPH	Nicotinamide adenine dinucleotide phosphate
NGF	Nerve growth factor

NFκB	Nuclear factor kappa B
NiTi	Nitinol
NJP	Non-junctional perforators
NMDA	N-methyl-D-aspartate
NNH	Number needed to harm
NNT	Number needed to treat
NO	Nitric oxide
NOS	Nitric oxide synthase enzyme
NSAID	Non-steroidal anti-inflammatory drug
NV	Neovascularisation
OCP	Oestrogen/progesterone contraceptive pill
OPN	Osteopontin
OPG	Osteoprotegerin
OR	Odds ratio
OxLDL	Oxidised low density lipoprotein
PAD	Peripheral arterial disease
PAF	Platelet activating factor
PAI	Plasminogen activator inhibitor
PAI-1	Plasminogen activator inhibitor-1
PAR	Protease activated receptor
PAR-1	Protease activated receptor-1
PAR-4	Protease activated receptor-4
PAU	Penetrating aortic ulcer
PC	Protein C
PCA	Poly (carbonate-urea) urethane
PCI	Percutaneous coronary intervention (angioplasty)
PCWP	Pulmonary capillary wedge pressure
PDGF	Platelet-derived growth factor
PDGFβ	Platelet-derived growth factor-β
PDS	Polydioxanone
PECAM-1	Platelet-endothelial cell adhesion molecule-1
PEDF	Pigment epithelium-derived factor
PES	Paclitaxel-eluting stent

PET	Positron emission tomography
PF4	Platelet factor 4
PGI_2	Prostacyclin
PGG_2	Prostaglandin G_2
PGH_2	Prostaglandin H_2
$PGEI_2/PGI_2$	Prostaglandin I_2
PGN	Peptidoglycan
PHN	Postherpetic neuropathy
PHZ	Para-anastomotic hyper-compliant zone
PI3K	Phosphatidylinositol 3-kinase
PIP2	Phosphatidylinositol 4,5-bisphosphate
PLC	Phospholipase C
PLOD	Procollagen lysyl hydroxylase
PMCA	Plasma membrane Ca^{2+} APTases
PMN	Polymorphonuclear leukocyte
POSS	Polyhedral oligomeric silsesquioxanes
PPAR	Peroxisomal proliferation activating receptor
PPI	Proton pump inhibitor
PRV	Polycythaemia rubra vera
PS	Protein S
PSGL-1	P-selectin glycoprotein ligand-1
PT	Prothombin time
PTCA	Percutaneous coronary angioplasty
PTFE	Polytetrafluoroethylene
PTS	Post-thrombotic syndrome
PUFA	Polyunsaturated fatty acid
PVI	Primary valvular incompetence
rAAA	Ruptured AAA
Rac	Ras activated cell adhesion molecule
RANTES	Regulated upon activation, normal T cell expressed and secreted
RAS	Renin angiotensin system
RCT	Randomised controlled trial

RF	Rheumatoid factor
RFA	Radiofrequency ablation
rhAPC	Recombinant human activated protein C
RNA	Ribonucleic acid
ROS	Reactive oxygen species
RR	Relative risk
RSD	Reflex sympathetic dystrophy
S1P	Sphingosine-1-phosphate
SAPK	Stress-activated protein kinase
SCF	Stem cell factor
SCS	Spinal cord stimulation
ScvO2	Superior vena cava venous oxygen saturation
SDF-1	Stromal-cell-derived factor-1
SERCA	Sarco/endoplasmic reticulum CaATPases
SEP	Serum elastin peptides
SES	Sirolimus-eluting stent
SEPS	Subfascial endoscopic perforator surgery
SFA	Superficial femoral artery
SFJ	Sapheno-femoral junction
SIRS	Systemic inflammatory response syndrome
SKCa	Small conductance calcium-activated potassium channels
SLE	Systemic lupus erythematosus
SMA	Smooth muscle alpha actin
SMC	Smooth muscle cell
SMP	Sympathetically maintained pain
SNARE	Soluble N-ethylmaleimide-sensitive factor activating protein receptors
SNP	Single nucleotide polymorphisms
SNRI	Serotonin/Noradrenaline reuptake inhibitors
SPJ	Sapheno-popliteal junction
SPP	Skin perfusion pressure
SR	Sarcoplasmic reticulum
SSRIs	Selective serotonin re-uptake inhibitors
SSV	Small saphenous vein

SVT	Superficial thrombophlebitis
STIM1	Stromal interacting molecule 1
TαCE	TNFα converting enzyme
TAAD	Thoracic aortic aneurysm disease
TAD	Thoracic aortic dissection
TAFI	Thrombin-activatable fibrinolysis inhibitor
Tc-99 MDP	Technetium-99 methylene diphosphonate
TCA	Tricyclic antidepressant
TCC	Total contact cast
TCR	T-cell receptor
TENS	Transcutaneous electrical nerve stimulation
TF	Tissue factor
TFPI	Tissue factor pathway inhibitor
TGF	Transforming growth factor
TGF-α	Transforming growth factor-alpha
TGF-β	Transforming growth factor-beta
TGL	Triglycerides
Th	T helper
TIA	Transient ischemic attack
TIMP	Tissue inhibitors of metalloproteinase
TLR	Toll-like receptors
TNF	Tumour necrosis factor
TNF-α	Tumour necrosis factor-alpha
tPA	Tissue-type plasminogen activator
TRP	Transient receptor potential
TRPC	Transmembrane receptor potential canonical
TRPV1	Transmembrane receptor potential Vanilloid-type
TXA2	Thromboxane A2
uPA	Urokinase
UT	University of Texas
VCAM	Vascular cell adhesion molecule
VCAM-1	Vascular cell adhesion molecule-1
VEGF	Vascular endothelial growth factor

VEGF-R	Vascular endothelial growth factor receptor
VIP	Vasoactive intestinal peptide
VLA-1	Very late activating antigen-1
VOCC	Voltage operated calcium channels
VPT	Vibratory perception threshold
VSMC	Vascular smooth muscle cells
VTE	Venous thromboembolism
VV	Varicose veins
vWF	von Willebrand factor
XO	Xanthine oxidase

26 • Pathophysiology and Principles of Management of the Diabetic Foot

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INTRODUCTION

The incidence of diabetes continues to grow at a staggering pace. The United States' Centers for Disease Control and Prevention estimate that 23.6 million people or 7.8% of the U.S. population has diabetes, with 1.6 million new cases being diagnosed each year.[1,2] These figures are even more astonishing when one considers worldwide estimates. Close to 4 million deaths in the 20–79 year old age group may be attributed to diabetes in 2010, accounting for 6.8% of global all-cause mortality in this age group.[3] The number of people with diabetes worldwide is expected to reach 366 million people by 2030, more than double the estimated 177 million people affected with the disease in 2000.[2] The increased disease prevalence is accompanied by an increase in associated comorbidities. The literature estimates that patients with diabetes have nearly a 25% lifetime risk of developing a foot ulcer with more than 50% of these ulcers becoming infected and requiring hospitalization.[4] In fact, at least 20% of all diabetes-related hospital admissions are due to diabetic foot ulcers. Associated with foot ulcers and infection is the incidence of amputation. It has been conservatively reported that, worldwide, a major amputation takes place every 30 seconds with over 2500 limbs lost per day.[5] At least 60% of all non-traumatic lower extremity amputations are related to complications of diabetes.[1] People with diabetes who have had one amputation have a 68% risk of having another in the next 5 years and have a 50% mortality rate in the 5 years following the initial amputation.[6] It is estimated that up to 85% of diabetic foot-related amputations could be prevented through prompt intervention and with centers directed at educating individuals about proper foot care.

The economic impacts on the patient, national healthcare system and economy also impose a great burden. Healthcare expenditures on diabetes are expected to account for 11.6% of the total healthcare

expenditure in the world in 2010.[3] The estimated global costs to treat and prevent diabetes and its complications are expected to total at least 376 billion USD in 2010, with some projections exceeding 490 billion USD. Between 22 and 27% of the total diabetes costs are attributable to lower extremity disease.[3,7]

PATHOPHYSIOLOGY OF THE DIABETIC FOOT

Neuropathy

Neuropathy, or the loss of protective sensation (LOPS) of the lower extremity, is the predominant etiology for diabetic foot ulceration. The absence of this most basic nociceptive mechanism results in the patient's inability to perceive local foot trauma both from intrinsic factors such as abnormal or faulty foot mechanics or deformity, as well as extrinsic factors such as foreign objects or improper footwear. These factors place the foot at increased risk for developing an ulcer, which can lead to amputation.

Neuropathy can be subdivided into sensory, motor, and autonomic categories.

Sensory neuropathy typically begins distally, moving proximally, and is symmetrical. It is often described as a 'stocking-glove' distribution.

Motor neuropathy results in intrinsic muscle wasting, causing a progressive deformity such as hammertoe, claw toe, and plantar flexion deformities of the metatarsals. These deformities can cause an increase in focal pressure at the areas of the interphalangeal joints of the digits and beneath the metatarsal heads, respectively, increasing the probability of ulcer formation.

Autonomic neuropathy is associated with pathology of the sympathetic nervous system. In the lower extremity, this usually results in the absence of sweat production causing drying and scaling of the skin, increasing the likelihood of cracking and fissuring. These cracks or fissures, especially in the heel, may then serve as a portal of entry for bacteria, creating an increased chance of infection.

Other factors precipitating diabetic foot ulcers have been identified and are listed in Table 26.1. A risk factor classification scheme has been developed based on LOPS and other comorbidities (i.e. vascular disease) (Table 26.2). This classification system provides treatment recommendations and suggested follow-up time.

Structural abnormalities/gait abnormalities

Structural deformities of the foot and ankle can be a potential cause of increased pressure and subsequent ulceration. The development of foot ulceration is often based on a biomechanical abnormality.[8,9] It is important to evaluate both feet of a patient for any potential problem areas. As described earlier, motor neuropathy can lead to the loss of intrinsic foot musculature causing a deformity (and subsequent overpowering of the intrinsics by the larger extrinsic muscles). Identification and management of any of the following will assist in prevention of potential ulceration: hammertoe, claw toe, bunion, tailor's bunion (bunionette), hallux limitus/rigidus, flat feet, high arched feet, Charcot deformities, or any postsurgical deformities such as amputations. Limited joint mobility should also be evaluated as it can cause an increase in vertical and shear force in certain areas, particularly to the plantar hallux, and lead to tissue breakdown. The Achilles tendon should be evaluated for any type of functional shortening causing equinus deformity. It is common to find an increase in glycosylation of soft tissues and tendons causing contracture of the muscles in the

TABLE 26.1: Risk Factors For Ulceration

General or Systemic Factors	Local Factors
• Uncontrolled Hyperglycemia • Duration of Diabetes • Peripheral Vascular Disease • Older Age • Chronic Renal Disease • Blindness or visual loss	• Peripheral Neuropathy • Structural Foot Deformity • Limited Joint Range of Motion • Trauma • Improperly fitted shoes • Callus • Prolonged Elevated Focal Pressure • History of Previous Ulcer or Amputation

TABLE 26.2a: American Diabetes Association Risk Classification for The Diabetic Foot.

Risk Category	Definition	Treatment Recommendations	Suggested Follow-Up
0	No LOPS, no PAD, no deformity	– Patient education including advice on appropriate footwear	Annually (by podiatrist)
1	LOPS ± deformity	– Consider prescriptive or accommodative footwear – Consider prophylactic surgery if deformity cannot be safely accommodated in shoes. Continue patient education	Every 3–6 months (by podiatrist +/– specialist)
2	PAD ± LOPS	– Consider prescriptive or accommodative footwear – Consider vascular consultation for combined follow-up	Every 2–3 months (by podiatrist +/– vascular surgeon)
3	History of ulcer or amputation	– Same as category 1 – Consider vascular consultation for combined follow-up if PAD present	Every 1–2 months –(by podiatrist)

TABLE 26.2b: The University of Texas (UT) Wound Classification System

UT System		
Grade	**Description**	**Stage**
1	Pre- or post ulcerative lesion	A–D
2	Superficial	A–D
3	Penetrated to tendon or capsule	A–D
4	Penetrates to bone	A–D

Stages: A = no infection or ischemia; B = infection; C = ischemia; D = infection and ischemia

posterior compartment of the leg.[10-12] This will cause an increase in plantar pressures in the forefoot making this area more prone to breakdown.

Gait evaluation and muscle testing should also be conducted to evaluate any potential abnormality with ambulation and muscle strength. Testing of the muscles in the lower extremity should be done both actively and passively, weight bearing and non-weight bearing. Plantar foot pressures can also be assessed with the use of a Harris ink mat or pressure sensitive foot mat.

Angiopathy

In combination with the risk factors for ulceration discussed above, the presence of vascular occlusive disease increases the risk of potential amputation. Vascular disease is a common finding in individuals with long-standing diabetes. While vascular insufficiency alone is not usually the primary cause of ulceration, inadequate perfusion can inhibit ulcer healing, leading to further tissue necrosis and the inability to clear infection. Table 26.3 provides a list of other potential risk factors for amputation. Most cases of vascular disease in individuals with diabetes, interestingly, affect the infrapopliteal vessels with relative sparing of the pedal vessels. In many instances, this allows for a distal bypass to a pedal target artery in order to increase the blood flow to the foot (Figure 26.1). The vascular examination is addressed in further detail later in this chapter.

DIAGNOSIS

History and rapid visual screening

A thorough history and physical examination of each patient presenting with diabetic foot pathology should include a history of pedal wounds, history of prior amputations, and lower extremity vascular interventions.[13,14] Physical examination should note the types of deformities present, neurological status, vascular status, and dermatological presentation. The patient should be instructed to remove both shoes and socks for their examination. A systematic approach should be taken in order to avoid missing any

TABLE 26.3: Risk Factors for Amputation:

- Peripheral Neuropathy (LOPS)
- Vascular Insufficiency (PAD)
- Infection
- Structural Foot Deformity
- Trauma
- History of prior foot ulcer or amputation
- Charcot foot
- Poor glycaemic control
- Older age
- Male gender
- Ethnicity (higher in Hispanics and African-Americans)

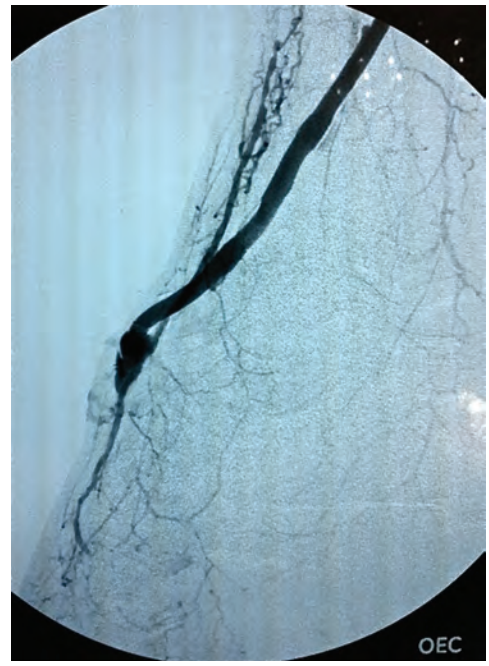

FIGURE 26.1: Dorsalis Pedis Bypass

important aspects of the examination. All surfaces of the foot and ankle should be evaluated including the nails, digits, interdigital webspaces, the soles, and the heels, inspecting for cracks, blisters or bullae, hyper/hypopigmentation, fissuring, calluses, and ulcers. The footwear should also be inspected for signs of wear patterns, foreign bodies, and any irregularities. Any gross deformities as described above can be identified during this rapid visual screening.[14]

Neurological examination

As stated earlier, peripheral sensory neuropathy is the major risk factor for the development of a diabetic foot ulcer. There are many simple, noninvasive examinations that can be performed to test and monitor sensation. A simple history of neuropathic symptoms such as tingling, burning, numbness, the feeling of insects crawling on the feet (formication) can help to identify those patients at risk for developing foot ulceration.[15]

Monofilament testing

One of the most common methods used to assess neuropathy is the use of the Semmes-Weinstein monofilament (10-g) wire.[14,15] The nylon monofilament is placed on ten different pre-determined locations on the foot and pressed down manually until there is a slight bend in the wire (Figure 26.2). The patient is instructed to say 'yes' if he or she thinks they feel slight pressure or sensation. If the patient is unable to feel the sensation at two or more locations then it is safe to assume that protective sensation is lost.

Vibration testing

Vibratory perception testing can be assessed utilizing several different modalities. The more traditional method of testing is by the use of a 128-Hz tuning fork. The tuning fork is struck and then placed on a prominent bony surface of the foot, such as the great toe or metatarsal head. The patient is instructed to identify when the vibration stops.

As an alternative, a vibratory perception threshold monitor (VPT – Diabetica Solutions, San Antonio, Texas, USA) (Figure 26.3) can be used to test for vibration perception threshold and is useful in identifying those at high risk for development of an ulcer. The instrument consists of a hand piece with a testing probe on the end, a motor, rheostat, and voltmeter. The probe is held gently on the distal aspect of each hallux, or distal most prominent area. The rheostat is slowly increased until the patient senses the vibration. Once the patient identifies the sensation, the rheostat is then decreased until the sensation is no longer felt. The average value of the two numerical readings is taken and the level of sensation is documented to the location where the numbers were obtained. Average values above 25 Volts

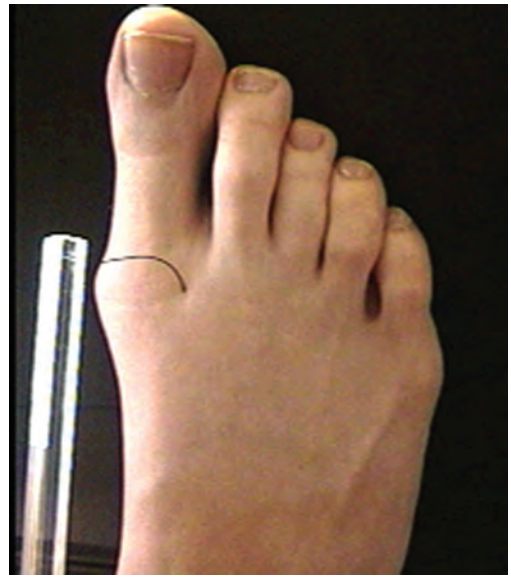

FIGURE 26.2: Proper use of S-W Monofilament when applied to the foot. Slight bend of the filament noted.

FIGURE 26.3: Vibratory Perception Threshold Monitor (VBT)

are considered positive for neuropathic changes.[14]

Dermatologic examination

Following assessment for any loss of sensation, evaluation of the integumen is a critical part of the foot screening. The skin can be evaluated for color, texture, turgor, quality, and presence of any areas of dryness or fissures. Calluses, if present, can be problematic as they indicate areas of increased pressure and an ulcer can form under the hyperkeratotic lesion, which may cause hemorrhage beneath the callus. Debridement of these areas is recommended in order to reduce a potential focus of pressure.[16, 17]

Appearance of the nails should also be noted. If there are nails that are incurvated or ingrown, these could be a potential area for skin breakdown and possible infection. Other nail issues to be aware of are onychomycosis (fungal nails), dystrophic nails, atrophy or hypertrophy, or paronychia (infected ingrown nail), as all can be potential problems.

Evaluation for any ulceration to the foot or lower leg should be assessed. Important characteristics of ulcers are depth, size, presence of fibrotic or granulation tissue, location, and whether or not the area appears to be infected.

Anatomy of occlusive disease – vascular examination

For decades, clinicians incorrectly believed that ischemia in diabetes was due to microvascular occlusion of arterioles, so-called small vessel disease.[18] However, this assumption has been disproven by subsequent study.[19,20] Diabetic vascular disease is a macrovascular phenomenon, commonly affecting infrapopliteal arteries with calcified stenoses and occlusions. More than 90% of patients have sparing of at least one major artery at the ankle level. The peroneal artery is often the last infrapopliteal artery to occlude; it provides blood flow to the foot through anterior and posterior perforating branches to the tibial arteries. Bypass to an infrapopliteal artery usually provides adequate blood flow to the foot, although some patients appear to have more compartmentalized pedal flow. Some patients with heel ulcers remain slow to heal

after dorsalis pedis artery bypass, suggesting inadequate pedal circulation.[21] Thus, if possible, patients with ischemic ulcers should preferentially receive a bypass to the arterial bed directly supplying the ulcer; this axiom is especially pertinent to patients with large heel ulcers.

Prediction of wound healing: assessment of perfusion

Pedal blood flow should be assessed before any surgical intervention on the diabetic foot. Absence of pedal pulses, dependent rubor, pallor on elevation, and loss of hair are clinical signs of advanced peripheral artery disease. If there is concern for ischemia, noninvasive testing is an appropriate initial diagnostic choice. The ankle-brachial index (ABI), while nearly 100% specific for PAD in non-diabetics, can be falsely elevated (>1.3) in diabetic patients due to medial calcinosis of the affected arteries. More useful are the systolic toe pressure and the toe-brachial index, since digital arteries are seldom calcified in those with diabetes.[22] Additional tests to assess foot perfusion include skin perfusion pressure,[23,24] and transcutaneous oxygen pressure (TcP_{O2}).[25-27]

Currently there is no single test or combination of tests that can always predict wound healing. Apelqvist and colleagues prospectively studied 314 consecutive patients with diabetic foot ulcers.[22] Primary healing occurred in 85% of patients with a toe pressure greater than 45 mmHg, while only 36% of patients with a toe pressure of 45 mmHg or less healed without an amputation. No patients with an ankle pressure less than 40 mmHg healed primarily. Kalani and colleagues also prospectively studied 50 patients with diabetic foot ulcers over a 12-month period.[26] A toe pressure of at least 30 mmHg had a 15% sensitivity, 97% specificity, and 67% positive predictive value for wound healing. In that same study, TcP_{O2} was also evaluated; a TcP_{O2} of at least 25 mmHg had an 85% sensitivity, 92% specificity, and 79% positive predictive value for wound healing. Likewise, Carter and colleagues reported that TcP_{O2} measurements correlated significantly with the risk of major amputation, with a relative risk of 2.55 for a TcP_{O2} of 20 mmHg of less and 2.22 for a TcP_{O2} of 30 mmHg or less.[27] In addition, an ankle pressure of 50 mmHg or less had a 5.83 relative risk of major amputation. Skin perfusion pressure (SPP) has also been used to predict wound healing. Faris evaluated 61 diabetic subjects with wounds and found that only 5% healed with an SPP less than 40 mmHg, while 88% of patients healed with an SPP higher than 40 mmHg.[23] Yamada and colleagues studied 211 subjects, half of who were diabetic, and compared SPP to other noninvasive methods to predict wound healing. They found that SPP was superior to ankle pressure, toe pressure, or TcP_{O2} for predicting wound healing. An SPP of 40 mmHg had a 72% sensitivity and 88% specificity for predicting wound healing.[24] Furthermore, the accuracy of prediction increased when a toe pressure greater than 30 mmHg was combined with SPP.

Arterial imaging

A number of techniques of arterial imaging are currently in use: arterial duplex scanning, computed tomographic angiography (CTA), magnetic resonance angiography (MRA), and digital subtraction arteriography (DSA). Duplex scanning is non-invasive and does not require administration of contrast. However calcification of the infrageniculate arteries can be problematic in many patients. Both CTA and MRA have the advantage of being noninvasive; however, both these studies are limited

in diabetic patients with impaired renal function. CTA requires a large bolus of iodinated contrast, and the gadolinium-based contrast used in MRA has been associated with nephrogenic systemic fibrosis.[28] In addition, MRA tends to overestimate the degree of arterial stenosis. Using selective catheterization techniques, DSA can produce high-quality images with low volumes of iso-osmolar contrast, thereby minimizing contrast-induced nephropathy. In addition, carbon dioxide can be selectively utilized as a 'contrast' agent; it is not nephrotoxic and quickly dissolves in blood before travelling to the lungs where it is readily expelled. DSA also has the advantage that angioplasty and stenting, if appropriate, may be performed at the time of diagnostic arteriography (Figure 26.4).

Soft tissue imaging

Imaging studies should be conducted to evaluate any underlying bone or soft tissue deformity. The diabetic foot is prone to many common and uncommon infectious and non-infectious processes due to the combination of vascular and neurological impairments. Imaging studies may be difficult to interpret and may lack specificity; therefore, a 'shotgun' approach to imaging studies should be avoided. Appropriate imaging studies should be ordered to establish

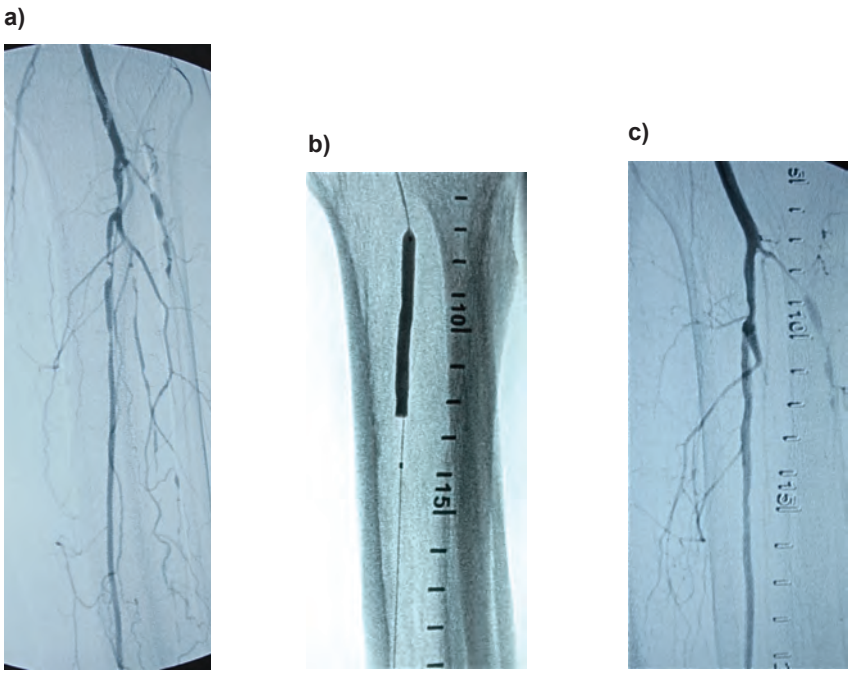

FIGURE 26.4: 56 year-old man with diabetes, renal insufficiency (serum creatinine 0.180mmol/L), absent foot pulses and non-healing dorsal foot ulcer for 6 weeks. Angiography showed normal arteries to the trifurcation and severe tibial artery occlusive disease with a long stenosis in dominant posterior tibial artery. Angioplasty markedly improved circulation and the wound healed with debridement, negative pressure wound therapy, and subsequent split-thickness skin graft placement.

a) long segment, proximal posterior tibial artery stenosis

b) 8 cm long, 4 mm percutaneous transluminal angioplasty (PTA) of proximal posterior tibial artery stenosis

c) excellent angiographic result after balloon angioplasty

or confirm a suspected diagnosis in order to properly manage the patient. For example, it is very difficult to differentiate acute Charcot arthropathy from osteomyelitis on plain radiographs. The reliability of imaging studies is diminished in the presence of arterial occlusive disease, Charcot arthropathy or after recent surgery or trauma.[29] A thorough history and physical examination and clinical overview by the treating physician must be correlated with the imaging studies in order to interpret them correctly.

Plain radiographs should be the initial study considered for patients that present with any signs or symptoms of foot problems. These studies help to identify osteomyelitis, fracture, dislocation, foreign bodies, soft tissue gas, arterial calcification as well as any other biomechanical or structural deformity. It should be noted that it might take time for acute osteomyelitis to be detected on plain film radiographs and serial radiographs or magnetic resonance imaging (MRI) should be considered if osteomyelitis is strongly suspected.

Computed tomography (CT) scans may be indicated if plain films fail to depict a suspected bone or joint deformity. CT offers higher anatomic detail and resolution in regards to bone and joints. 3-D CT reconstruction can be done to provide a 360-degree view and increased visualization of any suspected abnormality.

MRI is usually preferred over a CT scan for suspected osteomyelitis due to the increased resolution of the image. MRI scans also allow the physician to visualize the extent of the infection- osteomyelitis, deep abscess and septic joints, but also other soft tissue pathology such as tendon rupture.

Bone scans can also provide some useful data when evaluating the diabetic foot. Technetium-99 methylene diphosphonate (Tc-99 MDP) as well as indium-111 bone scans can be useful in determining the presence of osteomyelitis. However, while bone scans and other scintigraphic techniques are certainly possible tools to employ, their lack of specificity often makes their specific utility problematic, particularly if used in isolation. Advanced imaging studies beyond plain radiographs should not be routine. Rather, they should only be obtained to answer specific clinical questions when the results will alter management (e.g. concern over deep space infection with equivocal clinical picture).

CLASSIFICATION SYSTEMS

Diabetes mellitus foot risk classification

Based on a thorough history and physical examination, each patient should then be classified and assigned to a specific foot risk category as outlined in Table 26.2a. These categories were designed to direct and expedite the referral process to the necessary specialist and should also serve as a guide for subsequent follow-up visits. Increased categorical levels are associated with increasing risk for ulceration, hospitalization, and amputation.

University of Texas wound classification system

While many wound classification systems exist, The University of Texas (UT) ulcer classification system was developed to provide a more uniform evaluation of diabetic foot wounds. Like other classification systems the UT wound classification system builds on the depth-ischemia classification however, the UT system also considers infection.[30] The presence of infection and ischemia has been found to be more strongly predictive of outcome than the wound depth alone.[31] The UT system uses a 4 by 4 matrix (classes

A to D, wound depths 0 to 3) and evaluates 3 factors of ulceration, which include depth, infection, and peripheral arterial disease (PAD).[32] The frequency and level of amputation increases in deeper wounds and in the presence of infection and PAD.[30] Regardless of which specific classification is used, the key factors of depth, infection and ischemia should generally be communicated in some form. This classification is listed in (Table 26.2b).

CLINICAL PROBLEMS AND PRINCIPLES OF MANAGEMENT

Ulceration

Initial presentation of the diabetic foot usually entails the presence of an open ulcer, located on the plantar aspect of a patient's foot. Most patients will not suspect an ulcer on the bottom of their foot until they, or a loved one, notices blood either on the floor after walking or on a sock. Advising the patient that ambulation is contraindicated with an open ulcer is often challenging especially if the patient is neuropathic, as they have lost the 'gift of pain.'[33] However, reduction of pressure to the foot is essential for treatment. By effectively off-loading the area, the risk of continued trauma and resultant infection is decreased.

Epidemiology and risk factors

Ulceration of the diabetic foot is one of the single most common problems for which medical assistance is sought in a diabetic individual. Up to 25% of patients with diabetes will have a foot ulceration during their lifetime.[4] Remarkably, at least 50% of those ulcerations will become infected, and in 20% of those cases an amputation is required.[34] There are 81,000 major amputations performed on individuals with diabetes in the United States annually. Conservatively, 60% of all non-traumatic amputations occur in the diabetic patient.[35] Studies have shown that after a major amputation the contralateral limb develops a serious lesion in 50% of cases.[36] The 5 year adjusted mortality rate after a major amputation of a limb is 46%;[6] this is alarming and is higher than the mortality rate for many forms of cancer. It has also been shown in more than 85% of lower extremity amputations a wound was a critical aspect of the causal pathway.[8,34,37,38] The cost of treating diabetic foot ulcers is also growing at a staggering pace, reaching nearly $30 billion in the United States in 2007.[39]

Offloading

Numerous products are available to assist in redistributing pressure over a larger unit area (off-loading) on the foot. Some examples include removable cast walkers, non-weight bearing casts with crutch/walker assist, wedge sandals (either forefoot or heel wedge), healing sandals with a multilaminar, multidurometer foot bed. However, the choice of modality should be chosen with respect to the patient's functional status. For example, if a patient presents with a plantar forefoot ulcer, a wedge shoe that does not allow the forefoot to bear weight would be an appropriate device. However, if the patient is not properly educated on use of the device and/or has issues with balance, the offloading modality could cause the patient be unstable when walking. In regards to diabetic shoes, whether custom-made or off the shelf, it is important to keep in mind one key fact. While diabetic shoes are effective in preventing ulcerations, they are (in general) not very effective in healing them.

For many years the gold standard in off-loading an ulceration on the foot has been the total contact cast (TCC).[40] This fully weight-bearing cast consists of a multiple

layers of both fiberglass and plaster with minimal padding. When applied properly, the TCC allows the patient to be ambulatory. The TCC redirects pressure from the bottom of the foot, which is then redistributed over the bottom of the entire foot and up the cast. Some disadvantages of using a TCC is the time it takes to apply, the bulkiness for the patient, and the fact that the patient must wear the cast for 1 week intervals requiring weekly visits to the clinic. Newer TCC variants have reduced the time required for application of a traditional device whilst retaining many of the desirable therapeutic characteristics. An example of newer TCC variants can be seen in Figure 26.5.

Recent studies have evaluated the use of a removable cast walker rendered irremovable. This technique has been termed the 'instant total contact cast (iTCC).' This can be fabricated by simply adding a layer of fiberglass, cohesive bandage, or plaster around the leg portion (Figure 26.6). The iTCC has proven to be an effective and easy offloading device that ensures patient adherence.[41]

In many instances, proper off-loading may be all that is necessary to achieve wound healing. However, if wound healing fails in the midst of proper off-loading, adequate blood flow, and satisfactory nutritional status, surgical intervention may be warranted.

Non-vascular surgical treatment

Surgical management of the diabetic lower extremity can be a challenging and frustrating task, but can be ultimately rewarding both to the physician and patient upon healing of an ulcer and correction of the underlying cause of the deformity. Surgery in the absence of critical limb ischemia is based on three fundamental principles: presence or absence of neuropathy (LOPS),

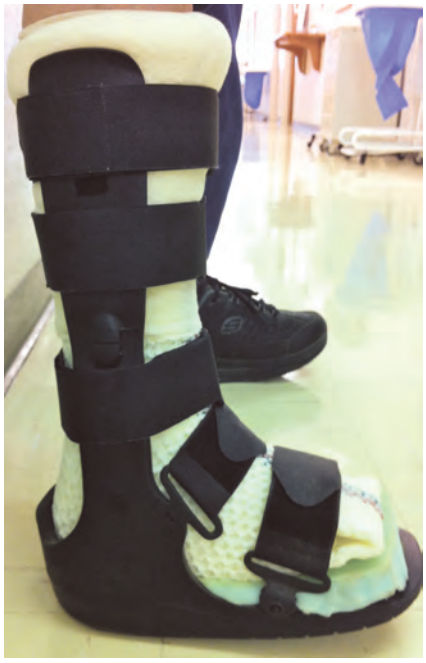

FIGURE 26.5: Anterior and lateral view of cast boot, which allows ambulation.

FIGURE 26.6: Example of an instant total contact cast (iTCC) with fiberglass wrapped around the leg portion, of an off-loading walking boot, to make it irremovable.

presence or absence of an open wound, and presence or absence of acute limb-threatening infection.[42]

A classification system has been developed outlining non-vascular surgical treatment of patients with diabetes and has been divided into four categories and highlighted in Table 26.4 and is based on indications and perceived risk.[42,43]

Class I: Elective

The goal of elective surgery is to reduce or eliminate any pain associated with a particular deformity and improve function.[42] Examples of these deformities include bunions, hammertoes and bone spurs, all in patients without peripheral neuropathy and with a low chance for ulceration. Basically any type of reconstructive surgical procedure can fall into this category with the exception of an amputation. Rarely are amputations done as an elective procedure. Only in the case of severe deformity or instability from a previous injury or surgical procedure, will an amputation be considered. Assuming good glycemic control, patients grouped to this class are not at any increased risk for the development of complications than corresponding patients without diabetes.[43]

Class II: Prophylactic

Procedures in this class are indicated to alleviate a deformity in a patient who is neuropathic however does not have the presence of an ulcer.[42] The goal of prophylactic surgery is to reduce the plantar sheer and vertical stresses. Many procedures for prophylactic surgery would be considered elective if the patient did not suffer from neuropathy. Several examples of procedures in this class include a Charcot foot reconstruction, a metatarsal head resection, a Keller arthroplasty, an Achilles tendon lengthening or a hammertoe repair.

Class III: Curative

Curative surgery often is identical to prophylactic surgery with the exception that procedures in this class are designed to speed the healing rate of an open wound but also to eliminate any potential recurrence of the ulceration.[42] Surgical procedures frequently utilized in this category may include exostectomy, digital arthroplasty, sesamoidectomy, single or multiple metatarsal head resection, joint resection or partial calcanectomy as well as Achilles tendon lengthening. Some surgeons may also elect to combine a plastic surgical flap and/or skin graft to help expedite healing.

Class IV: Emergency (Urgent)

Urgent procedures are performed to limit the spread of acute, limb and potentially life-threatening infection. Most often these procedures are done in the presence of significant ischemia. They require the removal of all infected and necrotic tissue to the level of viable soft tissue and bone and usually involve some level of amputation. When at all possible these procedures should be performed to allow for maximum amount of function to the extremity to be maintained.

With respect to any of the above classifications it is best to evaluate the vascular status of the patient to consider the necessity of any prior or subsequent arterial procedure that may be needed.[42]

Post-operative management

The management of the diabetic foot patient in the post-operative setting is much the same as a patient without diabetes. Adequate pain control, rest, elevation of the extremity and proper dietary intake are all recommended. One key difference is the timeframe for protected weightbearing or complete non-weightbearing. It is often

stated that diabetics require twice the healing time as a non-diabetic. That means that if a simple bunionectomy in a non-diabetic requires 4 weeks of protected weightbearing, the same procedure in a diabetic may require 6-8 weeks of protected weightbearing. The same can be said for a procedure requiring non-weightbearing. Most surgical procedures done for class 1–3 can be managed with protected weightbearing.[43] Most class 4 procedures will require some period of non-weightbearing or protection. The same can be said for any type of surgical implant or skin closure technique. For example, leaving skin sutures in place for an additional 1–2 weeks is advantageous over early removal to allow for the tissue to heal.

Surgical and non-surgical complications in this patient population are to be expected. Joint dislocation, bone fracture, surgical incision dehiscence, new ulceration or re-ulceration, and infection are all commonplace, not infrequently leading to re-hospitalization. Proper management by use of off-loading, local wound care, antibiotics and patient education will all be beneficial when dealing with any potential complication. It should also be kept in mind that not all diabetic foot complications can be prevented, but all can be adequately managed.

Infections

The Infectious Diseases Society of America (IDSA) has produced a classification system for diabetic foot infections, which is based on clinical signs and symptoms (Table 26.5). The IDSA divides infections into four categories, uninfected, mild, moderate, and severe.[44] These guidelines and classification system has become a reliable clinical predictor of the outcome of a diabetic foot infection.[45] It is important that initial antibiotic therapy cover the broad range of potential aetiological organisms in the diabetic foot until targeted therapy can be instituted once appropriate cultures have been obtained.

Charcot arthropathy

Charcot arthropathy is defined as a progressive condition characterized by joint dislocation, pathologic fractures, and severe destruction of the pedal framework (Figure 26.7). It is typically seen individuals with neuropathy and can be easily confused with acute infection or osteomyelitis. A

TABLE 26.4: Classification of non-vascular Diabetic Foot Surgery

Class	Type	Definition
1	Elective	Procedure performed on patient ***with protective sensation intact*** to eliminate pain or to improve function
2	Prophylactic	Procedure performed on patient ***with protective sensation absent but no open wound*** to reduce deformity and reduce occurrence/ recurrence
3	Curative	Procedure performed on patient **with an open wound** with the goal of promoting healing and reducing risk for recurrence
4	Emergency	Procedure performed with goal of limiting the spread of limb- or life-threatening infection

patient will often present with a red, hot, swollen extremity with no sign or history of ulcer or break in the skin (Figure 26.8). Usually all other constitutional signs of infection are absent and laboratory values are within normal limits.

Pedal manifestations of Charcot foot result in a debilitating deformity frequently leading to an amputation. While not well understood, several theories regarding the development of the disorder exist with many authors believing it is caused by a combination of the neurovascular and neurotraumatic theories. Neuropathy and trauma seem to be the common event preceding the development of an acute Charcot foot. As a result of the trauma and associated autonomic neuropathy, blood flow to the foot increases causing osteopenia and weakening of the bony structure. Fracture is often associated with unrecognized injury or minor trauma that might otherwise appear innocuous.[46,47] An unfortunate cycle continues as the patient ambulates, without pain, causing further destruction of the foot.

The initial diagnosis of acute Charcot is often clinically based on the characteristics listed above. It can become complicated as

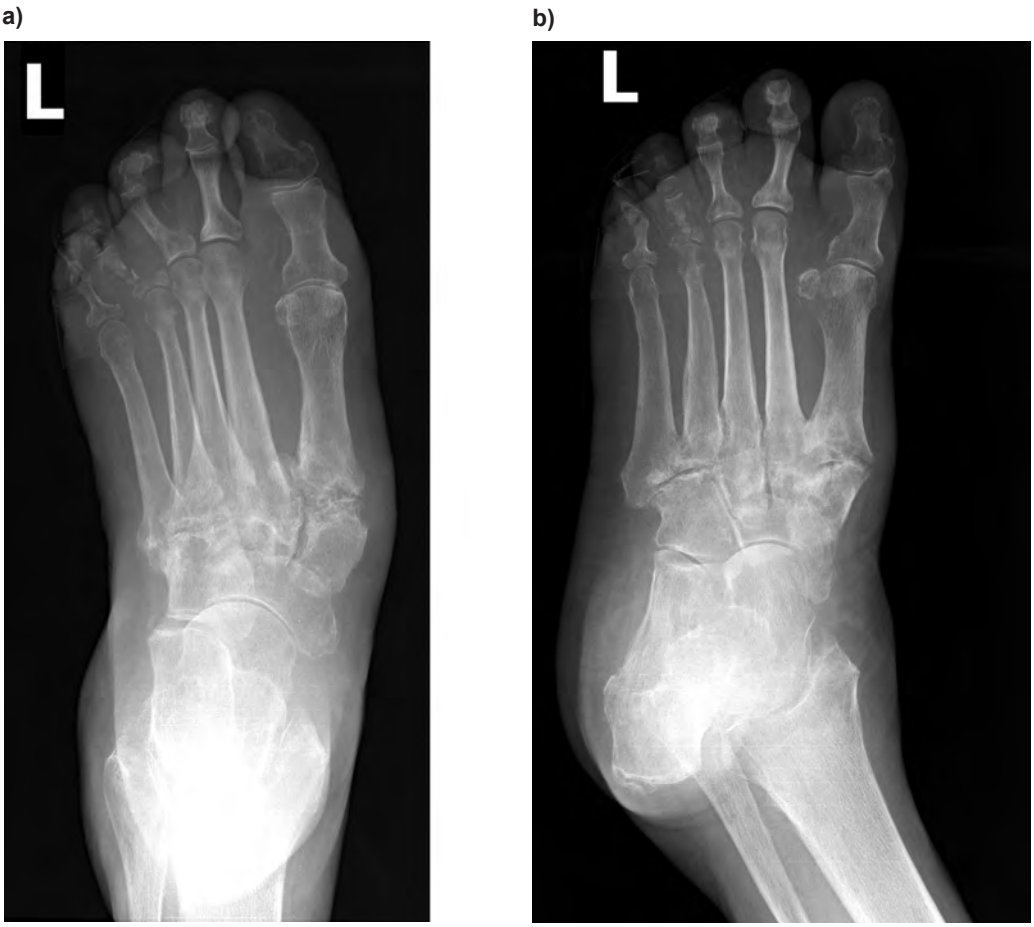

FIGURE 26.7a & b: X-ray of acute Charcot arthropathy. This plain x-ray demonstrates degenerative changes in the tarso-metatarsal joints (Lis-franc joints) with joint space narrowing and subchondrial sclerosis, consistent with Charcot arthropathy. Also noted is bone destruction and pathological fracture of the proximal phalanx of the 4th toe, indicative of osteomyelitis of this bone.

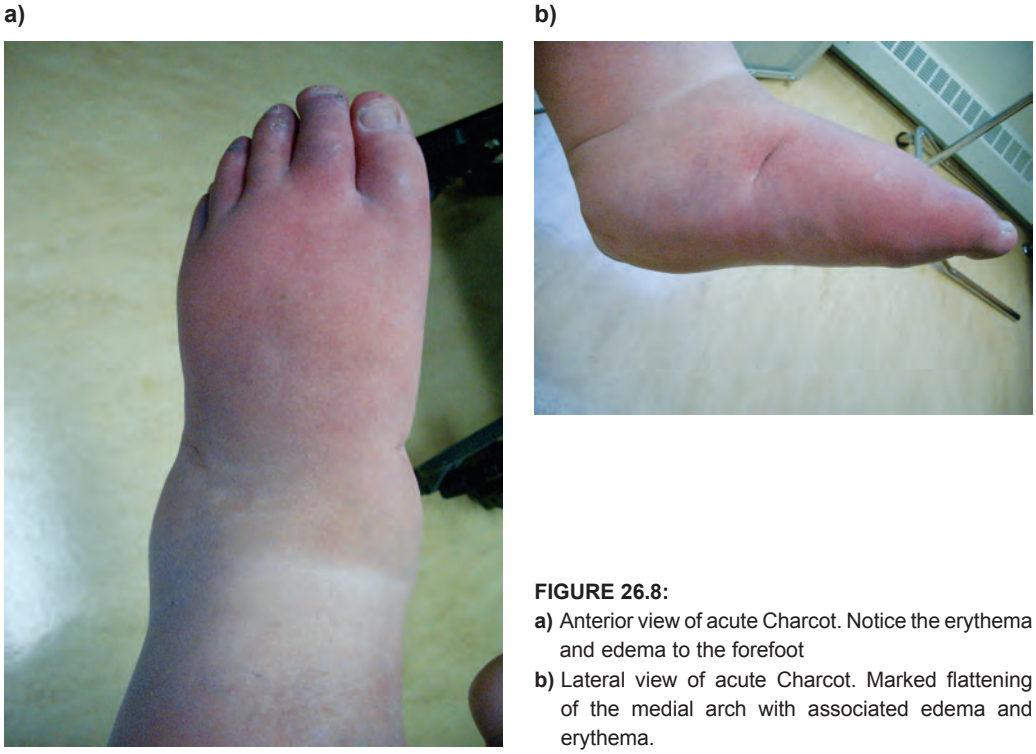

FIGURE 26.8:
a) Anterior view of acute Charcot. Notice the erythema and edema to the forefoot
b) Lateral view of acute Charcot. Marked flattening of the medial arch with associated edema and erythema.

patients often present with a concomitant ulcer, which raises questions of possible osteomyelitis. Someone presenting with the previously mentioned characteristics and lack of an open ulcer are more pathognomonic of acute Charcot arthropathy. Routine laboratory values are often of little use in diagnosing Charcot. They are valuable, however, with respect to excluding potential infection. An elevated WCC with a left shift is often seen with acute infection and not with acute Charcot. Elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) level may also be elevated in acute infection but they often respond to any inflammatory process and thus are nonspecific. The most important diagnostic aid in determination of acute Charcot is the high index of clinical suspicion when a neuropathic patient presents with a deformed or swollen foot.

Treatment of Charcot consists of immediate immobilization and stress reduction. Most physicians would recommend strict non-weight bearing to the affected limb. However, this then subjects the contralateral extremity to increased pressure thereby predisposing it to repetitive stress and the potential for ulceration or even acute Charcot.[48] Nonetheless, non-weight bearing by means of a short leg plaster or fiberglass cast, or TCC remains the gold standard in the acute phase of the disease.[40]

Following the initial period of off-loading, skin temperatures and edema of the affected extremity will decrease. At this point protected weight bearing is permitted, usually with some sort of assistive device. This can be achieved through use of a Charcot restraint orthotic walker (CROW) (Figure 26.9), total contact cast, fixed ankle walker, bivalved casts or patellar tendon-

bearing braces, to name a few. The average time of rest and immobilization in a patient that has undergone an acute Charcot event is 4 to 6 months before returning to permanent and proper footwear.

If offloading is unsuccessful or if the deformity is unstable, reconstructive surgery may be considered. The goal of surgical intervention in the Charcot foot is to provide a stable, plantigrade foot that may be appropriately accommodated with proper shoe gear.[49] Most surgical interventions consist of an exostectomy for prominent plantar bony prominences or 'rocker-bottom' deformities that cause ulcerations and could lead to potential infection.[50] Other, more complex procedures such as arthrodesis of the midfoot and hindfoot, use of external fixation, and intramedullary nail have also been used in the latent stage of the Charcot disease process.

FIGURE 26.9: Charcot Restraint Orthotic Walker (CROW) boot. A patient is able to ambulate with this device once the acute phase has passed.

PREVENTION

Diabetic foot care requires an interdisciplinary team approach given the progressive nature of the disease in the foot. It is unlikely that one individual medical or surgical specialty is able to manage all aspects of diabetic lower extremity disease and appropriately manage these patients. Recent evidence suggests a reduction in major amputation rates following the development of an interdisciplinary approach to limb salvage.[51] The components of a limb salvage team are predicated on the pathology at presentation. The core of the team typically starts with clinicians caring for the structural aspects of the foot (Podiatrists), along with clinicians caring for the vascular integrity of the lower extremity (Vascular Surgeons). Other specialties of the team, to add a more comprehensive care model may include Endocrinology, Infectious Diseases, Orthopaedic surgery, Physiotherapy, Plastic Surgery, Nursing and Orthotics/ Prosthetics.

Vasculopathy and neuropathy are two major contributors to diabetic foot disease and subsequent ulceration. Therefore, appropriate utilization of an interdisciplinary team approach, as stated above, will help to address the varying factors associated with lower extremity ulceration and reduce amputation. A diabetic rapid response acute foot team has been proposed in an effort to combine the knowledge of certain specialties to promote limb salvage.[52] This is an interdisciplinary team model whose core involves the ability to rapidly diagnose and provide treatment to patients with lower-extremity complications of diabetes, utilizing seven basic skill sets (Table 26.6). At the forefront of the team are members from podiatry (Toe) and members from vascular surgery (Flow). These specialties, with adjunctive teams added as necessary,

combine to collectively posses the ability to perform the 7 essential skills, stated above, to be effective in promoting limb salvage. This model has been effective at the University of Arizona's Southern Arizona Limb Salvage Alliance (SALSA) in which the Vascular Surgery/Podiatry team approach has been able to significantly reduce the number of major diabetes-associated lower-extremity amputations as well as provide a framework for prevention of diabetic ulcers. In fact, abundant data suggest that establishing such comprehensive diabetic foot care teams can significantly reduce the incidence of major amputation in both community and academic hospital settings.[53]

TABLE 26.5: Infectious Diseases Society of America (IDSA) Diabetic Foot Infection Classification

Clinical Presentation	IDSA Infection Severity	Threatened Limb Class
Wound without inflammation or purulence	Uninfected	Not applicable
Presence of >2 manifestations of inflammation, cellulitis <2cm surrounding ulcer, no systemic symptoms of infection	Mild Infection	Non-limb threatening
Presence of infection with >2 cm of surrounding cellulitis; any infection with presence of gangrene, abscess, deep tissue involvement or gas in tissue; no systemic signs/ symptoms of infection	Moderate Infection	Limb threatening
Presence of infection as above with the presence of systemic signs or symptoms	Severe Infection	Life and limb threatening

TABLE 26.6: Seven Essential Skills of a Diabetic Rapid Response Acute Foot Care Team (DRRAFT).

1. Ability to perform hemodynamic and anatomic vascular assessment with revascularization
2. Ability to perform a neurologic assessment
3. Ability to perform site-appropriate culture technique
4. Ability to perform wound assessment and staging/grading of infection and ischemia
5. Ability to perform site-specific bedside and intraoperative incision and debridement
6. Ability to initiate and modify culture-specific and patient-appropriate antibiotic therapy
7. Ability to perform appropriate postoperative monitoring to reduce risks of reulceration and infection

CONCLUSION

Treating the diabetic foot, but moreover the patient with diabetes, is an extremely challenging yet rewarding experience. As the incidence continues to grow worldwide the likelihood of people with diabetes constituting a significant proportion of a vascular and podiatric surgical practice is quite high. Common, seemingly trivial foot problems such as calluses, corns, ingrown nails, and dry scaly skin, may provide sufficient trauma to develop into limb-threatening problems. Early recognition and diagnosis of these factors combined with aggressive preventive measures and a multidisciplinary team approach will all be beneficial in the treatment of the diabetic foot with the ultimate goal of amputation reduction and prevention.

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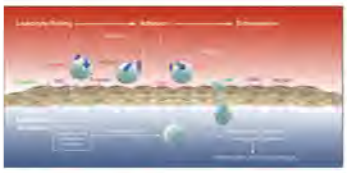
Cover design by Emma Spoehr



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Mechanisms of Vascular Disease

Edited by Robert Fitridge and Matthew Thompson

BARR SMITH PRESS

An imprint of
The University of Adelaide Press