Diabetic Foot Surgery:

A Review of Current Procedures

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Introduction

Foot pathology related to diabetes is complex, debilitating and costly to our healthcare system. The most frequent underlying etiologies are neuropathy, trauma, deformity, high plantar pressures and peripheral arterial disease (PAD).

There is consistent evidence that early identification and aggressive management of people with diabetes—through an integrated, multidisciplinary approach—can prevent problems becoming exacerbated and reduce the incidence of amputation. Treatment should always be patient-centred, and goals should include prompt lesion healing, preventing recurrence and quality of life factors.

Surgical management of diabetic foot ulcers can be either preventive or curative, depending upon the nature of the defect. This paper reviews the surgical procedures most often performed for foot pathologies resulting from diabetes, namely revascularization, surgical offloading and amputation.

Revascularization

People with diabetes have an increased incidence and severity of PAD compared with the general population.[1,2] Every 1% increase in glycated hemoglobin in people with diabetes corresponds to a 26% increase in the risk of PAD.[3]

PAD occurs when arterial plaques or stenoses form within the arterial lumen and impede blood flow. In the lower extremities, this process occurs slowly and over a wide area of the artery, most often occurring distally in the femoral-popliteal and tibial arteries in people with diabetes.[4-6] Blood flow can be temporarily diverted through smaller arterial branches via the collateral circulation. In moderate to severe stages of disease blood flow is severely restricted (i.e. stenosed) or completely blocked (i.e. occluded).

Clinical manifestations typically begin with intermittent claudication in the form of slow walking, calf cramping and leg fatigue.[7] In 50% of patients, however, symptoms of claudication are not present because of reduced activity or diabetes-associated peripheral neuropathy.[8] The symptoms may progress to disabling pain at rest, ulceration with or without infection, gangrene, limb loss and death.[9] Given the severity of outcomes, it is imperative that people with diabetes are monitored for the presence or worsening of PAD and appropriate referrals or interventions are undertaken to prevent and manage the complications of PAD.

Treatment of peripheral arterial disease

The treatment of PAD is dependent on the stage and extent of the disease, but may include risk-factor modification, exercise programs, consistent foot care, antibiotic use and surgical intervention.[4,6] Patients should be carefully monitored for changes to arterial circulation in the form of decreased or impalpable pulses, necrotic ulcerations, abnormal ankle–brachial index measurements (<0.9), disabling intermittent claudication, critical limb ischemia, rest pain or tissue loss (including ulceration). Any of these warrant further arterial testing and referral.[6,7,10]

Referral to a vascular surgeon is required to determine whether surgical intervention is needed to improve the vascular supply. Primary indications for revascularization include disabling claudication, critical limb ischemia, rest pain or tissue loss (including ulceration) refractive to conservative therapy.[6,8,10] Wound-related revascularization can be identified by the wound location and the artery preferentially requiring the return of arterial supply.[11] These wound

TABLE 1

Wound-related revascularization[11]

Location of wound	Artery that should be preferentially revascularized
Heel	Peroneal or posterior tibial
Plantar foot	Posterior tibial
Lateral ankle	Peroneal
Dorsal foot	Anterior tibial

locations and arteries can be identified by the angiosomes (anatomic areas and artery source) in Table 1. This may be helpful to clinicians treating wounds in people with PAD and an ischemic foot (i.e. a foot with impaired arterial blood flow).[11]

Endovascular interventions require determining the evidence, stage and extent of disease, as well understanding the expected benefits and risks related to these interventions.[6,10,12] The most common surgical interventions include arterial bypass and percutaneous transluminal angioplasty.[6,10,12] These are described in Table 2.

People with diabetes and PAD often have an altered distribution of lower-extremity disease, with severe arterial occlusive disease below the knee in the runoff vessels (collateral circulation). As this worsens, the success of percutaneous intervention declines.[4] Generally, percutaneous transluminal angioplasty with or without stenting is preferred for patients with focal disease and restorable runoff, while surgery (arterial bypass) is preferred for patients with diffuse disease and poor runoff.[4,6,12] Outcomes following arterial surgery are becoming increasingly positive as techniques are refined.[13–15]

Some limbs cannot be revascularized due to the lack of a target vessel, the unavailability of autogenous vein or irreversible gangrene beyond the midfoot (Figure 1).[12] In addition, those in poor general health may be at high risk of death if major revascularization is attempted. In these situations, a choice must be made between primary amputation and prolonged medical therapy.[6,10,12]

Conservative therapy for limbs with a questionable arterial supply or those that cannot be revascularized includes pain management, avoidance of further tissue damage and infection, avoidance of sharp debridement and moist wound healing, and the use of broad-spec-

TABLE 2

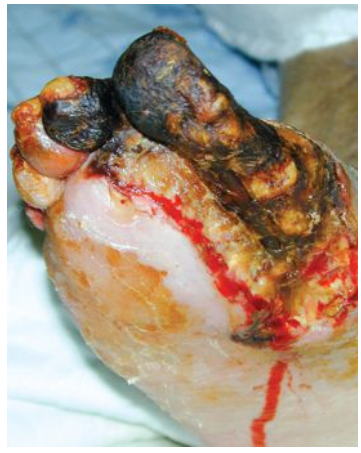
Comparison of revascularization procedures for peripheral arterial disease[6,10,12]

	Arterial bypass	Percutaneous transluminal angioplasty
Surgical definition	An open surgical procedure where a graft or new blood vessel is bypassed around an area of narrowing or blockage A healthy vein or synthetic material is used as a graft	Endovascular: a small percutaneous incision is made in the groin. The procedure os completed inside the artery using a catheter and interventional radiology May involve: • angioplasty—a balloon-tipped catheter is inflated and deflated, pressing plaque against the artery wall and opening a narrowed area; • atherectomy—a tiny blade, laser or rotating burr is inserted through the catheter to remove plaques; or • stenting—a mesh tube is placed into the vessel, using a catheter to reperfuse vessel
Expected benefit	Revascularization of all lesions	Focal disease (stenosis of larger, more proximal vessels) and improved claudication symptoms
Common arteries	Popliteal Tibial	Iliac Femoral
Durability	Greater durability	Long-term efficacy uncertain
Risks	Association with morbidity, mortality and graft occlusion is higher for people with diabetes Other problems may include bleeding, blood clots, infection, heart or lung complications, kidney problems and loss of the toe or foot	Restenosis, lower-limb salvage rates in people with diabetes, rupture of artery, bleeding, blood clots, heart or lung complications, kidney problems, loss of toe or foot and death

trum antiseptics.[16,17] In people with diabetes and advanced PAD, close collaboration is required between the patient, his/her family and caregivers, and the wound care practitioner, physician and vascular surgeon.[6,10,16]

People with diabetes and PAD require careful and ongoing monitoring to identify arterial changes in the progression of PAD that place a limb at risk for amputation.[6,12] Risk factor modification (lowering of blood pressure, cholesterol and blood glucose levels, smoking cessation and exercise), regular foot care and avoidance of foot injury are necessary to prevent and manage PAD.[6,10,17,18] Referral to a vascular surgeon for evaluation of the arterial supply should not be delayed when an arterial impairment is identified.[12] Surgical interventions may be necessary to improve the arterial supply, and outcomes are improving for those with diabetes and PAD.[10,12]

FIGURE 1



A person with diabetes and PAD presenting with significant necrotic 1 and 2 digits. This person was not a candidate for revascularization because of ill health.

Surgical offloading

Deformity, peripheral neuropathy and trauma often lead to foot ulceration.[19,20] Structural alterations in the foot resulting from deformity lead to high plantar foot pressures and increased dorsal, medial or lateral pressure on the foot. This can consequently place the foot at risk for ulceration.[20]

Surgical correction of problematic deformities may prevent ulcers from occurring and help to heal those that are present.[19] Conversely, the disadvantages of surgical offloading include a risk of complications, including postoperative wound infection, induction of acute neuro-osteoarthropathy and development of ulcers at other sites. Such risks can be minimized through careful patient selection and a thorough evaluation of comorbidities. Table 3 outlines the American Diabetes Association's risk classification system.[21]

Sharp surgical debridement

Sharp surgical debridement has multiple advantages, including appropriate evaluation of the wound bed, penetration of topical agents, changing the wound stage from a chronic to acute wound and reducing plantar pressure.[22]

Patients with foot ulcers with an adequate blood supply can undergo sharp surgical debridement of devitalized and necrotic tissue at the bedside. The procedure is quick, cost effective and painless (because of the presence of neuropathy). However, clinicians must

TABLE 3

Risk classification system of the Task Force of the Foot Care Interest Group of the American Diabetes Association[21]

Risk category	Definition	Treatment recommendations	Suggested follow-up
0	No LOPS, no PAD, no deformity	Consider patient education on foot care, including information on appropriate footwear	Annually (by generalist and/or specialist)
1	LOPS ± deformity	Consider prescriptive or accommodative footwear Consider prophylactic surgery if deformity is not able to be safely accommodated in shoes Continue patient education	Every 3–6 months (by generalist or specialist)
2	PAD ± LOPS	Consider the use of accommodative footwear Consider a vascular consultation for combined follow-up	Every 2–3 months (by generalist or specialist)
3	History of ulcer or amputation	Consider patient education on foot care Consider vascular consultation for combined follow-up if PAD present	Every 1–2 months (by specialist)

LOPS = loss of protective sensation; PAD = peripheral arterial disease

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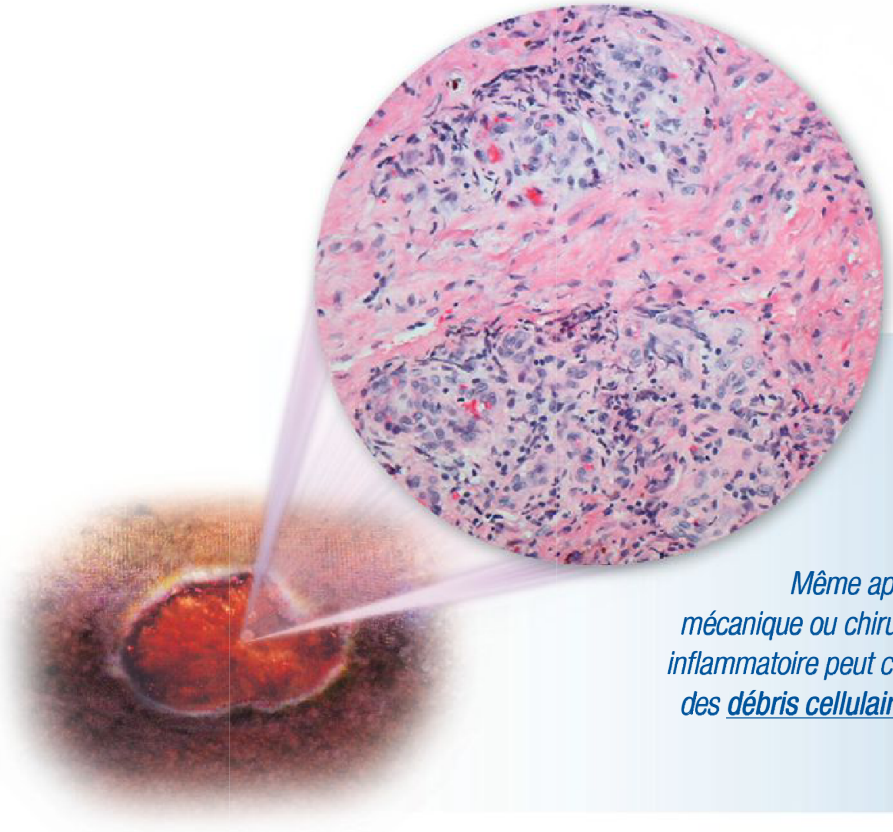

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Occasional slight transient erythema has been noted in surrounding tissue when applied outside the wound. One case of systemic hypersensitivity has been reported after 1 year of treatment with collagenase and cortisone.

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On a noté un érythème occasionnel et léger sur les tissus environnants lorsque l'application de l'onguent dépasse le pourtour de la plaie. Un cas d'hypersensibilité systémique a été rapporté après un an de traitement à la collagénase et à la cortisone.

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CLINICAL PHARMACOLOGY: Santyl® (collagenase) possesses the ability to digest insoluble collagen, undenatured and denatured, by peptide bond cleavage, under physiological conditions of pH and temperature. This ability makes it particularly effective in the removal of detritus from dermal lesions, contributing towards the more rapid formation of granulation tissue and subsequent epithelization of dermal ulcers and severely burned areas. Collagen in healthy tissue or in newly formed granulation tissue is not digested.

INDICATIONS: Santyl® (collagenase) is a sterile ointment indicated for the debridement of dermal ulcers or severely burned areas.

CONTRAINDICATIONS: Application is contraindicated in patients who have shown local or systemic hypersensitivity to collagenase.

WARNINGS: Debilitated patients should be closely monitored for systemic bacterial infections because of the theoretical possibility that debriding enzymes may increase the risk of bacteremia.

PRECAUTIONS: The enzyme's optimal pH range is 6 to 8. Significantly lower pH conditions have a definitive adverse effect on the enzyme's activity, and appropriate precautions should be carefully taken. The enzymatic activity is also adversely affected by detergents, hexachlorophene and heavy metal ions such as mercury and silver that are used in some antiseptics and by cobalt, magnesium and manganese. When it is suspected such materials have been used, the site should be carefully cleansed by repeated washings with normal saline before Santyl® (collagenase) ointment is applied. Soaks containing metal ions or acidic solutions such as Burow's solution should be avoided because of the metal ion and low pH. Cleansing materials such as hydrogen peroxide or Dakin's solution followed by sterile normal saline do not interfere with the activity of the enzyme. The ointment should be confined to the area of the lesion in order to avoid the possible risk of irritation or maceration of normal skin; however, the enzyme does not damage newly forming granulation tissue. A slight erythema has been noted occasionally in the surrounding tissue particularly when the enzyme ointment was not confined to the lesion. This can be readily controlled by protecting the healthy skin with a material such as zinc oxide paste. Since the enzyme is a protein, sensitization may develop with prolonged use.

ADVERSE REACTIONS: Although no allergic sensitivity or toxic reactions have been noted in the recorded clinical investigations to date, one case of systemic manifestations of hypersensitivity has been reported in a patient treated for more than one year with a combination of collagenase and cortisone. Irritation, maceration or erythema has been noted where prolonged contact of normal skin with Santyl® (collagenase) ointment has been allowed, either by application of the ointment to areas of normal skin or by excessive application of ointment to the wound crater with subsequent spread to normal skin when dressings are applied. The reported incidence for this type of reaction was 1.8%.

SYMPTOMS AND TREATMENT OF OVERDOSE: Symptoms: To date, the irritation, maceration or erythema reported on prolonged contact of normal skin with Santyl® (collagenase) ointment constitute the only symptoms of overdosage reported. **Treatment:** Santyl® (collagenase) ointment can be rendered inert by the application of Burow's solution USP (pH 3.6 - 4.4) to the treatment site. If this should be necessary, reapplication should be made only with caution.

DOSAGE AND ADMINISTRATION: For external use only. Santyl® (collagenase) ointment should be applied once daily, or more frequently if the dressing becomes soiled (as from incontinence) in the following manner: **(1)** Prior to application the lesions should be gently cleansed with a gauze pad saturated with sterile normal saline, to remove any film and digested material. If a stronger cleansing solution is required, hydrogen peroxide or Dakin's solution may be used, followed by sterile normal saline. **(2)** Whenever infection is present, as evidenced by positive cultures, pus, inflammation or odor, it is desirable to use an appropriate antibacterial agent. Should the infection not respond, therapy with Santyl® (collagenase) ointment should be discontinued until remission of the infection. **(3)** Santyl® (collagenase) ointment should be applied (using a tongue depressor or spatula) directly to deep wounds, or when dealing with shallow wounds, to a non-adherent dressing or film dressing which is then applied to the wound. The wound is covered with an appropriate dressing such as a sterile gauze pad and properly secured. **(4)** Use of an occlusive or semi-occlusive dressing may promote softening of eschar, if present. Alternatively, crosshatching thick eschar with a #11 blade is helpful in speeding up debridement then cleanse with sterile saline. It is also desirable to remove as much loosened detritus as can be done readily with forceps and scissors. **(5)** All excess ointment should be removed each time the dressing is changed. **(6)** Use of Santyl® (collagenase) ointment should be terminated when debridement of necrotic tissue is complete and granulation is well under way.

HOW SUPPLIED: Available in 30 gram tubes of ointment. Sterile until opened. Contains no preservative. Do not store above 25°C.

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PHARMACOLOGIE CLINIQUE : Santyl® (collagénase) a la capacité de digérer le collagène insoluble, non dénaturé et dénaturé, par clivage de la liaison peptidique à un pH et à une température physiologiques. Cette caractéristique le rend particulièrement efficace dans l'élimination des déchets des lésions dermiques favorisant ainsi la formation du tissu de granulation et l'épithélialisation ultérieure des zones dermiques ulcérées et gravement brûlées. Le collagène des tissus sains ou du nouveau tissu de granulation n'est pas digéré.

INDICATIONS : Santyl® (collagénase) est un onguent stérile indiqué pour le débridement des zones dermiques ulcérées ou gravement brûlées.

CONTRE-INDICATIONS : L'application est contre-indiquée chez les patients ayant présenté une hypersensibilité locale ou systémique à la collagénase.

MISE EN GARDE : Les patients atteints de conditions débilitantes doivent être surveillés étroitement pour éviter la généralisation des infections bactériennes. Les enzymes de débridement augmenteraient le risque de bactériémie.

PRÉCAUTIONS : Le pH optimal de l'enzyme est de 6 à 8. Un pH nettement inférieur à un effet nettement adverse sur l'action de l'enzyme et des précautions appropriées doivent alors être prises. L'action de l'enzyme est également contrariée par les détergents, l'hexachlorophène et les ions de métaux lourds, comme le mercure et l'argent, présents dans certains antiseptiques, et par le cobalt, le magnésium et le manganèse. Quand on soupçonne l'utilisation de ces produits, la zone affectée doit être soigneusement nettoyée par des lavages répétés avec une solution saline avant l'application de l'onguent Santyl® (collagénase). Les bains contenant des ions de métaux ou des solutions acides comme la solution de Burow doivent être évités en raison de l'ion métal et du faible pH. Les solutions nettoyantes comme l'eau oxygénée ou la solution de Dakin suivie d'une solution stérile saline n'entravent pas l'action de l'enzyme. L'application de l'onguent doit se limiter à la zone affectée pour éviter le risque possible d'irritation ou de macération de la peau saine. Cependant, l'enzyme n'altère pas le nouveau tissu de granulation. Un érythème bénin dans le tissu avoisinant pourrait se produire. Cela peut facilement être évité en protégeant la peau saine avec un produit comme de la pâte d'oxyde de zinc. Compte tenu de la nature protéique de l'enzyme présent dans le médicament, son emploi prolongé pourrait amener une sensibilisation.

EFFETS SECONDAIRES : Bien qu'aucune sensibilité allergique ni réaction toxique n'aient été notées à ce jour dans les compte rendus d'études, on a signalé un cas de manifestations systémiques d'hypersensibilité chez un patient traité pendant plus d'un an avec une association de collagénase et de cortisone. On a noté de l'irritation, de la macération ou de l'érythème dans le cas de contact prolongé de la peau normale avec l'onguent Santyl® (collagénase), soit par application de l'onguent sur les régions normales de la peau, soit par application excessive de l'onguent dans le cratère de la plaie, permettant à celui-ci de s'étendre à la peau normale lors de l'application des pansements. L'incidence signalée de ce type de réaction était de 1,8 %.

SYMPTÔMES ET TRAITEMENT DU SURDOSAGE: Symptômes: Jusqu'ici, l'irritation, la macération ou l'érythème signalés en cas de contact prolongé de la peau saine avec l'onguent Santyl® (collagénase) représentent les seuls symptômes signalés de surdosage. **Traitement :** On peut rendre l'onguent Santyl® (collagénase) inerte en appliquant la solution de Burow U.S.P. (pH 3.6-4.4) sur la plaie. La réapplication du produit, si elle est considérée nécessaire, ne se fera qu'avec prudence.

POSOLOGIE ET ADMINISTRATION : Pour usage externe seulement. L'onguent Santyl® (collagénase) doit être appliqué une fois par jour ou plus fréquemment si le pansement se souille (à cause d'incontinence par exemple) de la façon suivante : **(1)** Avant application, les lésions doivent être nettoyées doucement avec une gaze saturée d'une solution stérile saline normale pour enlever toute pellicule et toute matière digérée. Si l'on a besoin d'une solution nettoyante plus puissante, on peut utiliser de l'eau oxygénée ou de la solution de Dakin suivie de solution stérile saline normale. **(2)** En cas d'infection, révélée par la présence de cultures positives, de pus, d'une inflammation ou d'une odeur, il serait souhaitable d'employer un agent antibactérien approprié. Il faut interrompre le traitement au Santyl® (collagénase) jusqu'à rémission de l'infection, si l'infection ne se résorbe pas. **(3)** Appliquer Santyl® (collagénase) directement sur les blessures profondes à l'aide d'un abaisse-langue ou d'une spatule. Pour les plaies superficielles, appliquer l'onguent sur une compresse non adhérente ou un pansement transparent à être déposée sur la plaie; puis recouvrir d'un pansement approprié tel une compresse de gaze stérile adéquatement retenue. **(4)** L'utilisation d'un pansement occlusif ou semi-occlusif peut favoriser le ramollissement de l'escarre, le cas échéant. Ou, si l'on hachure une escarre épaisse à l'aide d'une lame numéro 11, on peut accélérer le débridement. Nettoyer alors avec une solution saline stérile. Il est également souhaitable d'enlever autant de détritus lâches que possible à l'aide de pinces et de ciseaux. **(5)** Enlever tout excès d'onguent à chaque renouvellement du pansement. **(6)** Arrêter les applications de l'onguent Santyl® (collagénase) dès que le tissu nécrosé est suffisamment débridé et que le bourgeonnement est bien entamé.

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ensure they have adequate training to perform this procedure, and that the procedure falls within their scope of practice.

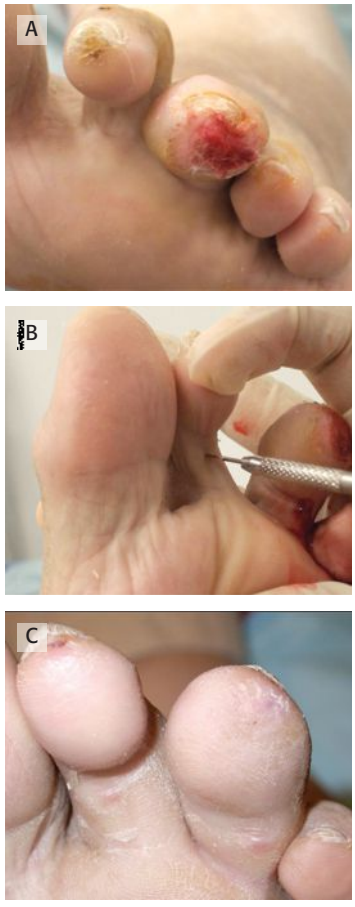
Achilles tendon lengthening

People with diabetes undergo structural changes in the Achilles tendons. These lead to changes in the function of tendons that may contribute to limited dorsiflexion/plantar flexion in the ankle joints. This is called ankle equinus.[23,24] Limited range of motion in the ankle joints may result in early heel rise and prolonged and excessive weight-bearing stress under the metatarsal heads, leading to increased forefoot pressure.[25,26] Others have suggested that the relationship between ankle equinus (dorsiflexion <5 degrees) and forefoot plantar pedal pressure is significant, but that ankle equinus plays only a limited role in causing forefoot pressure.[27,28]

Proponents of ankle equinus as a major factor in forefoot ulceration have shown that Achilles tendon lengthening to treat recurrent forefoot ulcerations increases the range of motion of the ankle joint from 0 to 9 degrees,[27] consequently reducing forefoot pressure. However, after 7 months—when plantar flexor/muscle power returns to preoperative levels, while range of motion in the ankle joint remains at the post-treatment level—plantar pressure returns to the level preoperatively recorded.[29,30]

Achilles tendon lengthening for the treatment of neuropathic ulcers causes a temporary reduction in forefoot pressure associated with changes in plantar flexor power, rather than ankle motion during gait; thus, it appears that the reduction in forefoot pressure is associated with changes in plantar flexor muscle power rather than ankle motion during gait. In addition, a study by Maluf et al. showed that by increasing range of motion in the ankle joint, rear foot pressure remained permanently increased while forefoot pressure was only temporarily decreased.[30] This may lead to a significant complication (i.e. increased risk of heel ulceration), which has been recorded in 15% of patients treated with Achilles tendon lengthening.[29] Thus, there are strong arguments against tendon Achilles lengthening for the treatment of forefoot foot ulcers.

FIGURE 2



Percutaneous flexor tenotomy. (A) Claw toe with apical ulceration. (B) Performing percutaneous flexor tenotomy. (C) Complete healing of the apical ulcer (3 weeks post-operatively).

Digital surgery

Digital surgery (hammer-toe correction, bunionectomy) can reduce bony prominences and ensure a better fit between footwear and the foot, thus preventing ulceration. Such offloading procedures have the greatest benefit in young, healthy, complication-free (i.e. without vascular disease or neuropathy) people with diabetes.[31] In older patients with diminished general health, the risk of complications from the surgery may outweigh the benefits.[32]

Percutaneous flexor tenotomy for claw-toe deformity

Claw-toe deformities are common in those who have had diabetes for more than 10 years.[33] Claw toe is defined as hyperextension of the metatarsophalangeal joint and flexion of the interphalangeal joint. This deformity may cause ulceration at the tip of the toe due to pressure, or at the dorsum of the proximal interphalangeal joint from rubbing against footwear.[34] Percutaneous flexor tenotomy may be performed to release the long flexor tendon of the toe (Figure 2).[35]

A retrospective study from 1 Canadian hospital demonstrated complete healing of all ulcerations and correction of the deformity with percutaneous flexor tenotomy.[36] Of the 34 toes reviewed, all had apical ulcers and 3 were complicated by osteomyelitis. Most ulcers healed within 3 weeks of percutaneous flexor tenotomy and those with osteomyelitis healed within an average of 8 weeks. There were no significant complications and the average length of follow-up was 13 months. Percutaneous flexor tenotomy can be considered an effective method for the management of claw-toe deformity and ulceration.[36]

Silicone injections

A randomized controlled trial injected silicone into diabetic feet at the metatarsal heads to increase tissue thickness and decrease peak plantar pressure. Although this proved effective in the short term, at 24-month follow up the tissue thickness area and plantar pressure had returned to pre-injection levels.[37]

Charcot foot

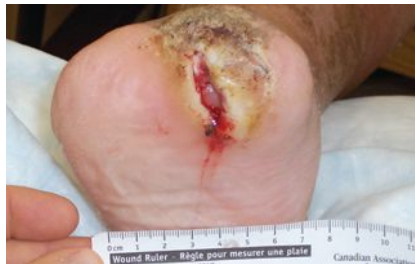
Surgical reconstruction of the Charcot foot is extremely valuable for patients with recurrent ulceration (with or without infection), severe instability or severe deformities that cannot be managed with footwear or braces.[20,38] The goal of this surgery is to stabilize and align the foot, thereby allowing patients to wear shoes and braces.[20] However, the risk of adverse events is high.

Charcot foot surgeries can be challenging and have frequent complications. Proper patient selection is important for limb salvage.[20]

Achilles tendon lengthening with plantar exostectomy

A chronic Charcot neuroarthropathy commonly represents as a rocker-bottom foot, in which the midfoot bones of the arch collapse and subsequently weight bear in areas that are unaccustomed to the high pressures. By combining Achilles tendon lengthening with removal of the bony prominences, a plantigrade foot with reduced plantar foot pressures may be produced.[20,38] A stable foot structure is necessary for this procedure.

FIGURE 3



A transmetatarsal amputation demonstrates pressure distribution issues that can often occur if supportive orthotic devices are not fitted appropriately.

Arthrodesis

Arthrodesis is a process of bony fusion that is carried out after ulcerations have healed.

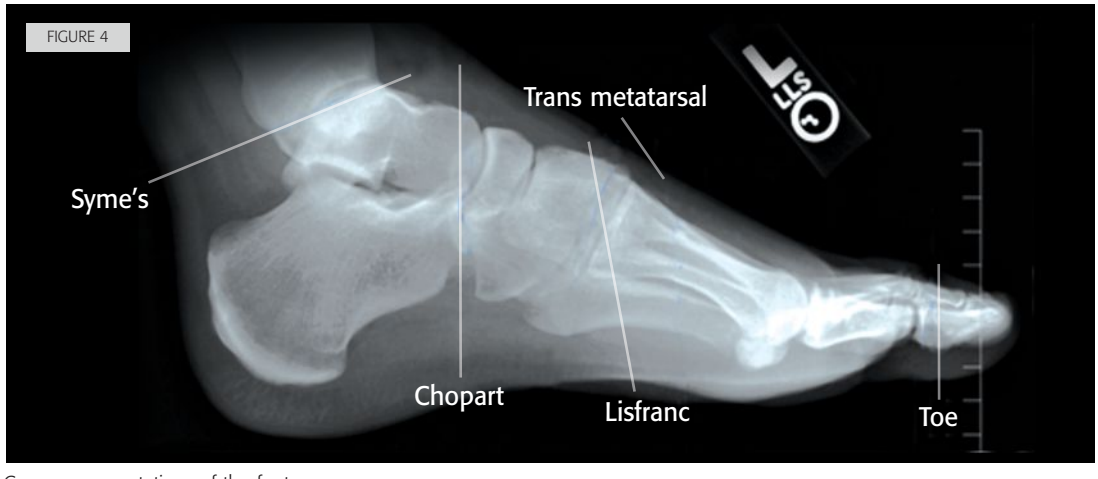
The choice of internal or external fixation depends on the quality of the bone.[20,39]

Amputation

The major indications for therapeutic amputation are trauma, ischemia, malignancy and infection.[40] In chronic wound situations, intractable pain and patient choice weigh heavily in the decision to proceed with amputation once limb salvage options have been exhausted. Surgical removal of portions of a foot or a total below-the-knee amputation may be required when best practice wound therapy is unable to close a foot wound. In these cases, minor amputations may be perceived as victories, as the tissue damage requiring removal will have been restricted.[41]

The surgical goal of amputation is to obtain a balance between wound healing and a functional limb. As with all limb salvage techniques, the arterial supply is directly associated with postoperative outcomes.[42] Adequate viable soft tissue coverage of the surgical wound is imperative to protect and cushion during eventual mobilization of the residual limb. As increasing amounts of the foot are removed, there is a corresponding increase in plantar pressures and instability due to altered biomechanics. These must be supported with appropriate foot wear and customized orthotics (Figure 3).

FIGURE 4



Common amputations of the foot.

TABLE 4

"Amputation cascade" of the foot

Level of amputation	Bony structures involved
Toe(s)	Part or all of the phalanges
Ray(s)	All of the phalanges and part or all of the respective metatarsal
Transmetatarsal	Through all of the metatarsals
Lisfranc	Disarticulation at the tarsometatarsal joints
Chopart	Disarticulation through the talonavicular and calcaneocuboid joints
Syme's	Disarticulation of the talus and tibia/fibula, with retention of the calcaneal fat pad

Although preoperative planning is essential for amputations, the exact level of amputation may be dictated intraoperatively due to sinus tracts, necrotic or infected tissue and skin flap bleeding. Common amputation levels are shown in Figure 4 and Table 4. Lisfranc and Chopart amputations have minimal if any functional benefits over Syme's amputation, despite preserving more of the foot,[43] and frequently result in recurrent ulcerations; in general, these are avoided. Syme's amputation is suitable for patients with diabetes; however, below-the-knee amputation is often favoured due to greater prosthetic options.

Most patients with diabetes and partial foot amputations require foot orthotics at a minimum to prevent further complications. Postoperatively, higher-level amputations are optimally coupled with preprosthetic rehabilitation planning.

The partial loss of a lower limb represents a major change in a person's life, but patients should be encouraged to approach amputation as the beginning of a new phase of life and not as the culmination of previous treatment failures.[44] Pain relief and the removal of a nonfunctional limb may greatly enhance quality of life.

Conclusion

As a component of care, surgery provides an effective method of addressing diabetic foot complications to offload, revascularize, manage infection and, ultimately, improve the quality of life for people with diabetes.

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References

1. Jude EB, Oyibo SO, Chalmers N, Boulton AJ. Peripheral arterial disease in diabetic and nondiabetic patients. *Diabetes Care*. 2001;24:1433–1437.

2. Suzuki LA, Poot M, Gerrity RG, Bornfeldt. Diabetes accelerates smooth muscle accumulation in lesions of atherosclerosis: lack of direct growth-promoting effects high glucose levels. *Diabetes*. 2001;50:851–860.

3. Selvin E, Marinopoulos S, Berkenblit G, et al. Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. *Ann Intern Med*. 2004;141:421–431.

4. Beckman JA, Creager MA, Libby P. Diabetes and atherosclerosis: epidemiology, pathophysiology, and management. *JAMA*. 2001;19: 2570–2581.

5. Hirsch AT, Criqui MH, Treat-Jacobson D, et al. Peripheral arterial disease detection, awareness and treatment in primary care. *JAMA*. 2001;286:1317–1324.

6. American Diabetes Association. Peripheral arterial disease in people with diabetes. *Diabetes Care*. 2003;26:3333–3341.

7. McDermott MM, Liu K, Greenland P, et al. Functional decline in peripheral arterial disease: associations with the ankle brachial index and leg symptoms. *JAMA*. 2004;292:453–461.

8. Norgren L, Hiatt W, Dormandy J, et al. Inter-society consensus for the management of peripheral arterial disease. *Int Angiol*. 2007; 26:81–157.

9. Imparato AM, Kim GE, Davidson T, Crowley JG. Intermittent claudication: its natural course. *Surgery*. 1975;78:795–799.

10. Dyet JF, Nicholson AA, Ettles DF. Vascular imaging and intervention in peripheral arteries in the diabetic patient. *Diabetes Metab Res Rev*. 2000;16(Suppl. 1):S16–S22.

11. Setacci C, DeDonato G, Setacci F, Chisci E. Ischemic foot: definition, etiology and angiosome concept. *J Cardiovasc Surg*. 2010:51:223–231.

12. Norgren L, Hiatt WR, Dormandy JA, et al. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASCII). *Eur J Vasc Endovasc Surg*. 2007;33(Suppl. 1):S1–S75.

13. Faglia E, Mantero M, Caminiti M, et al. Extensive use of peripheral angioplasty, particularly infrapopliteal, in the treatment of ischaemic diabetic foot ulcers: clinical results of a multicentre study of 221 consecutive diabetic subjects. *J Intern Med*. 2002;252:225–232.

14. BARI Investigators. Seven-year outcome in the Bypass Angioplasty Revascularisation Investigation (BARI) by treatment and diabetic status. *J Am Coll Cardiol*. 2000;35:1122–1129.

15. Van Gils CC, Wheeler LA, Mellstrom M, et al. Amputation prevention by vascular surgery and podiatry collaboration in high-risk diabetic and nondiabetic patients. The Operation Desert Foot experience. *Diabetes Care*. 1999;22:678–683.

16. Sibbald R, Williamson D, Orsted H, et al. Preparing the wound bed: debridement, bacterial balance and moisture balance. *Ostomy Wound Manage*. 2000;46:14–35.

17. Botros M, Goetti K, Parsons L, et al. Best practice recommendations for the prevention, diagnosis and treatment of diabetic foot ulcers: update 2010. *Wound Care Can*. 2010;8:6–40.

18. Apelqvist J, Bakker K, van Houtum WH, et al. The development of global consensus guidelines on the management of the diabetic foot. *Diabetes Metab Res Rev*. 2008;24(Suppl. 1):S116–S11.

19. Lavery LA, Peters EJG, Armstrong DG. What are the most effective interventions in preventing diabetic foot ulcers? *Int Wound J* 2008;5:425–433.

20. Frykberg R, Bevilacqua J, Habershaw G. Strategies to prevent and heal diabetic foot ulcers. *J Am Podiatr Med Assoc*. 2010;100:369–384.

21. Boulton AJ, Armstrong DG, Albert SF, et al. Comprehensive foot examination and risk assessment: a report of the task force of the foot care interest group of the American Diabetes Association, with endorsement by the American Association of Clinical Endocrinologists. *Diabetes Care*. 2008;31:1679–1685.

22. Young MJ. The effect of callus removal on dynamic plantar pressure in diabetic patients. *Diabet Med*. 1992:55–57.

23. Mueller MJ, Diamond JE, Delitto A, Sinacore DR. Insensitivity, limited joint mobility and plantar ulcers in patient with diabetes mellitus. *Phys Ther*. 1989;69:453–462.

24. Mueller MJ, Minor SD, Sahrmann SA, Schaaf JA, Strube MJ. Differences in the gait characteristics of patients with diabetes and peripheral neuropathy compared with age-matched controls. *Phys Ther*. 1994;74:299–313.

25. Zimny S, Schatz H, Pfohl M. The role of limited joint mobility in diabetic patients with an at-risk foot. *Diabetes Care*. 2004;27:942–946.

26. Lavery LA Armstrong DG, Boulton AJ; Diabetes Research Group. Ankle equinus deformity and its relationship to high plantar pressure in large population with diabetes mellitus. *J Am Podiatr Med Assoc*. 2002;92:479–482.

27. Mueller MJ, Sinacore DR, Hastings MK, Strube MJ, Johnson JE. Effect of Achilles tendon lengthening on neuropathic plantar ulcers: a randomized controlled trial. *J Bone Joint Surg Am*. 2003;85A:1436–1445.

28. Orendruff MS, Rohr ES, Sangeorzan BJ, Weaver K, Czerniecki JM. An equinus deformity of the ankle accounts for only a small amount of the increased forefoot plantar pressure in patient with diabetes. *J. Bone Joint Surg*. 2006:88:65–68.

29. Holstein P. Achilles tendon lengthening the panacea for plantar forefoot ulceration? *Diabetes Metab Res Rev*. 2004;20:37–40.

30. Maluf KS, Mueller MJ, Strube MJ, Engsberg JR, Johnson JE. Tendon Achilles lengthening for the treatment of neuropathic ulcers causes a temporary reduction in forefoot pressure associated with changes in plantar flexor power rather than ankle motion during gait. *J Biomech*. 2004;37:897–906.

31. Armstrong DG. Is prophylactic diabetic foot surgery dangerous? *J Foot Ankle Surg*. 1996;35:585–589.

32. Kravitz SR, McGuire JB, Sharma S. The treatment of diabetic foot ulcers: reviewing the literature and surgical algorithm. *Adv Skin Wound Care*. 2007;4:227–237.

33. Reiber G, Boyko E, Smith D. Lower extremity foot ulcers and amputations in diabetes. In: Harris M (ed). *Diabetes in America*. Bethesda, MD: National Institutes of Health; 1995:409–428.

34. Cavanagh P, Ulbrecht J. Biomechanics of the foot in diabetes. In: Bowker J, Levin ME, O'Neal LW (eds). *The Diabetic Foot*. St Louis, MO: Mosby; 1993:207–226.

35. Feeney MS, Willams RL, Stephen MM. Selective lengthening of the proximal flexor tendon in the management of acquired claw toes. *J Bone Joint Surg Br*. 2001;83:335–338.

36. Tamir E, McLaren AM, Gadgil A, Daniels TR. Outpatient percutaneous flexor tenotomies for management of diabetic claw toe deformities with ulcers: a preliminary report. *Can J Surg*. 2008;51:41–44.

37. van Siche CH. The effect of silicone injections in the diabetic foot on peak plantar pressure and plantar tissue thickness: 2-year follow up. *Arch Phys Med Rehabil*. 2002:919–923.

38. Catanzariti AR, Mendicino R, Haverstock B. Ostectomy for diabetic neuroarthropathy involving the midfoot. *J Foot Ankle Surg*. 2000;39:291–300.

39. Bevilacqua NJ, Rogers LC. Surgical management of Charcot midfoot deformities. *Clin Podiatr Med Surg*. 2008;25:81–94.

40. Göktepe AS, Cakir B, Yilmaz B, Yazicioglu K. Energy expenditure of walking with prostheses: comparison of three amputation levels. *Prosthet Orthot Int*. 2010;34:31–36.

41. Attinger CE, Meyr AJ, Fitzgerald S, Steinberg JS. Preoperative doppler assessment for transmetatarsal amputation. *J Foot Ankle Surg*. 2010; 49:101–105.

42. Condie DN, Bowers R. Amputations and disarticulations within the foot: prosthetic management. In: Smith D, Michael JW, Bowker JH (eds). *Atlas of Amputations and Limb Deficiencies: Surgical, Prosthetic, and Rehabilitation Principles*, 3rd ed. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2004.

43. Lepäntalo M, Biancari F, Tukiainen E. Never amputate without consultation of a vascular surgeon. *Diabet Metab Res Rev*. 2000;16 (Suppl. 1):S27–S32.

44. Ng VY, Berlet GC. Evolving techniques in foot and ankle amputation. *J Am Acad Orthop Surg*. 2010;18:223–235.