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Gary Keller, author of “The One Thing,” says that “people who are crazy enough to think they can change the world, are usually the ones who do.” We embarked upon the mission of educating health care professionals in Nigeria on the art of treating the diabetic foot 2 years ago. Dr. Afokoghene R ISIAVWE, of Rainbow Specialist Medical Centre in Lagos recognized the shortfall of training for foot and ankle disorders in general, and the untoward consequences patients living with diabetes were experiencing as a result of this shortfall. Finally, after years of attempting to set up training conferences, the Rainbow Specialist Medical Centre partnered with The Podiatry Institute, Decatur, Georgia USA, to facilitate the inaugural training conference in March 2014. We were also able to gain support of the World Diabetes Foundation as they too share in the mission to increase training and education around the care of the diabetic foot.

This manual serves as the initial iteration of a training guide to educate healthcare professionals in Nigeria on treatment of feet in persons living with diabetes. It is broken into modules that begins with basic anatomy and physiology of the foot and ankle. Other highlights include algorithms recognizing at risk diabetic feet during screening, and several pathways for treatment of the various disorders one may encounter. We also aim to simplify the initial and subsequent exams through standardized exam protocols. Finally, various treatment protocols are explored including conservative and surgical options as well as when such treatment should be implemented. Research and clinical experience in The United States has proven that a large majority (greater than 85%) of diabetic foot ulcers are preventable with proper foot and ankle care provided by a podiatrist (or other trained foot and ankle specialist). This is important because untreated diabetic foot ulcers, more often than not, lead to amputation, and the 5 year survival rate after major lower extremity amputation is less than 50%. Therefore if we can prevent lower extremity amputations, we can save lives. This manual gives the user the tools to set up adequate treatment facilities as well as the ability to confidently identify at risk patients, screen newly diagnosed diabetics, educate patients and family members on proper foot care and hygiene, and establish treatment regimen that can potentially saves limbs, and ultimately saves lives.

It is an incredible honor to be a part of the team that brings education and training on treatment of the diabetic foot to the Lagos, Nigeria. I have no doubt that these initial efforts will be the catalyst that changes the way healthcare professionals approach and treat patients living with diabetes in Nigeria and the continent of Africa as a whole.

Rahn A. Ravenell, DPM - Team Leader - Nigeria Conference on the Diabetic Foot - Board Certified, American Board of Foot and Ankle Surgery - Fellow of the American College of Foot and Ankle Surgeons - Board of Directors, The Podiatry Institute - Coastal Podiatry, LLC Mount Pleasant, SC USA
Worldwide, Diabetes Mellitus is reaching epidemic proportions. According to the International Diabetes Federation, the majority of new cases would come from developing countries, with prevalence in Africa likely to double within the next two decades. Already burdened with infectious diseases Africa, and indeed Nigeria cannot afford to watch while her citizens come down with complications of diabetes mellitus. Nigeria, the most populous black nation is also expected to have the highest number of persons living with diabetes on the African continent. Indeed local data show we are already beginning to experience increase in Diabetes prevalence in Nigeria, and with this increased prevalence accompanying increase in complications like diabetes mellitus related foot ulcers and amputations. A trip to many of the medical and surgical wards of the Government owned health institution would give you an idea of the burden of the diabetes foot ulcer in Nigeria, which also accounts for lengthy hospital stays and loss of productivity and income to the affected individual and his family. One also cannot comprehensively begin to describe the emotional, psychological, and financial implications of diabetes related foot ulcers and amputations.

The aim of the Diabetes Podiatry Initiative Nigeria is to empower health care workers to provide good foot care services to persons Living with diabetes in Nigeria and to create awareness about the need for a structured foot care training program in Nigeria. We at Rainbow Specialist Medical Centre Nigeria in collaboration with the The World Diabetes Foundation through this initiative aim to raise the standards for foot care practice in Nigeria, especially as it relates to Diabetes Mellitus.
### Module 1: Anatomy and Physiology of the Foot

#### SELECTED ANATOMY & NORMAL PHYSIOLOGY

#### OSTEOLOGY

Table 1-1: Leg and foot ossification dates.

<table>
<thead>
<tr>
<th>OSSICLE</th>
<th>PRIMARY OSSIFICATION CENTER APPEARS (YEARS)</th>
<th>EPIPHYSIS APPEARS (YEARS)</th>
<th>OSSIFICATION CENTERS FUSE (YEARS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal phalanx</td>
<td>Birth</td>
<td>2-3 (base)</td>
<td>15-21</td>
</tr>
<tr>
<td>Middle phalanx</td>
<td>Birth</td>
<td>2-3 (base)</td>
<td>15-21</td>
</tr>
<tr>
<td>Distal phalanx</td>
<td>Birth</td>
<td>2-3 (base)</td>
<td>15-21</td>
</tr>
<tr>
<td>1st metatarsal</td>
<td>Birth</td>
<td>2-3 (base)</td>
<td>15-18</td>
</tr>
<tr>
<td>2nd metatarsal</td>
<td>Birth</td>
<td>2-3 (head)</td>
<td>15-18</td>
</tr>
<tr>
<td>3rd metatarsal</td>
<td>Birth</td>
<td>2-3 (head)</td>
<td>15-18</td>
</tr>
<tr>
<td>4th metatarsal</td>
<td>Birth</td>
<td>2-3 (head)</td>
<td>15-18</td>
</tr>
<tr>
<td>5th metatarsal</td>
<td>Birth</td>
<td>2-3 (head)</td>
<td>15-18</td>
</tr>
<tr>
<td>Medial cuneiform</td>
<td>3-4</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Middle cuneiform</td>
<td>3-4</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Lateral cuneiform</td>
<td>Birth-1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Cuboid</td>
<td>Birth-1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Talus</td>
<td>Birth</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Calcaneus</td>
<td>Birth</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Navicular</td>
<td>3-4</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Sesamoids</td>
<td>9-11</td>
<td>5-12 (apophysis)</td>
<td>15-20</td>
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<tr>
<td>Fibula</td>
<td>Birth (shaft)</td>
<td>2 (distal)</td>
<td>11-14 14-21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3-4 (proximal)</td>
<td></td>
</tr>
<tr>
<td>Tibia</td>
<td>Birth (shaft)</td>
<td>2 (distal)</td>
<td>17-19 19-21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Birth (proximal)</td>
<td></td>
</tr>
</tbody>
</table>
ACCESSORY OSSICLES
These are developmental anomalies, often separations of normal processes or tubercles, and need to be differentiated from avulsion fractures if there is a history of injury.

<table>
<thead>
<tr>
<th>Accessory ossicle</th>
<th>Location</th>
</tr>
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<tbody>
<tr>
<td>Os tibiale externum (accessory navicular)</td>
<td>Posteromedial aspect tuberosity of navicular, within insertional fibers of tibialis posterior.</td>
</tr>
<tr>
<td>Os Vesalianum</td>
<td>Proximal to well-formed tip of the tuberosity of the 5th metatarsal base; to be differentiated from fracture of the tip of the 5th metatarsal base, or nonunited or fragmented apophysis.</td>
</tr>
<tr>
<td>Os peroneum</td>
<td>Sesamoid bone within the peroneus brevis tendon insertion at the 5th metatarsal base.</td>
</tr>
<tr>
<td>Os supranaviculare (talonaviculare)</td>
<td>Dorsal aspect of talonaviculare joint.</td>
</tr>
<tr>
<td>Os intermetatarseum</td>
<td>Between the medial cuneiform and the 1st and 2nd metatarsal bases.</td>
</tr>
<tr>
<td>Os sustentaculi</td>
<td>Posterior aspect of sustentaculum tali.</td>
</tr>
<tr>
<td>Os calcaneus secondarius</td>
<td>Dorsum anterior process of the calcaneus, at the junction of the calcaneus, cuboid, head of the talus and the navicular.</td>
</tr>
<tr>
<td>Os trigonum</td>
<td>The separated posterolateral tubercle of the talus; to be distinguished from the intact trigonal process and fracture thereof (Shepherd’s fracture).</td>
</tr>
<tr>
<td>Os subfibulare</td>
<td>Distal to the tip of the fibular malleolus; to be distinguished from an avulsion fracture of lateral malleolus.</td>
</tr>
<tr>
<td>Os subtibiale</td>
<td>Distal to the tip of the tibial malleolus; to be distinguished from an avulsion fracture of the medial malleolus.</td>
</tr>
<tr>
<td>Os cuneo-1-metatarsale-1-plantare</td>
<td>Plantar aspect of the 1st metatarsal-medial cuneiform articulation.</td>
</tr>
</tbody>
</table>
ARTHROLOGY

INTERPHALANGEAL JOINTS (IPJ) (Fig. 1.1):

Ginglymus (hinge) joints with capsule that is hooded dorsally by the fibrous extensor expansion and the plantar ligament (flexor plate); reinforced with medial and lateral collateral ligaments running obliquely from the head of one phalanx to the base of the next, in a proximal-dorsal to distal-plantar direction. A plantar IPJ sesamoid may be present.

LESSER METATARSOPHALANGEAL JOINTS (MTPJ) (Fig. 1.2):

Spheroidal joints contained within a capsule that is contiguous with the extensor hood expansion dorsally, and the thickened flexor (plantar) plate. The capsule is reinforced medially and laterally by collateral and suspensory ligaments. The collateral ligament runs obliquely, proximal-dorsal to distal-plantar, from the metatarsal head to the phalangeal base. The suspensory ligament is a continuation of the extensor hood expansion that descends vertically to the plantar plate, which is tethered to the adjacent MTPJ flexor plate by the deep transverse intermetatarsal ligament. A plantar sesamoid may be invested within the flexor plate of a lesser MTPJ.

FIRST METATARSOPHALANGEAL JOINT (1ST MTPJ) (Fig. 1.3)

The 1st MTPJ is of particular importance because of the sesamoid apparatus and its relationship to the deformities of hallux valgus and varus. The tibial and fibular sesamoids are tethered by the intersesamoidal and plantar sesamoidal ligaments, present medially and laterally, running from each sesamoid to the proximal phalangeal base. The conjoined head of adductor hallucis inserts plantar-lateral into the fibular sesamoid, the 1st MTPJ lateral ligaments, and the base of the proximal phalanx.
TARSOMETATARSAL JOINTS (TMTJ, LISFRANC’S JOINT) (Fig. 1.4):

Complex consisting of articulations of the metatarsal bases with the cuneiforms and the cuboid, stabilized by inset of the base of the 2nd metatarsal (keystone) into the intercuneiform recess. The complex is arched dorsally in both the frontal and sagittal planes. There are 3 capsular elements: medial, investing the interface between the 1st metatarsal base and medial cuneiform; intermediate, investing the interface between the 2nd and 3rd metatarsal bases and the intermediate and lateral cuneiforms; and lateral, investing the interface between the 4th and 5th metatarsal bases and the cuboid. The capsule is reinforced by dorsal intercuneiform and cuneocuboid, tarsometatarsal, intermetatarsal base, and plantar tarsometatarsal ligaments. Lisfranc’s plantar ligament runs obliquely from the medial cuneiform to the 2nd metatarsal base plantarly.

CALCANEOCUBOID JOINT (CCJ):

Saddle-shaped interface invested in capsule reinforced with dorsal, lateral, and medial ligaments. The medial ligament is actually the lateral, or calcaneocuboid, portion of the bifurcate ligament. The joint is also supported by the extracapsular long plantar calcaneocuboid ligament, which extends from the calcaneal tuberosity to the bases of the 2nd-5th metatarsal bases.

TALOCALCANEONAVICULAR JOINT (TCNJ):

Commonly referred to as the talonavicular joint, an essentially condylar joint complex that suspends the head of the talus in the midfoot’s acetabulum pedis. The acetabulum pedis consists of the concavity of the posterior surface of the navicular, the anterior and middle facets of the sustentaculum tali of the calcaneus, and the plantar calcaneonavicular (spring) ligament. The TCNJ’s capsule is reinforced by the spring ligament, the calcaneonavicular portion of the bifurcate ligament, and dorsal talonavicular ligaments. The spring ligament is crucial to arch support.

MIDTARSAL JOINTS (MTJ):

Complex consisting of the talonavicular and calcaneocuboid joints, and functions reciprocally with the subtalar (talocalcaneal) joint. The STJ and MTJs are generally considered a reciprocating complex. The transverse (Kite’s angle) and sagittal plane radiographic cyma lines are useful guides to subluxation of the MTJ.

SUBTALAR JOINT (STJ):

A modified ginglymus (hinge) joint displaying triplanar motion that occurs primarily in the frontal plane, as inversion and eversion. Anatomically, the STJ is defined as the interface between the posterior facets of the calcaneus and the talus. Functionally, the STJ includes the posterior facets of the calcaneus and talus, as well as the anterior and middle calcaneal facets of the sustentaculum (an anatomical component of the talocalcaneonavicular joint), and the sinus tarsi. The sinus tarsi consists of the dorsal concavity of the neck of the talus and the plantar sulcus between the posterior facet and the sustentaculum tali of the calcaneus. The sinus tarsi is widest laterally, and is reinforced posteriorly by the talocalcaneal Y-ligament, which also envelopes the FHL tendon between the posterior processes of the body of the talus. The posterior facets are stabilized anteriorly,
TALOCRURAL (ANKLE) JOINT:
A modified ginglymus (hinge) joint that displays triplanar motion that occurs primarily in the sagittal plane, as dorsiflexion and plantarflexion. The ankle mortise consists of the concave distal tibial-bearing surface (plafond), the triangular facet of the lateral malleolus, the comma-shaped facet of the medial malleolus, and the anterior portion of the distal tibiofibular syndesmotic ligament.

The capsule may communicate with the peroneal tendon sheath, and is reinforced by the deltoid ligament (medial collateral) and the lateral collateral ligament. The deltoid ligament consists of the deep anterior tibiotalar component; and superficial tibionaviculcar, tibiocalcaneal, and posterior tibiotalar components. The lateral collateral ligament consists of the intracapsular anterior talofibular (ATFL), and the extracapsular calcaneofibular (CFL) and posterior talofibular (PTFL) ligaments. The ATFL resists ankle plantarflexion, and anterior subluxation (anterior drawer stress) of the talus out of the mortise. The CFL is deep to the peroneal tendons, and inversion injury often disrupts both the CFL and the peroneal sheath. Clinically and radiographically, anterior drawer and inversion stress manipulation of the lateral collateral ligaments, and more commonly MRI, are used to assess the injured ankle.

TIBIOFIBULAR JOINTS
The tibiofibular joints include the proximal, interosseous, and distal tibiofibular joints. The proximal joint is planar, and supported by anterior and posterior ligaments. The interosseous membrane (IO) consists of obliquely oriented, dense fibrous connective tissue running from proximal-medial to distal-lateral from the tibia to the fibula. The fibula is also situated slightly posterior to the tibia, (important when transferring tendon through the IO membrane). The distal tibiofibular joint is supported by anterior, IO, and posterior ligaments. The tibiofibular joints allow motion in frontal and transverse planes, and resists ankle dorsiflexion as the wider anterior portion of the talar dome engages the mortise.

MYOLOGY
The intrinsic pedal muscles comprise 4 layers in the plantar vault, innervated by the deep peroneal (EDB; 2nd, 3rd and 4th dorsal IO), medial plantar (FDB, FHB, abductor hallucis, 1st lumbrical), and lateral plantar (QP, abductor digitii minimi, flexor digiti minimi, all IO, all lumbricals except the 1st, and adductor hallucis) nerves.

Plantar Layer I
Abductor Hallucis
origin—medial calcaneal wall insertion—tibial sesamoid and medial base of proximal phalanx of hallux

Flexor Digitorum Brevis
origin—calcaneal tuberosity, divides at base of proximal phalanx insertion—plantar surface of middle phalanx
**ARTHROLOGY**

**Abductor Digiti Quinti**

Origin—lateral calcaneal wall
Insertion—lateral aspect base of proximal phalanx (Fig. 1.7).

**Plantar Layer II**

**Quadratus Plantae**

Origin—2 calcaneal heads
Insertion—lateral aspect of FDL tendon before it divides (Fig. 1.8).

**Lumbricales**

Origin—1st, from medial aspect of FDL to 2nd toe; 2nd, from contiguous aspects of 1st and 2nd FDL tendons; 3rd, from contiguous aspects of 2nd and 3rd FDL tendons; 4th, from contiguous aspects of 3rd and 4th FDL tendons
Insertion—medial aspect of mid-portion of proximal phalanges and fibrous expansion of the dorsal hood of the 2nd-5th toes (Fig. 1.9).

**Plantar Layer III**

**Flexor Hallucis Brevis**

Origin—medial arm from tendons of tibialis posterior inserting into the metatarsal bases, and lateral arm from the cuboid, 3rd cuneiform, peroneus longus tendon, and long and short plantar ligaments
Insertion—base of proximal phalanx on medial and lateral aspects, after investing 1st MTPJ sesamoids and plantar plate (Fig. 1.10).
Adductor Hallucis origin—oblique head arises from 2nd, 3rd, 4th metatarsal bases. Insertion—into fibular sesamoid, plantar plate, and lateral aspect base of proximal phalanx.

Flexor Digiti Minimi Brevis origin—plantar aspect of cuboid and 5th metatarsal base. Insertion—plantar aspect base of proximal phalanx of 5th toe (Fig. 1.12).

Plantar Layer IV

Dorsal Interossei (IO) origin—1st, adjacent surfaces of 1st and 2nd metatarsals; 2nd, adjacent surfaces of 2nd and 3rd metatarsals; 3rd, adjacent surfaces of 3rd and 4th metatarsals; 4th, adjacent surfaces of 4th and 5th insertion—1st, base of proximal phalanx of 2nd toe medially; 2nd-4th, lateral aspect of bases of proximal phalanges of toes 2, 3, and 4 (Fig. 1.13).

Plantar Interossei (IO)

Origin—medial aspect of 3rd, 4th, 5th metatarsal shafts and bases. Insertion—medial aspect of bases of proximal phalanges of toes 3, 4, and 5 (Fig. 1.14).
**TENDONS, SHEATHS AND BURSAE**

**Tendon Structure**

Tendons consist of dense regular connective tissue made up of tropocollagen units, created by fibroblasts, and organized to form collagen fibers. The fibers are supported within endotenon, and grouped into fasciculi which are contained within an outer epitenon. The epitenon defines the anatomical tendon. Golgi tendon organs within tendon fibers inhibit skeletal muscle contraction when excessive tension is registered.

The organized tendon is further surrounded, outside of the epitenon, by a loose, areolar and highly vascularized paratenon, wherever the tendon courses a straight line. Paratenon is contained deep to, and adherent to, the deep fascia (muscle fascia); or it is adherent to a neighboring intermuscular septum (fascia) between intact skeletal muscle bellies; or it may be adherent to deeper periosteum.

**Tendon Sheath and the Gliding Mechanism**

A tendon sheath exists where a tendon changes direction, such as about the ankle deep to the extensor, peroneal, and flexor retinaculae. The sheath is distinct from paratenon and consists of a fibrous outer septum with a synovial lining, much akin to joint capsule.

Synovial fluid bathes the tendon within the sheath. Within the sheath, on the tendon’s deep (non-friction) surface, a synovium lined fold of connective tissue called mesotenon, conveys vascularity and further supports the tendon. Mesotenon attaches to the epitenon at the hilus. At the proximal margin of the tendon sheath a double fold of paratenon, termed a plicae duplicata, invaginates a short distance into the sheath and adheres to epitenon. Similarly, at the distal margin of the sheath, a single fold of paratenon, termed a plica simplex, protrudes into the sheath. As muscle contracts, the plicae unfold and elongate as the tendon glides within the sheath as the tendon changes direction, or within paratenon where the course is straight.

**Tendon Blood Supply**

Tendon has three primary sources of blood supply: proximally, at the myotendinous junction perimysial blood vessels from the muscle belly; centrally, from paratenon and/or mesotenon; and distally, insertional periosteal vessels from bone. Synovial fluid within the sheath, and local lymphatics within the paratenon, also nourish and drain metabolites from the tendon. Occasionally, a condensed, highly organized fibrous connection, know as a vinculus, may also convey vascularity between closely approximated tendons. The Master Knot of Henry, between the tendons of FHL and the more superficial (plantar) FDL, at a level consistent with the distal margin of the sustentaculum, is just such a vinculus. Vinculi also exist between FHB and FDL near their phalangeal insertions.

**Subfascial and Subcutaneous Bursae.**

A variety of bursae occur in the foot and ankle. Bursae protect tendon and muscle from excessive friction or pressure caused by adjacent muscle, ligament or bone, or external forces in the case of an adventitious bursa. Subfascial bursae include the retrocalcaneal or pre-Achilles bursa, those at the insertions of TA, TP, and the IO; and those between the bellies of adductor digitii minimi and the 5th metatarsal, and the belly of FHB and the medial cuneiform. Subcutaneous bursae are usually adventitious in origin, and may present at the head of the 1st and 5th metatarsals, plantar to the tuberosity of the calcaneus (present in about 50% of specimens), at the medial and lateral malleoli, and occasionally posterior to the insertion of the Achilles tendon.

**NEUROLOGY**

The lower extremity nerve supply originates in the lumbosacral spine, and specifically involves spinal nerve roots L4-S3. The spinal nerve roots traverse the lumbosacral plexus to form the sciatic nerve, which divides into the tibial nerve and the common peroneal nerve near the junction of the middle and distal thirds of the thigh.
<table>
<thead>
<tr>
<th>Muscle</th>
<th>Peripheral Nerve</th>
<th>Spinal Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tibialis anterior</td>
<td>Deep peroneal</td>
<td>L₄,₅</td>
</tr>
<tr>
<td>Extensor digitorum longus</td>
<td>Deep peroneal</td>
<td>L₄,₅</td>
</tr>
<tr>
<td>Extensor hallucis longus</td>
<td>Deep peroneal</td>
<td>L₄,₅</td>
</tr>
<tr>
<td>Peroneus tertius</td>
<td>Deep peroneal</td>
<td>L₄,₅</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>Tibial</td>
<td>S₁,₂</td>
</tr>
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<td>Soleus</td>
<td>Tibial</td>
<td>S₁,₂</td>
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<td>Plantaris</td>
<td>Tibial</td>
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<td>Tibial</td>
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<td>Flexor hallucis longus</td>
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<td>Plantar interossei</td>
<td>Lateral plantar</td>
<td>S₁,₂,₂</td>
</tr>
<tr>
<td>First, second dorsal interossei</td>
<td>Lateral plantar</td>
<td>S₂,₃</td>
</tr>
<tr>
<td>Third, fourth dorsal interossei</td>
<td>Lateral plantar</td>
<td>S₂,₃</td>
</tr>
</tbody>
</table>
COMMON PERONEAL NERVE

The common peroneal nerve trifurcates near the head of the fibula, forming the lateral sural cutaneous nerve, the deep peroneal nerve, and the superficial peroneal nerve.

Lateral Sural Cutaneous Nerve This nerve ultimately anastomoses with the medial sural cutaneous branch of the tibial nerve, to form the sural nerve.

The Deep Peroneal Nerve (Anterior Tibial) (Fig. 1.15)

This nerve begins at the peroneal muscular hiatus between the fibula and peroneus longus, then passes deep to EDL on the IO membrane to innervate TA, EHL, EDL, and PT. At the ankle, it divides into medial and lateral terminal branches.

The lateral terminal branch passes deep to, and innervates, EDB and then yields three interosseous branches which supply the 2nd, 3rd, and 4th dorsal IO.

The medial terminal branch runs parallel and lateral to the DP artery. The nerve divides at the first interspace into two dorsal digital nerves supplying adjacent sides of the great and second toes, and the first dorsal interosseous muscle (which is also innervated by the lateral plantar nerve).

The muscular branches of deep peroneal nerve supply all anterior leg muscles, including peroneus tertius.

The Superficial Peroneal Nerve

The superficial peroneal nerve supplies both the peroneus longus and brevis muscles, then divides to form the medial and lateral dorsal cutaneous nerves.

The medial dorsal cutaneous nerve (Fig. 1.16) divides into two dorsal digital nerves, the medial dorsal digital branch that communicates with the medial terminal branch from deep peroneal nerve, to supply the medial aspect of the hallux.

The lateral dorsal digital branch supplies the adjacent aspects of the 2nd and 3rd toes dorsally. The lateral dorsal cutaneous (Lemont’s) nerve divides into a medial branch that supplies the adjacent sides of the 3rd and 4th toes, and a lateral branch that supplies the adjacent sides of the 4th and 5th toes.
TIBIAL NERVE

The tibial nerve traverses the calf deep to the intermuscular septum between the superficial and deep crural compartments, and in the distal third of leg runs parallel and medial to the tendoAchillis. The tibial nerve yields the medial sural cutaneous nerve that unites with the lateral sural cutaneous branch of the common peroneal nerve, to form the sural nerve.

The Sural Nerve

The sural nerve courses distally through the leg, then posterior and inferior to the lateral malleolus, en route to the lateral aspect of the foot and 5th toe. Just distal to the lateral malleolus, the sural nerve sends a communicating branch dorsally to anastamose with the intermediate dorsal cutaneous nerve. The tibial nerve also provides articular branches that innervate the knee and ankle. In the calf, the tibial nerve innervates the popliteus, gastrocnemius, soleus, plantaris, TP, FDL, and FHL muscles. Prior to bifurcation into the medial and lateral plantar nerves, the tibial nerve yields the medial calcanean branch that emerges through the laciniate ligament to innervate the skin of the heel medially and plantarly. (Fig. 1.17)

The medial calcanean nerve can be injured or entrapped in scar tissue following medial exposure (DuVries incision) of the heel, such as in plantar calcaneal spur surgery. The division of the tibial nerve into the medial and lateral plantar nerves usually occurs near the dorsal margin of the tarsal tunnel, however the bifurcation can occur at any level deep to the laciniate ligament, and occasionally it occurs proximal to the ligament. In many cases of tarsal tunnel syndrome, operative inspection reveals a far distal bifurcation of the tibial nerve at the porta pedis where the medial plantar nerve enters the anterior chamber, and the lateral plantar nerve enters the posterior chamber, of the calcaneal tunnel which is the distal continuation of the tarsal tunnel deep to abductor hallucis. The anterior and posterior canals are separated by a fibrous septum coursing from the deep surface of abductor hallucis to the medial wall of the body of the calcaneus plantar to the sustentaculum tali.

PLANTAR NERVE SUPPLY

Medial Plantar Nerve (Fig. 1.18)

The medial plantar nerve is usually slightly larger than the lateral plantar nerve, and traverses the 3rd canal of the flexor retinaculum along with the medial plantar vessels. The medial plantar nerve yields cutaneous branches innervating the medial aspect of sole; muscular branches that supply FDB, FHB, abductor hallucis and the 1st lumbrical; the proper digital branch to the plantar-medial aspect of the hallux; and three common digital nerves that yield proper digital nerves to the contiguous surfaces of the 1st and 2nd, 2nd and 3rd, and 3rd and 4th toes. The 1st common or 2nd proper digital nerve yields a branch to innervate the 1st lumbral muscle. The 3rd common or 4th proper digital nerve yields a branch that communicates with the lateral plantar nerve, and is often the site of Morton’s neuroma. The proper digital nerves supply the digital pulp, and the tips and sides of the toe,
The Lateral Plantar Nerve

The lateral plantar nerve courses through the porta pedis deep to the plantar fascia, and yields muscular branches to quadratus plantae and abductor digiti minimi; cutaneous branches to the lateral aspect of the sole; a superficial branch that divides into common and proper digital branches, and a deep branch. The proper digital branch supplies the lateral aspect of the 5th toe; and the flexor digiti minimi brevis as well as the 3rd plantar and 4th dorsal IO muscles. The common digital branch usually communicates with the digital branch of the medial plantar nerve (often the site of Morton’s neuroma), before dividing into proper digital branches to the contiguous surfaces of the 4th and 5th toes. The deep branch of the lateral plantar nerve supplies all of the IO muscles except the 4th dorsal and 3rd plantar in the 4th intermetatarsal space, all of the lumbricales except the 1st lumbral, and adductor hallucis.

Saphenous Nerve

The saphenous nerve is the terminal continuation of the femoral nerve, and courses through the thigh to emerge from the adductor canal to become subcutaneous and continue distally along the anteromedial aspect of the leg and foot. It yields a branch to the skin over the ankle, and a branch that courses distally to innervate the medial aspect of the tarsus and great toe.

ANGIOLOGY

ARTERIAL SYSTEM

The arterial supply to the lower extremities originates with the abdominal aorta, which bifurcates into right and left common iliac arteries, which then further divides to form internal and external iliac arteries. The external iliac artery becomes the femoral artery at the distal margin of the inguinal ligament. The femoral artery is palpable in the groin, and courses distally through the thigh to become the popliteal artery, which is palpable in the popliteal fossa. The popliteal artery yields muscular, cutaneous, and articular (knee) branches. The popliteal artery bifurcates to form the anterior and posterior tibial arteries at the lower border of popliteus.

The anterior tibial artery courses through the crural IO membrane to enter the anterior compartment of the leg where it descends to the ankle, where it becomes the dorsalis pedis artery. The anterior tibial artery courses between TA and EDL in the superior third of the leg, between TA and EHL in the middle third, deep to the tendon of EHL just proximal to the ankle and between the tendons of EHL and EDL at the level of the ankle. The branches of the anterior tibial artery include:

1. Posterior recurrent tibial artery, posterior to IO membrane
2. Anterior recurrent tibial artery, which joins the circumpatellar network
3. Muscular branches to TA, EDL, EHL, and peroneus tertius
4. Anterior medial malleolar artery
5. Anterior lateral malleolar artery
The anterior leg muscles are supplied by muscular branches of the anterior tibial artery. The anterior medial malleolar artery anastomoses with branches of the posterior tibial and medial plantar arteries. The anterior lateral malleolar artery anastomoses with the perforating branch of the peroneal and lateral tarsal arteries. The dorsalis pedis artery, the second largest source supplying the foot, continues to the 1st intermetatarsal space, where it courses as the deep plantar branch to join the plantar arch (Fig. 1.19). The branches of the dorsalis pedis artery include:

1. lateral tarsal artery; supplying EDB
2. medial tarsal artery
3. arcuate artery; yielding the 2nd, 3rd, and 4th dorsal metatarsal arteries
4. 1st dorsal metatarsal artery
5. deep plantar perforating branch

The dorsal metatarsal arteries lie in the corresponding intermetatarsal spaces, deep to the extensor tendons and dorsal to the dorsal IO muscles. Except the first dorsal metatarsal artery, which yields the deep plantar perforating artery, the metatarsal arteries yield posterior and anterior perforating branches at the level of the metatarsal base and MTPJ, respectively. The arteries continue distally as common digital arteries, which divide into proper dorsal digital arteries that are of smaller diameter than the plantar digital arteries.

The posterior tibial artery, the largest source supplying the foot, is a terminal branch of the popliteal artery and courses through the leg to the third canal of the flexor retinaculum, then divides into medial and lateral plantar arteries deep to abductor hallucis in the calcaneal canals (Fig. 1.20). The branches of the posterior tibial artery include:

1. circumflex fibular artery, which supplies soleus
2. peroneal artery, which supplies soleus, TP, FHL, PL, PB, and the fibula; and the perforating peroneal branch (third largest source supplying the foot) that pierces the IO membrane proximal to the ankle to join with branches of the anterior tibial artery
3. nutrient artery to tibia, the largest nutrient artery in the body
4. muscular branches to soleus, TP, FHL, FDL
5. communicating artery that anastomoses with peroneal artery
6. medial malleolar branches
7. medial calcanean branches, which supply tendoAchillis and medial heel
8. medial plantar artery, medial to the medial plantar nerve
9. lateral plantar artery, which becomes the plantar arch and supplies all of the muscles of the sole, except abductor hallucis, FDB, and 1st dorsal IO muscle.
The plantar arch courses lateral to medial toward the first intermetatarsal space, where it anastomoses with the deep plantar perforating branch of the dorsalis pedis artery. The plantar arch separates the 3rd and 4th muscle layers, and yields anterior and posterior perforating arteries that anastomoses with corresponding perforators from the dorsum.

The plantar arch yields 4 plantar metatarsal arteries, the first of which consists of the union of the lateral plantar and deep plantar branches. The plantar metatarsal arteries become common and then proper digital arteries to the corresponding toes. The plantar digital arteries are larger than the dorsal digital arteries. In the hallux, the lateral plantar digital artery is the largest, while in the lesser toes the medial plantar digital arteries are largest. In the hallux, the dorsal digital arteries extend to the toe tip, as do the plantar digital arteries, the dorsal and plantar hallucial digital arteries supplying the hallux equally distal to the interphalangeal joint. In the lesser toes, dorsal digital arteries extend to the level of the proximal ITPJ, while plantar digital arteries extend to the toe tip and then retrograde to supply the dorsal aspect of the toe, including the nail bed (Fig. 1.21).

VENOUS SYSTEM

The dorsal venous system of the foot and ankle consists of superficial and deep networks. The deep dorsal venous plexus converges to form the medial marginal vein. The superficial dorsal venous plexus is immediately subcutaneous, and contains the dorsal venous arch. The dorsal veins drain into the greater and lesser saphenous veins.

On the plantar aspect, a superficial venous plexus drains into the deep venous plexus, which ultimately converges into the medial and lateral plantar veins, and communicates with the dorsal system via perforating veins.

LYMPHATIC SYSTEM

Superficial lymphatics drain the skin of the toes, sole and heel, forming a medial system that drains into the inguinal lymph nodes and a lateral (rays 3-5) system that drains into the popliteal lymph nodes. The deep lymphatic system forms collecting ducts located dorsally, laterally (peroneal), and plantarly, and drain into major lymphatics corresponding to the adjacent anterior tibial, peroneal, and posterior tibial vessels. The deep system drains primarily into the popliteal lymph nodes.

CUTANEOUS ANATOMY

The skin consists of the epidermis and dermis (Fig. 1.22). The dermis consists of both reticular and papillary layers, and contains microcirculatory elements (arterioles, capillaries, venues, glom, and lymphatics), nerves and the annexed. Skin annexed include echini sweat glands and ducts, hair follicles and arrestor pile muscles, sebaceous glands at the base of the
hair follicle (pilosebaceous gland), and the toenails and perionychium. Near the nail bed, arterioles shunt directly to venules via the Hoyer-Susquet canal, to effect the glomus body important in temperature regulation. Eccrine glands are present on all pedal skin surfaces, and are innervated by sympathetic nerves. Pilosebaceous glands are only present on dorsal skin. Deep in the dermis, near the subcutaneous fat-superficial fascia junction, lie the Pacinian (Pacini-Vater) corpuscles important in touch-pressure sensation. The epidermis serves as a barrier and contains five strata: basale, spinosum, granulosum, lucidum, and corneum. Melanocytes with dendritic processes exist amongst the living cells of the stratum basale, and are responsible for melanin production which serves to protect underlying living cells from the mutagenic effects of UV radiation. Langerhans immune cells, much like macrophages, as well as Merkel's sensory cells also exist in the epidermis.

Relaxed Skin Tension Lines (RSTL) (Fig. 1.23)

The skin's intrinsic tension is oriented such that maximum tension is directed parallel to the long axis of the extremity. Intrinsic skin tension is generated by the forces of underlying bone and soft tissue prominence, as well as joint motion and extrinsic forces upon the skin. RSTL are oriented perpendicular to the long axis of the leg and foot, and can be clinically identified with the pinch test. As a rule, elective skin incisions should be made parallel to the RSTL, as long as the exposure allows access to the underlying target structures and does not unduly violate vital structures (vessel, nerve, tendon).
Overview - Bony Anatomy

A - Tibia
B - Fibula
C - Talus
D - Calcaneus
E - Navicular
F - Cuboid
G, H, I - Cuneiforms
J, K, L, M, N - Metatarsals
O, P, R, S - Phalanges

“Bony” Problems in Diabetics

AKN bony prominence can cause problems:
- Button
- Bass J-P
- Rocker Deformity
- Platform - acquired
- Calveus foot

Bones rotate about joints in 1 of 3 planes of motion
- Sagittal (dorsiflex/plantarflex)
- Frontal (evert/invert)
- Transverse (abduction/adduction)
Motion may occur in multiple planes

Overview - Joints

Overview - Skin

Epidemis
- external barrier
Dermis
- nourishes epidermis
- resist mech. forces
Dermoepidermal junction
- undulating
capillary plexus

Demonstration of Supination & Pronation of a Right Foot
Module 2: Equipment for Basic Foot-care Clinic

Comfortable reception area with ample seating for patients.

Window to receive patients. There should be a door to separate treatment area from reception area.

Treatment room with chair. The chair, preferably should be adjustable to access the plantar aspect of the foot without the practitioner contorting their body. This however is not a necessity. A stool for sitting is acceptable if the treatment chair is stationary.

Work Desk: This is where supplies are kept. This area should have a sink as well as cabinets and drawers. Items pictured from left to right: Ethel chloride (cold topical anesthetic), Alcohol, Peroxide, Container with wooden probes, Container with gauze sponges, Procedure gloves, Foot model, Antiseptic wipes, Sharps disposal container, Hand antibiotic gel.

Diagnostic Instruments: Reflex hammer, measuring tape, monofilament wire, tuning fork (used to test vibratory sensation and temperature sensation).

Treatment Instruments: Bandage scissors, Heavy duty, double action nail nippers, Small tissue nippers, #15 surgical blade on a #3 blade handle, Forceps, Dermal Curette.
Autoclave: Used to sterilize instruments

Simple Dressing Supplies: Self-adherent tape, elastic bandage, Ace compression bandage, gauze roll.

Simple Wound Care:
- Gauze - Used either wet or dry to cover wound
- Mupirocin Ointment - Antibiotic gel
- Silvadene Cream - Antibiotic cream
- Cotton Tipped Probes
- Silver Nitrate - Cauterize wound base, hemostasis
- Saline - Cleanse wound or moisten gauze for dressing

Storage Area:
Ample in size to store all reserve supplies.
Module 3: Foot Screening

The following section provides tools to help you and your staff incorporate diabetes foot exams into clinical practice and improve patient outcomes. Research indicates that when tools like these are used by providers, more examinations of lower extremities are performed, patients at risk for amputation are identified, and more patients are referred for podiatric care. Using these tools also will help providers increase the proportion of persons with diabetes who have at least an annual foot examination and reducing the frequency of foot ulcers and lower extremity amputations in persons with diabetes.

Current clinical recommendations call for a comprehensive foot examination at least once a year for all people with diabetes to identify high risk foot conditions. People with one or more high risk foot conditions should be evaluated more frequently for the development of additional risk factors. People with neuropathy should have a visual inspection of their feet at every contact with a health care provider. In communities where the prevalence and incidence of diabetes foot problems are high, providers may determine that inspecting feet at every visit – for both low and high risk patients – is warranted. The following tools will help you incorporate diabetes foot exams into your practice.

Flow Chart for Diabetes Foot Exams*

## Diabetes Foot Exam Procedures

<table>
<thead>
<tr>
<th>Category of Patient</th>
<th>Recommended Procedure</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persons with:</td>
<td>Comprehensive foot exam to identify high risk foot conditions. A physician or other trained health care provider should:</td>
<td>Annually or when a new abnormality is noted</td>
</tr>
<tr>
<td>• Type 1 diabetes</td>
<td>• Assess skin, hair and nails, musculoskeletal structure, vascular status, and protective sensation.</td>
<td></td>
</tr>
<tr>
<td>• Type 2 diabetes</td>
<td>• Inspect footwear for blood or other discharge, abnormal wear patterns, foreign objects, proper fit, appropriate material, and foot protection.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Educate about self-care of the feet.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Educate about the importance of blood glucose monitoring including the use of the Hemoglobin A1c test.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Reassess metabolic control.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Management plan.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• The subsequent foot care management plan depends on risk category, foot status, and metabolic control.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• High risk patients should be referred to a health care provider with training in foot care.</td>
<td></td>
</tr>
<tr>
<td>Persons at:</td>
<td>Visual foot inspection to identify foot problems. A physician or other trained staff should perform the foot inspection.</td>
<td>At every visit</td>
</tr>
<tr>
<td>• High risk</td>
<td></td>
<td>As warranted</td>
</tr>
<tr>
<td>• Low risk</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Refer to chart on page 13 for definitions of risk)
Foot Exam Instructions

Visual Foot Inspection

Objectives
- Quickly identify an obvious foot problem.
- Document foot inspection findings.
- Determine the need for a comprehensive foot exam.
- Schedule follow-up care and referrals.

Instructions
A physician, nurse, or other trained staff may complete this inspection.

1. Inspect the foot between the toes and from toe to heel.
   Examine the skin for injury, calluses, blisters, fissure, ulcers, or any unusual condition.
2. Look for thin, fragile, shiny, and hairless skin—all signs of decreased vascular supply.
3. Feel the feet for excessive warmth and dryness.
4. Remove any nail polish. Inspect nails for thickening, ingrown corners, length, and fungal infection.
5. Inspect socks or hose for blood or other discharge.
6. Examine footwear for torn linings, foreign objects, breathable materials, abnormal wear patterns, and proper fit.
7. If any new foot abnormality is found, the patient should be scheduled immediately for a comprehensive foot examination.
8. Document findings in the medical record.

Frequency of Inspection Current clinical recommendations call for visual inspection of the feet:
- At every visit for people who have neuropathy.
- At least twice a year for people with one or more high risk* foot conditions to screen for the development of additional risk factors.
- At least annually, or more often if warranted, for low risk feet.*

Clinical Documentation
The following should be documented in the medical record:
- Results of the annual comprehensive foot examination including risk assessment.
- Results of the visual foot inspection.
- Occurrence of patient education.

Measures
Short-term Impact: A successful program will show an increase in the percentage of the population with diabetes for whom the following is documented:
- A comprehensive foot exam and risk assessment in the past year.*
- A visual foot inspection at each routine visit in the past year.
- Foot care education in the past year.

A survey could be conducted to ask patients to report when they last had a sensory test, foot inspection, and self-care education in the past year.

Intermediate-term Impact: A successful program will show a decrease in the incidence of hospital admissions or emergency room visits for lower extremity infections, osteomyelitis, and ulcerations.

Long-term Outcomes: A successful program will show a decrease in the incidence of distal and proximal lower extremity amputations.
In populations where the prevalence and incidence of diabetes foot problems are high, providers may determine that inspection of the feet at every visit — for both low and high risk patients — is warranted.

Annual Comprehensive Diabetes Foot Exam

Objectives: Collect the necessary data to assess feet for risk of complications. Completing the comprehensive annual foot exam will enable you to:

• Determine the patient’s risk status.
• Document foot exam findings.
• Determine the need for therapeutic foot wear.
• Determine the need for referral to foot care specialists.
• Schedule self-management education.
• Develop an appropriate management plan.
• Schedule follow-up care and referrals.

Instructions

Use copies of the annual comprehensive foot exam form to document findings, or incorporate the assessment questions and foot exam into an already existing overall diabetes care plan. A physician or other trained health care provider should conduct the foot exam. Prepare the patient for examination by removing shoes and socks/hose.

I. Presence of Diabetes Complications Complete the questions as directed.

Question 1: Does the patient have any history of the macro- and micro-vascular complications of diabetes or a previous amputation?

Patients who have been diagnosed with peripheral neuropathy, peripheral vascular disease or cardiovascular disease are likely to have had diabetes for several years and to be at risk for diabetes foot problems. A positive history of a previous amputation places the patient permanently in the high risk category. Specify the type and date of amputation(s).

Question 2: Does the patient have a foot ulcer now or a history of foot ulcer?

A positive history of a foot ulcer places the patient permanently in the high risk category. This person always has an increased risk for developing another foot ulcer, progressive deformity of the foot, and ultimately, lower limb amputation.

II. Current History Complete the questions as directed.

Question 1: Is there pain in the calf muscles when walking—i.e., pain occurring in the calf or thigh when walking less than one block that is relieved by rest?

This question is to determine whether the patient experiences intermittent claudication when walking. This pain is an indication of peripheral vascular disease or impaired circulation.

Question 2: Has the patient noticed any changes in the feet since the last foot exam?

Patients may notice changes in skin and nail condition or sensory perception if they are performing self-tests with a monofilament.

• Collect the necessary data to assess feet for risk of complications.
• Determine the patient’s risk status.
• Document foot exam findings.
• Determine the need for therapeutic foot wear.
• Determine the need for referral to foot care specialists.
• Schedule self-management education.
• Develop an appropriate management plan.
• Schedule follow-up care and referrals.

Questions 3 and 4: Has the patient experienced any shoe problems? Has the patient noticed any blood or other discharge in socks or hose?

New shoes can cause unexpected pressure and irritate underlying skin. Blood or other discharge from a foot wound can be the first indication of a
severe foot problem.

**Question 5: What is the patient’s smoking history?**

Cigarette smoking is a major risk factor for microvascular and macrovascular disease and is likely to contribute to diabetes foot disease.

**Question 6: What is the patient’s most recent hemoglobin A1c test result?**

Elevated hemoglobin A1c values are independently associated with a twofold risk of amputation.

**III. Foot Exam Complete the questions or fill in the items as directed.**

**Item**

1. **Condition of the skin, hair and toenails.**

   Questions: Is the skin thin, fragile, shiny and hairless? Are the nails thick, too long, ingrown, or infected with fungal disease?

   - Examine each foot between the toes and from toe to heel. Record any problems by drawing or labeling the condition on the foot diagram. Skin that is thin, fragile, shiny, and hairless is an indication of decreased vascular supply. Loss of sweating function may cause cracking of the skin and fissures that can become infected.
   - Remove any nail polish. Check toenails to see if they are ingrown, deformed, or fungal. Thick nails may indicate vascular or fungal disease. If severe nail or dry skin problems are present, refer the patient to a podiatrist or a nurse foot care specialist.
   - Measure, draw in, and label the patient’s skin condition.
   - Measure and draw on the form any corns, calluses, pre-ulcerative lesions (a closed lesion, such as a blister or hematoma), or open ulcers.
   - Use the appropriate symbol to indicate what type of lesion is present—i.e., callus, ulcer, redness, warmth, maceration, pre-ulcerative lesion, fissure, swelling or dryness.

   Maceration is present if the tissue is friable, moist, and soft.

   - Label areas that are significantly dry, red, or warm (warmer than other parts of the foot or the opposite foot).

   **Musculoskeletal Deformities**

   - Foot deformities may be the result of diabetic motor neuropathy. The function of intrinsic muscles is lost, causing the toe digits to buckle as other muscles become imbalanced.
Footwear Assessment

Question 1. Does the patient wear appropriate shoes?
Question 2. Does the patient need inserts?
Question 3. Should corrective footwear be prescribed?

Check inside shoes for foreign objects, torn lining, and proper cushioning. Improper or poorly fitting shoes are major contributors to diabetes foot ulcerations. Counsel patients about appropriate footwear. All patients with diabetes need to pay special attention to the fit and style of their shoes and should avoid pointed-toe and open-toe shoes, high heels, thongs and sandals. Assess the material and construction of footwear.

Unbreathable and inelastic materials such as plastic should be avoided. Recommend use of materials such as canvas, leather, suede, and other materials that are breathable and/or elastic. Footwear should be adjustable with laces, Velcro, or buckles. Record the results of your footwear assessment. Properly fitted athletic or walking shoes are recommended for daily wear. If off-the-shelf shoes are used, make sure that there is room to accommodate any deformities. High risk patients may require depth-inlay shoes or custom-molded inserts (orthoses), depending on the degree of foot deformity and history of ulceration.

Education

Question 1: Has the patient had prior foot care and other relevant diabetes education?
Question 2: Can the patient demonstrate appropriate foot care?

Indicate whether the patient has received prior education by checking yes or no in the blank. Patient education about foot care and other aspects of self-care is an essential component of preventive diabetes care. Observe whether the patient can demonstrate appropriate self-care of the feet. Refer for smoking cessation counseling if necessary. Determine whether the patient understands the need for, and results of, hemoglobin A1c tests.
Muscle wasting occurs. The plantar fat pad becomes displaced and the metatarsal heads become more prominent. Limited joint mobility occurs and contributes to the potential for toe and foot injury. If Charcot foot is present, there are severe bone and joint changes and the foot is swollen and warm to the touch.

**Pedal Pulses**
Check the pedal pulses (posterior tibial and dorsalis pedis) in both feet and note whether pulses are present or absent.

**Sensory Exam**
The sensory testing device supplied in this kit is a 5.07 (10-gram) Semmes-Weinstein nylon monofilament mounted on a holder that has been standardized to deliver a 10-gram force when properly applied. Research has shown that a person who can feel the 10-gram filament in the selected sites is at reduced risk for developing ulcers. Because sensory deficits appear first in the most distal portions of the foot and progress proximally in a “stocking” distribution, the toes are the first areas to lose protective sensation.

- The sensory exam should be done in a quiet and relaxed setting. The patient must not watch while the examiner applies the filament.
- Test the monofilament on the patient’s hand so he/she knows what to anticipate.
- The five sites to be tested are indicated on the examination form.
- Apply the monofilament perpendicular to the skin’s surface (see diagram A below).
- Apply sufficient force to cause the filament to bend or buckle, using a smooth, not a jabbing motion (see diagram B below).
- The total duration of the approach, skin contact, and departure of the filament at each site should be approximately 1 to 2 seconds.
- Apply the filament along the perimeter and NOT ON an ulcer site, callus, scar or necrotic tissue. Do not allow the filament to slide across the skin or make repetitive contact at the test site.
- Press the filament to the skin such that it buckles at one of two times as you say “time one” or “time two.” Have patients identify at which time they were touched. Randomize the sequence of applying the filament throughout the examination.
Foot ulceration is the most common single precursor to lower extremity amputations among persons with diabetes. Treatment of infected foot wounds comprises up to one quarter of all diabetic hospital admissions in the US and Britain, making this the most common reason for diabetes related hospitalization in these countries. Risk factors identified include peripheral neuropathy, vascular disease, limited joint mobility, foot deformities, abnormal foot pressures, minor trauma, a history of ulceration or amputation, and impaired visual acuity. These and other putative causative factors are shown in Figure 1.

Peripheral sensory neuropathy in the face of unperceived trauma is the primary factor leading to diabetic foot ulcerations. Approximately 45% to 60% of all diabetic ulcerations are purely neuropathic, while up to 45% have neuropathic and ischemic components (24, 51). According to an important prospective multicenter study, sensory neuropathy was the most frequent component in the causal sequence to ulceration in diabetic patients.

Other forms of neuropathy may also play a role in foot ulceration. Motor neuropathy resulting in anterior crural muscle atrophy or intrinsic muscle wasting can lead to foot deformities such as foot drop, equinus, hammertoe, and prominent plantar metatarsal heads. Ankle equinus with restricted dorsiflexory range of motion is fairly common in patients with diabetic neuropathy and can be a consequence of anterior crural muscle atrophy.
Risk Factors for Amputation

- Neurotherapy
- Peripheral arterial disease (PAD)
- Infection
- History of prior foot ulcer or amputation
- Structural foot deformity
- Trauma
- Charcot foot
- Impaired vision
- Poor glycemic control
- Older age
- Male Sex
- Ethnicity (greatest rates in blacks & Hispanics)

Figure 3: The risk factors for amputation are multifactorial and similar to those for ulceration.

Peripheral Arterial Disease Causes

- Atherosclerosis
- Inflamed Blood Vessels
- Injury to Limbs
- Unusual Anatomy
- Radiation Exposure

Peripheral Arterial Disease Risk Factors

- Smoking
- Diabetes
- Obesity (BMI > 30)
- Hypertension (>140/90)
- Hypercholesterolemia
- Advanced Age
- Family History (PAD or CAD)
- Increased levels of Homocysteine

Ankle Brachial Index

- Compares blood pressure in ankle with blood pressure in arm

<table>
<thead>
<tr>
<th>ABI Value</th>
<th>Interpretation</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above 1.1</td>
<td>Abnormal Vessel Function</td>
<td>Vascular Referred</td>
</tr>
<tr>
<td>1.0-1.1</td>
<td>Normal Range</td>
<td>None</td>
</tr>
<tr>
<td>0.9-1.0</td>
<td>Acceptable</td>
<td>Management</td>
</tr>
<tr>
<td>0.8-0.9</td>
<td>Moderate Risk</td>
<td>Vascular Referred</td>
</tr>
<tr>
<td>Under 0.5</td>
<td>Severe Disease</td>
<td>Vascular Referred</td>
</tr>
</tbody>
</table>

Peripheral Arterial Disease Symptoms

- Intermittent Claudication
- Leg Numbness
- Foot pain at night
- Non-Healing Ulcers
- Pigmentary Changes in Skin
- Pedal Hair Loss
- Skin or Nail Lesion
- No Pulse or Weak Pulse
- Erectile Dysfunction

DN Definition and Classification

- Diabetic neuropathy is not a single entity but a number of different syndromes, ranging from asymptomatic to clinical manifestations depending on the classes of the nerve fibers involved.
- San Antonio Convention: 3 groups of disturbances
  - Autonomic neuropathy
  - Sensory neuropathy
  - Motor neuropathy
- Small-fiber neuropathies (DSN)

Small-Fiber Neuropathies (DSN)

- Clinical Manifestations
  - Tolerance, abnormal sweating, and atrophy
  - Altered thermal sensation
  - Decreased function and reflexes: extensor plantar response

- Treatment of Painful Diabetic Neuropathy

- American Academy of Neurology 2011 Guidelines
- Effective
- Probable Effectiveness
- Painful neuropathy: 100 to 300 mg daily
- Acetaminophen, 1 to 3 g daily
- Tramadol, 50 to 300 mg daily
- Ketamine, 10 to 90 mg daily
- Gabapentin, 300 to 3,600 mg daily
- Pregabalin, 300 to 600 mg daily
- Tramadol-IR, 50 to 300 mg daily
- Oxycodone, 5 to 90 mg daily
- Baclofen, 25 to 100 mg daily
- Methylphenidate, 20 to 100 mg daily
- Mirtazapine, 9 tablets daily
- Amitriptyline, 50 to 150 mg daily
- Venlafaxine, 20 to 200 mg daily
- Citalopram, 20 to 100 mg daily
- Vortioxetine, 20 to 30 mg daily
- Duloxetine, 0 to 50 mg daily
- Tramadol, 0 to 100 mg daily
- Olanzapine, 10 to 20 mg daily
- Varenicline, 0 to 2 mg daily
- OxyContin, 5 to 10 mg daily
- Zolpidem, 10 to 30 mg daily
- Zolfit, 0 to 2 mg daily
- Selegiline, 2 to 15 mg daily
- Metoclopramide, 0 to 30 mg daily
- Amantadine, 100 to 400 mg daily
- Perphenazine, 10 to 50 mg daily
- Haloperidol, 1 to 5 mg daily
- Clozapine, 200 to 600 mg daily
- Quetiapine, 25 to 200 mg daily
- Citalopram, 20 to 100 mg daily
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- Cita...
PATHWAY #1

DIABETIC FOOT DISORDERS

**Significant History**
- Duration of Diabetes
- Previous ulceration, infection, Charcot, amputation
- Pain / sensation
- PAD or prior revascularization

**Significant Findings**

**Dermatologic**
- Erythema
- Warmth
- Cellulitis
- Ulcer
- Trophic changes

**Musculoskeletal**
- Swelling
- Deformity
- Joint mobility
- Joint dislocation

**Neurologic**
- Degree of neuropathy assessed by Semmes-Weinstein monofilaments, vibratory, proprioception

**Vascular**
- Absent or asymmetric pedal pulses
- Dependent rubor
- Gangrene

**Laboratory Tests**
- CBC with differential
- ESR, CRP
- Blood glucose
- Hb A1c

**Diagnostic Imaging**
- Plain radiographs
- Imaging studies
- CT
- MRI
- Bone scan

**Noninvasive Vascular Studies**
- Arterial Doppler: ABI, toe pressures, waveforms
- Transcutaneous oxygen tensions

**Radiographic Findings**
- Bone density
- Joints/bones involved
- Osteolysis
- Deformity
- Fractures
- Dislocation
- Soft tissue edema
- Vascular calcifications

**Infection**
- Proceed to Pathway #4
  - Cellulitis
  - Abscess
  - Osteomyelitis

**Ischemia**
- Proceed to Pathway #3

**Ulceration +/- Deformity**
- Proceed to Pathway #5

**Charcot**
- Proceed to Pathway #2

**Charcot Treatment**
PATHWAY #2

DIABETIC PERIPHERAL ARTERIAL DISEASE

SIGNIFICANT HISTORY

- Rest Pain
- Previous ulceration or infection
- Claudication
- Smoker
- Metabolic syndrome

SIGNIFICANT FINDINGS

Examination

- Dermatologic: trophic changes, ulcer, gangrene
- Vascular: Poor or non-palpable pedal pulses

Clinical Maneuvers

- Elevation pallor
- Dependent rubor

DIABETES PAD

Noninvasive Vascular Studies

- Arterial Doppler: waveforms, ABI's & toe pressures,
- Transcutaneous oxygen tensions

INVASIVE VASCULAR STUDIES

- Angioplasty
- Endovascular
- Open bypass grafting

Medical Management

- Antilipemic agents (statins)
- Antiplatelet therapy
- Vasodilators

Follow-Up

- Patient education
- Smoking cessation
- Protective shoes
- Periodic foot care
- Reconstructive foot surgery as needed

Gangrene or extensive tissue loss in face of unreconstructable PAD

Consider amputation
PATHWAY #3

DIABETIC FOOT ULCERATION

SIGNIFICANT HISTORY
- Duration of ulcer
- Previous ulceration
- Pain / sensation
- Vascular history
- *Additional Diagnostic Procedures as indicated

Refer to Pathway #1

General Foot Exam
- Vascular
- Neurologic
- Structural deformity
- Dermatologic

Vascular
- Palpate pedal pulses
- Noninvasive vascular studies

Diagnostic Imaging
- Plain radiographs
- Imaging studies
- CT
- MRI
- Bone scan
- Ultrasound

Ulcner Examination
- Classification
- Size, depth
- Location
- Deformity
- Extent of necrosis
- Probe to bone

Presence of GANGRENE
- PAD

Peripheral vascular Consultation
- INFECTION

ULCERATION +/- Deformity

Proceed to Pathway #2
- Tx PAD

RE-EVALUATE

Initial Ulcer Treatment

WOUND CARE
- Debridement
- Sharp
- Enzymatic
- Hydrotherapy
- Ultrasound
- Moisture balance / dressings
- Advanced wound management
- Growth factors
- Bioengineered tissues
- HBO
- Negative pressure (NPWT)

OFF-LOADING
- Bed rest
- Surgical shoe / healing sandal
- Bracing
- Total contact casts
- Wheelchair
- Crutches

OSTEOMYELITIS

WOUND HEALED

Surgical Management
- Debridement
- Soft tissue
- Bone
- Exostectomy
- Correct deformity
- Plastic surgery

WOUND FAILS TO HEAL

- Re-evaluate vascularity
- Re-evaluates for infection / osteomyelitis
- Biopsy to assess for malignancy

If ulcer recurs, treat appropriately,
- Re-evaluate vascularity
- Rule out osteomyelitis

Long-term management of healed ulcer
- Patient education
- Frequent re-evaluation
- Protective shoes, etc see below:
- Bracing
- Extra depth shoes
- Custom molded shoes
- Multiple density insoles
- Orthoses

OSTEOMYELITIS

Proceed to Pathway #4
**Module 4**

**PATHWAY #4**

**DIABETIC FOOT INFECTION**

**SIGNIFICANT HISTORY / FINDINGS**
- Trauma (injury), puncture wound, foreign body
- Ulceration or gangrene
- Swelling, drainage, odor
- Systemic signs: fever, chills, malaise
- Diabetes duration / control

**NON-LIMB-THREATENING INFECTION**
- $\leq 2\text{cm}$ cellulitis
- Superficial ulcer
- Does NOT probe to bone
- Limited edema, inflammation
- No bone / joint involvement
- No systemic toxicity
- No significant ischemia

**Diagnostics**
- Oral temperature
- Deep wound culture from bone of ulcer / wound tissue specimen if possible
- Diagnostic imaging
  - X-rays, CT, bone scan
- Vascular evaluation
- Metabolic testing
  - CBC with differential blood culture
  - ESR, CRP
  - Blood glucose
  - Renal metabolic profile

**LIMB-THREATENING INFECTION**
- $>2\text{cm}$ Cellulitis
- Edema, pain, lymphangitis
- Drainage, odor
- Probe wound for extensions
- Systemic signs: hypotension, cardiac arrhythmia (systemic toxicity)
- Ischemic changes

**Outpatient Management**

**TREATMENT**
- Surgical debridement of callus & ALL necrotic tissue
- Wound care - see Pathway #3
- Empiric antibiotic coverage followed by culture directed antibiotics
- Close monitoring of progress
- Hospital admission if infection progresses or wound / foot deteriorates

**Hospital Admission**

**TREATMENT**
- Surgical debridement of ALL necrotic tissue
- Exploration & drainage of abscess
- Surgical resection of osteomyelitis
- Open wound management
- Empiric antibiotics modified by culture directed antibiotics
- Advanced wound management
  - Negative pressure (NPWT)
  - See Pathway #3
  - Repeated wound debridement PRN
  - Revascularization, as needed
  - Foot-sparing reconstructive procedures
  - Definitive amputation, if necessary

**CONSULTATIONS as Necessary**
- Endocrinology
- Vascular surgery
- Podiatric surgery
- Infectious disease
- Nephrology
- Cardiology
- General surgery

**INFECTION RESOLVES**

**Non-Infected Ulcer**
- Proceed to Pathway #3

**Outpatient Care**
- Antibiotics
- Home wound care
- Off-loading
- Office podiatric care

**Open Wound / Ulcer or Healed Foot**
- Proceed to Pathway #3
PATHWAY #5

CHARCOT FOOT

SIGNIFICANT HISTORY
- Onset of morphologic changes
- Progressive / static
- Erythema
- Swelling
- Tumor: type, when, repetitive
- LOPS: + pain
- Previous ulcer &/or Charcot
- Long-standing diabetes

SIGNIFICANT FINDINGS

- Dermatologic
  - Erythema
  - Wound
  - Cellulitis
  - Serous
  - +/- Ulcer

- Musculoskeletal
  - Swelling
  - Deformity
  - Joint dislocation
  - Equinus

- Neurologic
  - LOPS
  - Autonomic neuropathy
  - Motor neuropathy
  - Absent DFNs

- Vascular
  - Palpable pedal pulses
  - Swelling

Diagnostic Imaging
- Plain radiographs
- Imaging studies
- CT
- MRI

Radiographic Findings
- Joints/bones involved
- Osteolysis
- Fractures
- Bone density
- Soft tissue edema
- Vascular calcifications
- Deformity

* Additional Diagnostic Procedures as indicated

DIAGNOSIS

TREATMENT OF CHRONIC CHARCOT

Foot Unstable
- Bracing
- Extra depth shoes
- Custom molded shoes
- Multiple density insoles
- Orthoses

If ulcer recurs, treat appropriately, see Pathway #3

Foot Stable
- Supportive measures
- Therapeutic footwear
- Patient education
- Periodic evaluation to prevent recurrence

Convert to Stable Foot

Remains unstable
Chronic ulceration
Chronic osteomyelitis
Consider amputation

Once quiescent, treat as chronic

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PATHWAY #6

SURGERY OF THE DIABETIC FOOT

**SIGNIFICANT HISTORY/FINDINGS**
- Ulceration/open wound
- Progressive/static
- Infection
- Charcot +/− deformity
- Orthopedic deformity

Critical limb ischemia (PAD)
* PAD if present, already addressed

Refer to previous Pathway

**NO WOUND**
- Presence of foot deformity
  - Intact protective sensation
  - ELECTIVE SURGERY
    - TO TREAT A PAINFUL FOOT DEFORMITY
    - Structural correction of musculoskeletal deformity

**WOUND**
- Presence of ulcer, open wound, necrotic tissue or acute infection/abscess
  - CURATIVE SURGERY
    - TO ASSIST IN HEALING AN OPEN WOUND
    - Repair deformity
    - Resect infected bone or joint
    - Plastic surgical reconstructive flaps or wound closure
  - PREHOSPITAL SURGERY
    - TO REDUCE RISK OF ULCERATION OR AVOID REULCERATION
    - Structural correction of musculoskeletal deformity
  - EMERGENT SURGERY
    - TO ARREST OR LIMIT PROGRESSION OF ACUTE INFECTION
    - Ablative surgery aimed at elimination of infected and necrotic tissues

Proceed to Pathway #2
Module 5: Skin And Nail Pathology

Clinical Exam of the Foot and Ankle

- **Skin**
  - Color, Moisture, Temperature
  - Texture, Mobility, Turgor
- **Anatomy**
  - Entire lower leg
  - Foot and Ankle
- **Bony Prominences**
  - Talus, Calcaneous
  - 1st and 5th metatarsal
- **Toes**
  - Web spaces
  - Nail unit
  - Bone prominences
- **Sole of the Foot**

Calluses

- **Callus**
  - Diffuse thickening of the outermost layer of the skin, the stratum corneum, in response to the repeated friction or pressure
- **Calluses of the foot: terminology**
  - Heloma Dura: *Cora* dorsal aspect of the toe
  - Heloma Molle: Callus in the web space of a toe
- **Aquired Keratoderma: Conical horn like projection**
  - Areas of pressure
  - Infections
  - Medications
  - Etc.

Aquired Keratoderma

- **Aquired Keratoderma**

Calluses: Treatment

- **Manual Debridement**
- **Keratolytics**
- **Off weighting techniques**
- **Shoe gear modification**

Porokeratosis

- **Porokeratosis Punctata**
- **Porokeratosis Plantaris Discreta**
  - **Adults**
  - **Cronial keratotic depressions**
- **Punctate**
  - Generally occurs on the creases of Palms
**Module 5**

**Pyogenic Granuloma**
- Friable, 5-10 mm papule
- Occurs after trauma: Onychocryptosis
- Can be induced by retinoids, Protease Inhibitors
- Biopsy: Excessive granulation tissue
- Treatment: Surgical removal, Electrodesiccation of base

**Fungal Infections**
- **Superficial (epidermis)**
  - Tinea pedis, cruris, ungulium, capitis
  - Tinea versicolor
  - Candidiasis
- **Dermatophytoses**
  - Fungi parasitize keratin(stratum corneum)
  - Nail or hair
- **Candidiasis**
  - Yeast infection of mucous surfaces and moist skin
- **Tinea Vescicular**
  - Yeast infection skin surface

**Tinea Pedis**
- **Fungal Infection of the foot**
  - Very common
  - Disease of shoe-wearing people
  - Public facilities
- **Clinical patterns**
  - Interdigital
  - Diffuse Plantar
  - Vesiculobullous
- **Complications**
  - Cellulitis: web space superinfection gram positive
  - Gram negative web space infection: Pseudomonas
  - Tinea corporis
  - Erythrasma: Corynebacterium minutissimum

**Tinea Pedis: Clinical Patterns**
- **Interdigital**
- **Vesiculobullous**
- **Diffuse plantar**
- **Tinea pedis**

**Tinea Pedis: Clinical Patterns**

**Super Infection**
Tinea Pedis: Treatment

- **Foot hygiene**
  - Dry feet after bathing
  - Foot powder
  - Synthetic socks, wicking socks
  - Change shoes/socks at least daily

- **Topical antifungals**
  - Imidazole (fungistatic): miconazole, clotrimazole, ketoconazole
  - Allylamines (fungicidal): terbinafine, naftifine, butenafine

- **Oral antifungals**

Oral Antifungals—Rarely needed

- **Terbinafine**: 250 mg QD for 12 weeks/90 days
  - Drug interactions: warfarin
  - Risks: abnormal LFT’s 3%, loss of taste 2.7% (reversible)

- **Itraconazole**: 200 mg BID with food, 1 week per month, x3
  - Drug interactions: warfarin, cyclosporine, lovastatin, phenytoin, viagra, xanax, plendil, versed
  - Risks: abnormal LFT’s, drug interactions

Pitted Keratolysis

- Superficial infection by corynebacterium
- Prolonged wetness
- Punched out loss of stratum corneum
- Feet stink/smelly

Pitted Keratolysis

- **Treatment**
  - Dry feet
  - Change socks/shoes
  - Topical antiperspirants
  - Topical antibiotics:
  - Erythromycin or clindamycin solution

Plantar Warts

- **Human papilloma virus (HPV)**
  - HPV 2 and 4

- **Plantar wart patterns**
  - Discrete
  - Mosaic
  - Cystic

- **Differential diagnosis**
  - Clavi
  - Porokeratosis

Plantar Warts
Plantar Warts

- Treatment
  - No treatment
  - Salicylic acid
  - Cantharidin
  - Pentoxifylline
  - Laser
  - Cryotherapy
  - Imiquimod topical
  - Fluorouracil topical
  - Surgical excision

Contact Dermatitis

- Two Types
  - Irritant Contact Dermatitis: Resolves in a few days
  - Allergic Contact Dermatitis: Resolves in 1-3 weeks

- Allergic Contact Dermatitis
  - Type IV cell mediated immune response: Helper T cell
  - Rhue Dermatitis: Poison Oak, Ivy Etc.
  - Latex allergy
  - Medication Dermatitis
  - Shoe Allergy

- Shoe Allergy
  - Mercaptopbenzothiazole (MBT) (rubber accelerator) #1
  - Rubber boot allergy
  - Plastics

Contact Dermatitis

- Shoe Allergy

Malignant Skin Lesions

- Squamous Cell Cacinoma
  - Chronic sun exposure
  - 10 fold increase risk to organ transplant recipients
  - Red plaque, ulceration or wart like

- Malignant Melanoma
  - Risk factors: Sun exposure, Heredity
  - 80% cured by early diagnosis

- Atypical Nevi
  - Irregular border
  - Multiple colors
  - Multiple Topographies (Elevated centrally, flat at borders)
  - Associated with increased risk for melanoma

Malignant Skin Lesions

- Malignant Melanoma

Malignant Skin Lesions

- Acral Lentiginous Melanoma
Malignant Skin Lesions

- SCC

Common Soft Tissue Masses

- Plantar Fibromatosis
  - Benign reactive lesions of the fibrous tissue
  - Involves medial and central bands of the plantar fascia
  - Can be hereditary, local trauma, associated with Dupuytren's contractures
  - Treatment: Off-loading, cortisone injections, excision

- Ganglion Cyst
  - Benign collection of synovial fluid
  - Herniation of a tendon sheath or joint capsule
  - Treatment: aspiration or surgical excision

Common Soft Tissue Masses

- Plantar Fibromatosis

Nail Disorders

- Can be used to diagnose cutaneous or systemic disease
- Anatomy of the Nail Unit
- Common Nail Changes
- Infection of the Nail Unit
- Tumors of the Nail Complex
- Systemic Diseases

Nail Anatomy

- Complex Unit
  - Hyponychium
  - Eponychium
  - Matrix
  - Nail Plate
  - Nail Bed
  - Medial Nail Fold
  - Lateral Nail Fold

Nail Anatomy

- Nail Matrix
  - Formation on new nail substance
  - Stratified squamous epithelium
  - Apical cells differentiate into hard keratin

- Nail Plate
  - 0.5mm thick in women and 0.6mm in men
  - Grows continuously
  - Growth rate 12-18 months toenails, 6 months fingernails

- Nail Bed
  - Derived from the matrix epithelium
  - Nail plate is firmly attached
  - Moves forward with nail plate at the same rate
Onychoschizia

- Lamellar Sheets of nail peel off
  - Females >> Males
  - Increases with age
  - Etiology unknown, Water exposure
  - Treatment: Protect, Moisture

Subungal Hemorrhage

- Trauma: most of the time trivial
- Hemorrhage with subungal involvement
- Often causes onycholysis

Subungal Hemorrhage

- Treatment
  - Debride nail plate
  - Differential: Melanoma vs hyperpigmented streak
  - If any doubt: Biopsy and histological review

Onycholyse

- Multiple Causes!
  - Trauma
  - Onychomycosis
  - Candidiasis
  - Psoriasis
  - Ect.
- Treatment
  - Nail debridement
  - Eliminate dead space
  - Treat underlying infection if suspected

Green Toenail

- Pseudomonas overgrowth
  - Green pigment production
- Onycholytic nail
  - Dead space
- Treatment
  - Debride nail back
  - Eliminate dead space
  - Tobramycin ophthalmic sol.

Onychocryptosis

- Hallux >> all other nails