17 Surgical Treatment of the Ulcerated Foot

John M. Giurini

Abstract

Foot ulceration with infection continues to be one of the leading causes of hospitalization for patients with diabetes mellitus. It has been previously reported that the incidence and prevalence of diabetic foot ulcerations is believed to be 15%. The rate of recidivism remains a staggering 50% with the majority of these ulcerations recurring within 18 months. This has significant economic ramifications on the health care system when one considers that the average total direct cost of healing an infected ulceration not requiring amputation is approximately $17,500 per episode.

Successfully treating diabetic foot infections and ulcerations requires a thorough understanding of the risk factors for ulcerations and amputations. It requires taking advantage of advances in antimicrobial therapy, wound healing strategies including topical growth factors, negative pressure wound therapy (NPWT), improved vascular interventions, and a more aggressive surgical approach where indicated. The key components for successful outcomes require the establishment of treatment algorithms utilizing the above advances and the identification of a dedicated team of health care professionals to manage these complex problems.

Keywords

Preoperative evaluation • Anesthesia techniques • Surgical approach • Forefoot procedures • First ray • Lesser digits • Lesser metatarsal procedures • Lesser metatarsal osteotomy • Lesser metatarsal head resection • Ulcer excision • Panmetatarsal head resection • Midfoot procedures • Ostectomy • Exostectomy • Fasciocutaneous flap • Medial column fusion • Hindfoot procedures • Calcanectomy • Tendo-Achilles lengthening • Midfoot arthrodesis • Triple arthrodesis • Pantalar arthrodesis

J.M. Giurini, DPM (✉)
Division of Podiatry, Beth Israel Deaconess Medical Center, West Campus, 1 Deaconess Road, Boston, MA 02215, USA
e-mail: jgiurini@bidmc.harvard.edu

A. Veves et al. (eds.), *The Diabetic Foot: Medical and Surgical Management*, Contemporary Diabetes,
DOI 10.1007/978-1-61779-791-0_17,

Introduction

Diabetes continues to have a significant socio-economic impact on the health care system of the United States. It was estimated that there were approximately 16 million people in 1997 with either diagnosed or undiagnosed diabetes mellitus in the United States [1]. Ten years later the CDC reports, there are nearly 24 million diabetic people or 7.8% of the population [2]. The incidence of diabetes does not appear that it will slow down any time soon. A recent study projects that by the year 2050 nearly 30% of the United States population will have diabetes [3].

As the incidence of diabetes rises, so will the cost of providing care rise to no one's surprise. In 1987, $20.4 billion was spent in direct and indirect costs of care for diabetic patients. In 1997, this had risen to $91.8 billion [1]. By 2007, the total cost for providing care to patients with diagnosed diabetes nearly doubled to $174 billion [2].

Foot ulceration with infection continues to be one of the leading causes of hospitalization for patients with diabetes mellitus. It has been previously reported that the incidence and prevalence of diabetic foot ulcerations is believed to be 15% [4]. The rate of recidivism remains a staggering 50% with the majority of these ulcerations recurring within 18 months. This has significant economic ramifications on the health care system when one considers that the average total direct costs of healing an infected ulceration not requiring amputation is approximately $17,500 per episode [5].

Diabetic patients remain 15 times more likely to undergo a major lower extremity amputation than nondiabetic patients with the total number of major limb amputations being around 71,000 [2]. In 1993, this number was around 50,000 [6]. The cost for lower extremity amputation ranges between $30,000 and $33,500 [5]. In 1993 this amount was $600 million [1, 4]. The Department of Health and Human Services had set a goal of a 40% reduction in the number of diabetic amputations by the year 2000 [7]. Needless to say, we have not met that goal.

Successfully treating diabetic foot infections and ulcerations requires a thorough understanding of the risk factors for ulcerations and amputations. It requires taking advantage of advances in anti-microbial therapy, wound healing strategies including topical growth factors, negative pressure wound therapy (NPWT), improved vascular interventions, and a more aggressive surgical approach where indicated. The key components for successful outcomes require the establishment of treatment algorithms utilizing the above advances and the identification of a dedicated team of health care professionals to manage these complex problems [8–11].

Goals of Surgery

The goals of surgery in patients with neuropathy differ from the goals of surgery in patients with normal sensation. It is important that these goals are clearly delineated and understood by both the patient and the surgeon.

The primary reason for surgical intervention in patients with normal sensation is to correct an underlying deformity and reduce or eliminate a patient's pain. In the absence of pain as in the neuropathic foot, the primary goal of surgery is to reduce the risk of lower extremity amputation by correcting a structural deformity which may lead to ulceration, or to eliminate a focus of osteomyelitis (Table 17.1).

Another important distinction to make is the difference between elective surgery, prophylactic surgery, and urgent surgery, as it relates to diabetic foot surgery. Elective surgery implies the presence of a deformity that can be corrected surgically but does not put the patient or the limb at immediate risk. Oftentimes, these deformities in the diabetic patient can be managed without surgery. There are clearly clinical situations where this type of conservative approach is in the patient's best interest.

Table 17.1 Surgical goals in the insensate patient

• Reduce risk for ulceration/amputation
• Reduce foot deformity
• Provide stable foot for ambulation
• Reduce pain
• Improve appearance of foot

Prophylactic surgery is surgery performed to prevent a more serious event. In the case of the diabetic patient with neuropathy, this event is most likely some type of imminent amputation. This implies the presence of a deformity and a history of a chronically recurrent ulceration that puts the limb at risk. The goals of surgery in this scenario are to eliminate the deformity, reduce the risk of reulceration, and reduce the risk of amputation.

Urgent surgery is self-explanatory. These patients commonly present with foul-smelling ulcerations with purulent drainage and cellulitis. Necrosis and abscess formation are not uncommon. These patients require immediate surgical intervention. The ultimate goal of surgery is to control the infection, to prevent the patient from becoming septic, and to save as much of the foot and/or leg as possible.

Fig. 17.1 Failure to recognize critical ischemia resulted in surgical failure in diabetic patient with autonomic neuropathy

Preoperative Evaluation

A detailed present and past medical history, past surgical history, list of current medications, and identification of risk factors such as smoking and nephropathy are critical to proper preoperative risk assessment. Patients with long-standing diabetes mellitus will often present with cardiac and renal complications which must be managed to reduce the morbidity and mortality of a local foot procedure [12]. The surgeon is well advised to obtain consultations with cardiology, nephrology, and endocrinology whenever appropriate.

The vascular evaluation of the diabetic foot requires special attention. Diabetic patients with strongly palpable pedal pulses will usually heal a local foot procedure without difficulty. Patients who have weakly palpable or nonpalpable pulses at the level of the dorsalis pedis or posterior tibial artery require further vascular evaluation in the form of pulse volume recordings or a formal vascular surgery consultation. Lower extremity revascularization is often necessary prior to limb-sparing foot surgery [13]. Patients with autonomic neuropathy however require special mention. These patients will often present with pink, warm skin on the surface of the foot. This can be easily mistaken for a foot with good arterial perfusion even in the presence of critical ischemia (Fig. 17.1).

It has become increasingly important in recent years to obtain a detailed social history. More of the burden for the patient's aftercare is being placed on the patient's family. The majority of patients will require daily dressing changes and prolonged periods of non-weight bearing. For this reason, visiting nurses, home health aides, and physical therapists have become vital members of the multidisciplinary team. In situations where there is less than adequate support for these services at home, admission to a rehabilitative center should be considered. These factors should be identified early in the course of the patient's hospitalization so that discharge planning can proceed in a timely and stress free manner.

Anesthesia Techniques

The presence of profound peripheral sensory neuropathy and the localized nature of many of these procedures make local anesthesia with monitored intravenous sedation ideal for diabetic patients undergoing foot surgery. Epidural or general anesthesia should only be contemplated when more extensive surgery is being considered. This includes most major procedures of the hindfoot and ankle. It should be remembered that either of these techniques increases the perioperative morbidity and mortality. The final choice of anesthesia should be made following discussion with the anesthesiologist and the patient's primary medical doctor and with a clear understanding of the procedure being performed.

Surgical Approach

Prior to definitive surgery or correction of an underlying deformity, the foot must be free of any acute infection. This implies that any area of undrained sepsis has been adequately drained and all necrotic tissue debrided to healthy granular tissue. The proper technique for draining wounds is to incise the wound in such a fashion to promote dependent drainage. As the patient lies recumbent in bed with the extremity elevated, the wound drains from distal to proximal (Fig. 17.2) [14]. Multiple stab incisions with the use of Penrose drains should be avoided as they do not promote dependent drainage. Any tissue that appears infected or necrotic should be sharply excised at this time, including any exposed or infected bone. The wound is then packed widely open and inspected daily for the resolution of sepsis, cellulitis, and the development of healthy granulation tissue. The goal of this initial surgical debridement is to convert an acute infection into a chronic wound. While negative cultures following initial debridement are preferred, it is not a prerequisite for definitive surgery and wound closure as additional surgical debridement is performed at the time of wound closure.

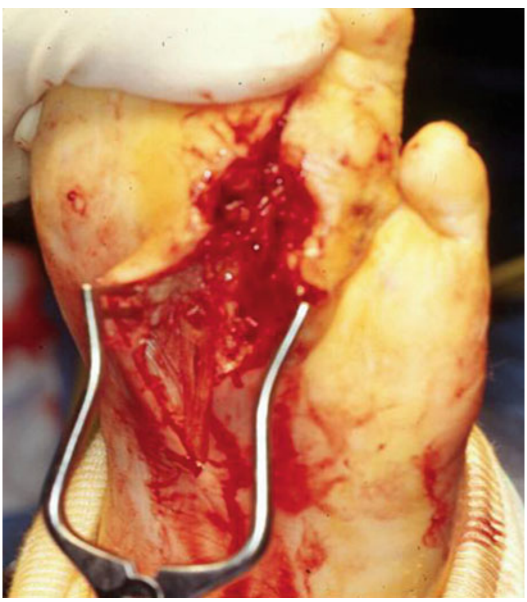

Fig. 17.2 An appropriate incision and drainage of infection should allow dependent drainage as the patient lies recumbent in bed

Forefoot Procedures

First Ray

While there are no studies showing the incidence of ulcers by location, ulcerations of the first ray (hallux and 1st metatarsal) are clearly among the most common ulcers treated. Common sites of ulcerations include the following: (1) plantarmedial aspect of the hallux, (2) distal tip of the hallux, (3) directly plantar to the interphalangeal joint of the hallux, (4) directly plantar to the metatarsophalangeal joint, (5) directly plantar to the first metatarsal head, (6) medial aspect of the first metatarsal head. The primary reason that this area is so susceptible is the combination of increased weight-bearing forces across this joint and faulty biomechanics which can lead to excessive pronation [15–17]. Excessive pronation leads to a medial transfer of the weight-bearing forces through the medial longitudinal arch, the first metatarsal, and ultimately the hallux [18].

Any structural deformity such as osteoarthritis, hallux limitus/rigidus, or severe plantarflexion will increase the susceptibility of this joint to ulceration by altering the biomechanics of the joint. Assessing the underlying structural or mechanical cause of the ulceration is vital to understanding the reasons for ulceration and for selecting the most appropriate procedure.

Ulcerations of the hallux, either plantarmedial or directly plantar to the interphalangeal joint, are most commonly related to abnormal biomechanics

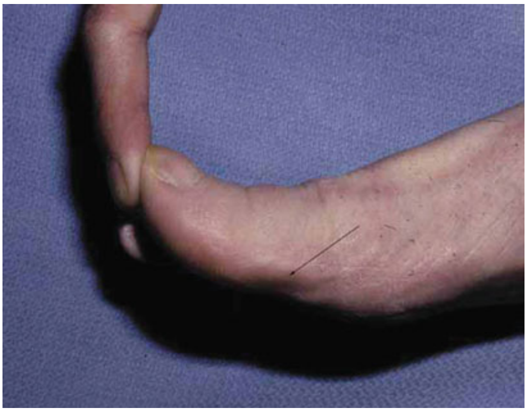

Fig. 17.3 A common location for ulcerations of the great toe is on the plantarmedial aspect of the interphalangeal joint of the hallux. The most common reason for these ulcerations is a hallux limitus

of the first ray resulting from excessive pronation. This is often manifested by the development of callus on the medial aspect of the hallux ("medial pinch" callus) or limitation of motion at the 1st metatarsophalangeal joint (i.e., hallux limitus) (Fig. 17.3). Hyperextension of the interphalangeal joint occurs to compensate for this lack of motion [19, 20]. Other less common causes of ulceration are an enlarged medial condyle on the distal phalanx or the presence of an interphalangeal sesamoid bone, in which case the ulceration is typically directly plantar to the interphalangeal joint.

The surgical treatment of this entity clearly depends on the underlying cause. When the cause of the ulceration is related to lack of adequate motion, restoring motion by way of an arthroplasty of the hallux interphalangeal joint (HIPJ) or of the 1st metatarsophalangeal joint (MTPJ) can be helpful. Resection of the head of the proximal phalanx relieves excessive plantar pressure and allows for resolution of the ulceration [21]. This procedure can also be employed when osteomyelitis of the head of the proximal phalanx is suspected. Occasionally, resection of an enlarged medial condyle can be effective in eliminating the callus. This, however, can result in instability of the joint and development of Charcot joint disease. In cases where there are significant degenerative changes at the level of the 1st MTPJ or complete lack of dorsiflexion, it is best to resect the base of the proximal phalanx and increase motion at this joint.

Surgical treatment of ulcerations directly plantar to the first metatarsal head can be addressed with excision of one or both sesamoid bones. During the propulsive phase of gait, the sesamoids migrate distally and plantarly, thus becoming more prominent. In the intrinsic minus foot, this could serve as a potential pressure point and site of ulceration.

The basic indication for sesamoidectomy is the presence of a chronically, recurrent ulceration directly plantar to the first metatarsal head without clinical or radiographic evidence of osteomyelitis of the 1st metatarsal head [22]. Contraindication for this procedure is the presence of significant degenerative changes of the 1st MTPJ or osteomyelitis of the 1st MTPJ. These are best treated with a Keller or 1st MPTJ arthroplasty. Additionally, the presence of a rigid plantarflexed 1st ray may be a relative contraindication to sesamoidectomy.

When the ulceration is found to extend to the level of the joint, osteomyelitis should be clinically suspected. Treatment must involve complete resection of the infected bone and joint. The procedure of choice is resection of the first MTPJ with excision of the ulceration. Although there may be alternate methods for addressing this problem surgically, there are clear advantages to utilizing this approach rather than allowing the ulcer to heal by secondary intention. By excising the ulceration, all infected, nonviable tissue is removed. It also allows for excellent exposure of all potentially infected tissues, such as the flexor hallucis longus tendon and the sesamoids which are commonly involved. Wounds which are closed primarily heal more predictably and with less scarring. As a rule, these wounds heal in 3–4 weeks. The healing rate of wounds which are allowed to heal by secondary intention is dependent on size and depth. The longer these wounds remain open, the greater the risk of secondary infection, as patient compliance often becomes an issue. While disadvantages exist to closing these wounds primarily, it is our philosophy that the benefits of primary closure outweigh the risks.

Fig. 17.4 The presence of synovial drainage from an ulceration is indicative of joint involvement and requires resection of that joint

Fig. 17.5 Osteomyelitis of the 1st metatarsophalangeal joint is best addressed by elliptical excision of the ulcer with resection of the joint. Adequate resection of the 1st metatarsal should be performed to assure complete eradication of infected bone

The indication for first MTPJ resection with ulcer excision is the presence of an ulcer directly plantar to the first MPJ with direct extension into the joint. This is best determined by the ability to pass a blunt sterile probe through the ulceration and palpate bone. Additionally, the presence of clear, viscous drainage is indicative of synovial fluid. This is an ominous sign, as this can only come from the joint itself (suggesting a tear in the joint capsule) or from the sheath of the flexor hallucis longus tendon (Fig. 17.4). Even in the presence of negative X-rays, this finding is sufficient to make a clinical diagnosis of osteomyelitis [23].

An elliptical incision is made which completely excises the ulceration. It is recommended that the ratio of incision length to width is at least 3:1. This allows the wound to be closed with as little tension as possible. This incision is full-thickness and is carried down to the first metatarsal joint (Fig. 17.5). This should excise all necrotic, infected tissue. At this point, the flexor hallucis longus tendon will be visible. Typically, focal necrosis within the body of the tendon is visualized, indicating infectious involvement. It is therefore best to sacrifice the tendon in order to prevent recurrence of the infection. Removal of the long flexor tendon will often require performing a lengthening procedure of the long extensor tendon on the dorsum of the foot. Failure to perform this could result in an extensus deformity of the great toe, making shoe fit difficult.

Once the tendon is removed, the sesamoids will be visualized. These should also be sacrificed as these are intra-articular structures and are in direct communication with the first MTPJ. The base of the proximal phalanx and the cartilage of the 1st metatarsal head are now resected. While it is preferred to leave as much of the first metatarsal behind as possible to maintain function, enough metatarsal head must be resected so as to remove all focus of osteomyelitis.

The wound is closed by using full thickness nonabsorbable sutures. 2-0 and 3-0 polypropylene (Prolene®) is generally a good choice, as it is nonabsorbable and monofilament. Sutures should be placed evenly and used to coapt skin edges with as little tension as possible. Deep sutures are generally avoided since they can serve as a potential focus of infection and may be difficult to retrieve at a later date if necessary. It is advisable to pack the proximal 1.0 cm of the wound with a

Fig. 17.6 Motor neuropathy is characterized by wasting of the instrinsic musculature in the arch of the foot. This typically results in deformities such as hammertoes, claw toes, or plantarflexed metatarsals

2 × 2 gauze sponge to promote drainage and avoid the development of a hematoma. This is usually removed after 24–48 h. This portion of the wound is then allowed to heal by secondary intention. The postoperative care mandates a period of total non-weight bearing of at least 4 weeks. Early ambulation will result in wound dehiscence, persistent drainage, postoperative infection and possible hypertrophic scar. The sutures are left in place this entire time.

Lesser Digits

Atrophy of the intrinsic muscles of the foot commonly occurs with the development of motor neuropathy. This can result in forefoot deformities such as hammertoes and claw toes (Fig. 17.6) [24]. When sensory neuropathy is also present, ulcerations develop over the proximal interphalangeal joint, at the distal tip of a toe or on adjacent sides of toes. Amputation of a lesser toe rarely results in long-term complications with the exception of loss of the second toe. This can precipitate a hallux valgus deformity. When ulceration is discovered early enough and treated aggressively, amputation of a toe can be avoided, thus maintaining function as well as appearance.

Hammertoes are either classified as reducible or nonreducible. A reducible hammertoe implies the deformity is being held by contractures of the soft tissues while a nonreducible deformity suggests there has been bone and joint adaptation as well as extensive soft tissue contractures. A reducible deformity is often amenable to correction by a tenotomy of the corresponding flexor tendon. A #61 Beaver blade is used to make a small stab incision just proximal to the flexor crease of the affected toe. It is then advanced until the flexor tendon can be palpated. The blade is used to transect the flexor tendon in a transverse direction. The tenotomy is facilitated by applying a gentle dorsiflexory force on the toe. This will put the flexor tendon under tension making it easier to palpate. Upon release of the tendon, the digital deformity can be felt to relax.

A nonreducible deformity requires resection of the phalangeal head as well as a release of the soft tissue. A proximal interphalangeal joint arthroplasty can be combined with excision of the ulcer. In long-standing hammertoe deformities, there may be a concomitant contracture at the level of the metatarsophalangeal joint, often indicative of a subluxation or even a dislocation. When dislocated, an area of high focal pressure can develop on the ball of the foot under the corresponding metatarsal head. This is often manifested as callus or even ulceration. Failure to recognize this fact can lead to incomplete correction of the deformity and failure to resolve the ulceration. The contracture at the metatarsophalangeal joint often requires a tenotomy and capsulotomy of the joint. If the joint cannot be relocated following release of the soft tissue alone, a shortening osteotomy of the metatarsal may be necessary to relocate the joint and relieve the plantar pressure.

Osteomyelitis of the tip of the distal phalanx can often be treated by local excision of the distal phalangeal tuft and primary closure of the ulceration. If, however, there is any concern of residual infection the wound may be left open and closed on a later date.

Lesser Metatarsal Procedures

The area under the lesser metatarsal heads represents the next most common location of diabetic foot ulcerations. Common causes of high foot pressures include abnormal foot mechanics, plantarflexed metatarsals, limited joint mobility, and prior surgical intervention [25–27]. While there are no definitive studies on ulcer incidence and location, it appears that the second metatarsal is more susceptible to ulceration. This is most likely due to the second metatarsal's dependence on the mechanics of the first ray. When excessive pronation of the medial column occurs, there is increased weight transfer and pressure to the lateral metatarsals. This is most often manifested by the development of callus under the 2nd metatarsal head. After the 2nd metatarsal, the typical order of ulcer development is the 3rd metatarsal then the 5th followed by the 4th.

Selection of surgical procedures for ulcerations under the metatarsal heads requires careful evaluation of the ulcer. A critical determinant in the surgical management of these ulcerations is whether osteomyelitis is present.

Lesser Metatarsal Osteotomy

The primary goal of procedures to surgically treat metatarsal head ulcerations is to alleviate areas of high focal pressure. A metatarsal osteotomy can serve as a valuable adjunct in the management and resolution of these ulcerations [28]. The primary indication is the presence of a chronically recurrent ulceration under a metatarsal head without direct extension into bone. An incision is made dorsally over the involved metatarsal. Once the surgical neck is identified, a through and through osteotomy is made. A variety of techniques have been described for this osteotomy. We prefer either the V-type osteotomy with the apex directed toward the joint or the Weil osteotomy with screw fixation. The dorsal to plantar V-osteotomy provides a stable bone cut resistant to medial or lateral dislocation (Fig. 17.7). A small collar of bone can be resected allowing for both shortening and elevation of the metatarsal. This is often desirable when the metatarsophalangeal joint is either subluxed or dislocated. The metatarsal head is then elevated to the same level of the adjacent metatarsals. Fixation of the osteotomy with a .045 Kirschner wire is recommended. However, in the presence of an open ulceration, this is contraindicated. Fixation and stability is achieved by impacting the head onto the shaft. The patient is then maintained non-weight bearing for 4–6 weeks to allow for early bone healing.

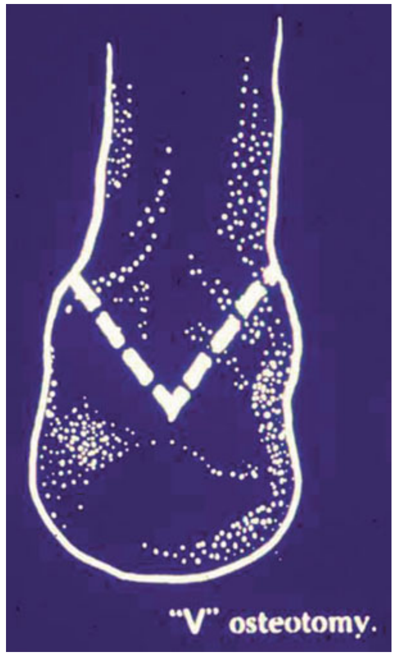

Fig. 17.7 A dorsal to plantar V-osteotomy through the surgical neck of the lesser metatarsal allows for adequate relief of plantar pressure overlying an ulceration. The medial and lateral wings of the "V" decrease the risk of medial or lateral dislocation of the metatarsal head

The Weil osteotomy can also be performed in this clinical situation [29]. In this approach, a 45° osteotomy is created in a dorsal-distal to plantar-proximal direction at the level of the surgical neck. It is then fixated with a single 2.0 cortical screw (Fig. 17.8). The advantage of the Weil osteotomy is that it shortens the metatarsal with little risk of dorsal dislocation. The Weil osteotomy works well in patients with a relatively normal to flatfoot. However, in patients with a rigid anterior cavus foot, the amount of proximal translocation is often not enough to resolve the ulceration. The V-osteotomy is the preferred procedure in this group of patients.

Complications following metatarsal osteotomies include transfer calluses or ulcerations and stress fractures of adjacent metatarsals. These most commonly result when the metatarsal head is elevated above the plane of the adjacent metatarsals. The risk of transfer problems can be reduced if the patient is fitted with an accommodative custom orthosis postoperatively. This will

Fig. 17.8 An alternative osteotomy is the Weil where the bone cut is directed at a 45° angle from a dorsal distal to plantar proximal direction and is fixated with a single 2.0 cortical screw

allow for more even distribution of weight-bearing forces across all metatarsal heads. Shoe gear modification may also assist in this role.

Lesser Metatarsal Head Resection with Ulcer Excision

An alternative approach for relieving plantar pressure is to resect the offending metatarsal head entirely. While this will result in resolution of the ulceration, this carries a high incidence of transfer lesion or ulceration. For this reason, it is preferred to perform this procedure only when osteomyelitis of the metatarsal head is suspected and there is no alternative but to resect the offending metatarsal head.

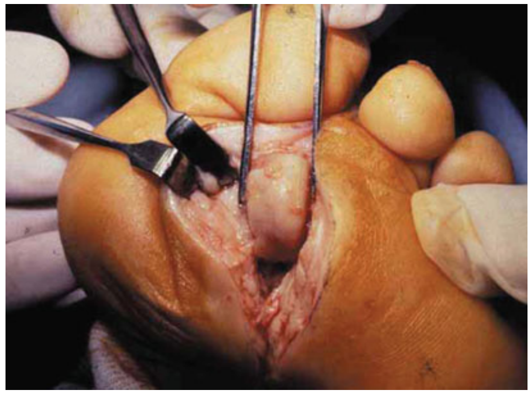

Fig. 17.9 An osteomyelitic lesser metatarsal head can be resected through a plantar elliptical incision excising the ulceration in toto

Resection of the metatarsal head can be approached through a dorsal linear incision centered directly over the metatarsal head. It should be remembered that the base of the corresponding proximal phalanx should also be resected as this structure is contiguous with the metatarsal head and is also involved. The ulcer is then allowed to heal by secondary intention.

An alternate approach is to resect the metatarsal head through a plantar approach while excising the ulceration. The advantage of this approach is that all necrotic and infected tissue can be excised and all tissue be directly inspected (Fig. 17.9). Following resection of the metatarsal head, the wound can be closed primarily as described for 1st MTPJ resection.

The postoperative care requires that sutures are left in place for a minimum of 3 weeks and the patient is kept totally non-weight bearing for 3–4 weeks. The patient is maintained on oral antibiotics until the sutures are removed. Long-term complications include possible transfer lesions or ulcerations and stress fractures due to the altered weight-bearing surface. It is therefore recommended that patients be fitted with an appropriate orthotic device to distribute pressures evenly.

Panmetatarsal Head Resection

Weight-bearing forces are designed to be evenly dispersed across all metatarsal heads. This weight-bearing interdependence between the metatarsal heads has been previously described first by Morton and later by Cavanagh [15, 16]. Disruption of this relationship will alter normal weight distribution. This can occur from trauma to the metatarsals resulting in dorsiflexed or shortened metatarsals as seen in stress fractures, the atrophic form of Charcot joint resulting in dissolution of metatarsal heads or prior surgical resection of metatarsal heads for osteomyelitis.

The recidivistic nature of diabetic foot disease makes multiple metatarsal procedures common in this patient population. Osteomyelitis of multiple metatarsal heads was previously treated by transmetatarsal amputation. This procedure was popularized by Dr. Leland McKittrick of the New England Deaconess Hospital and was responsible for saving thousands of limbs [30]. It is not without its complications however. Ulcerations at the distal stump and equinovarus contractures are common long-term complications (Fig. 17.10a, b). Patients have difficulty psychologically accepting this procedure because it will often require special shoe gear that draws attention to the fact they have had an amputation.

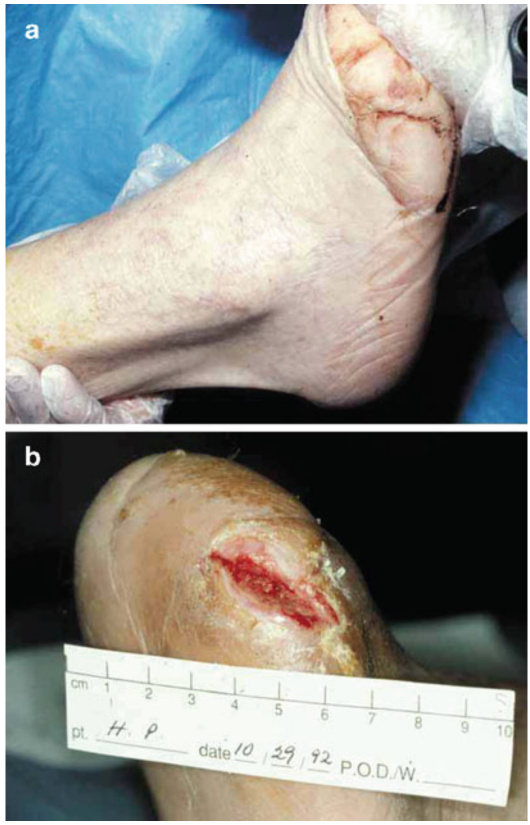

Fig. 17.10 (**a**) A common complication following transmetatarsal amputation is contracture of the Achilles tendon and subsequent equinus deformity. This can lead to characteristic lesions at the distal end of the TMA. (**b**) A distal lateral ulceration of a TMA with an underlying equinovarus deformity

The panmetatarsal head resection and its variations were originally described for the treatment of painful lesions in patients with rheumatoid arthritis [31–34]. Jacobs first described the use of the panmetatarsal head resection for the successful treatment of chronic neuropathic ulcerations [35]. This report was subsequently followed by a report by Giurini et al. where a larger series of patients were studied and an alternate technique was described [34]. Similar success rates were cited. Over the years, the panmetatarsal head resection has replaced the TMA as the procedure of choice in patients with recurrent ulcerations following prior surgical resection of metatarsal heads [36, 37].

The primary indication for the panmetatarsal head resection is the presence of chronically recurrent neuropathic ulcerations on the plantar aspect of the foot following prior metatarsal head resections or ray amputations. It is our belief that if two or more metatarsals have already been resected or need to be resected to eliminate osteomyelitis, the patient would be best served by a panmetatarsal head resection (Fig. 17.11). At first, this may appear to be a drastic, aggressive approach. However, experience has shown that this approach may actually spare patients additional trips to the operating room for transfer ulcerations.

Various surgical approaches have been described for the panmetatarsal head resection. Dorsal approaches, plantar approaches or a combination of the two have been performed with equal success [38]. When possible the preferred approach is the four incision dorsal approach: one incision directly over the 1st metatarsal, one

Fig. 17.11 Prior resection of two metatarsal heads and the presence of osteomyelitis of a remaining metatarsal head is indication for panmetatarsal head resection

between the 2nd and 3rd metatarsals, one directly over the 4th metatarsal, and one directly over the 5th metatarsal. This approach has the following advantages: allows adequate exposure of all metatarsal heads, decreases the potential for retraction injury on the skin edges, and maintains adequate skin islands so as not to affect vascular supply. Because the primary indication for this procedure is the presence of an open ulceration with osteomyelitis, the most common approach is to combine a dorsal incision with a plantar incision which excises the ulceration. The plantar wound and all necrotic tissue can then be excised, the involved metatarsal head(s) can be resected and the wound closed primarily as previously described.

The surgical technique for resection of the metatarsal heads has already been described. The most important technical point to remember in performing this procedure is to maintain the metatarsal parabola. This typically means that the 1st and 2nd metatarsals are left approximately the same length while the 3rd, 4th, and 5th metatarsals are each successfully shorter. Failure to maintain this relationship can lead to recurrent ulceration and additional surgery. If a prior metatarsal head resection or ray amputation has already been performed, then a perfect parabola may not be achievable. In that case, the metatarsal parabola should be recreated with the remaining metatarsals. Additionally, the extensor tendons are identified and are retracted. This will maintain the function of these tendons during the gait cycle affording this procedure the prime advantage over the TMA.

Midfoot Procedures

Surgery in the region of the midfoot is most commonly necessary following foot deformities resulting from neuroarthropathic (Charcot) joint disease. The most common location of Charcot joint involves the tarsometatarsal (Lisfranc's) joints but other joints in the midfoot may also be affected [39, 40]. Instability of Lisfranc's joint often results in a rocker-bottom deformity of the midfoot with plantarmedial ulceration. This is primarily due to subluxation of the 1st metatarsal and medial cuneiform creating a plantar prominence. Ulcerations on the plantar and lateral aspect of the foot are not uncommon. These result from plantar extrusion of the cuboid from a Charcot process at the calcaneocuboid joint [41]. These pose a significant management problem, as they are typically recalcitrant to conservative measures. There is no single surgical procedure that can be applied to all ulcers in this location. Therefore, a flexible approach to these lesions is required. Surgical approaches may involve simple ostectomy with or without fasciocutaneous flap or primary arthrodesis of unstable joints.

Ostectomy

This is the simplest approach to chronic plantar ulcerations of the midfoot. This is reserved for those deformities that have their apex directly plantar to the 1st metatarsal-medial cuneiform joint and where the midfoot is not hypermobile.

The depth of the ulceration will dictate the best surgical approach. A direct medial incision which is centered over the joint is preferred when the ulceration is superficial and not involving bone. This will allow for excellent visualization of the joint and the prominent bone. The prominence can then be resected from medial to lateral either with an osteotome or with a saw. The goal should be to remove an adequate amount of bone to alleviate the plantar pressure and not create a new bony prominence which could create a new source of irritation and ulceration, thus negating the benefits of this procedure.

Ulcerations which communicate with bone and show signs of osteomyelitis clinically are best managed by excision of the ulceration with bone resection and primary closure of the ulceration. In addition to removing the infected bone, the ability to close the ulceration primarily without tension is an additional goal. This approach can be used when the ulcer is located either plantar central or plantar lateral in the midfoot. The most likely etiology for these ulcerations is plantar displacement of the cuboid. When the ulceration measures less than 2.5 cm, this surgical approach can be used. The use of closed suction irrigation is also recommended in order to prevent hematoma formation which can lead to wound dehiscence or infection.

One of the more difficult ulcerations to manage is an ulcer located centrally in the midfoot secondary to plantar subluxation of the cuboid bone. This is the type 5 of the Harris and Brand classification of Charcot joint disruption (pattern II in the Sanders classification) and has been described as being very resistant to conservative care [39, 40]. Resolution of these ulcerations often requires surgical intervention of some type.

Exostectomy with Fasciocutaneous Flap

When ulcerations of this type exceed 2.5 cm in diameter, primary excision with closure is often not possible and an alternate technique must be sought [41]. Ulcerations of this size are typically excised circumferentially to the level of the cuboid bone. This will allow removal of all necrotic, infected tissue as well as any hyperkeratotic margins bordering the ulcer. The joint capsule and periosteum of the cuboid are next encountered which are reflected off the underlying cuboid. This will expose the peroneal groove of the cuboid bone. The peroneus longus will often be found running in the groove. When possible this should be retracted so as to protect it from inadvertent injury. On rare occasions, however, it may be necessary to sacrifice the peroneus longus in order to gain adequate exposure of the bony prominence. The peroneal groove is next resected with the use of an osteotome and mallet. Once completed, the wound should be carefully inspected for any remaining bony prominence or bone spicules which can serve as a new point of pressure and possible ulceration.

Fig. 17.12 The flexor digitorum brevis muscle is commonly used for closure in large plantar ulcerations following ulcer excision and exostectomy of the offending bone

This procedure will often leave a relatively large dead space which can serve for the collection of a hematoma. It is best to fill this dead space with a muscle flap which will serve two purposes: (1) it will decrease the dead space following the bony resection; (2) it will provide a layer of soft tissue between the underlying bone and the overlying skin (Fig. 17.12). The flexor digitorum brevis muscle is well suited for this purpose because of its anatomic location and ease of dissection. The muscle is rotated laterally to cover the cuboid. A full thickness fasciocutaneous flap which is based on the medial plantar artery is then rotated from medial to lateral to cover the actual ulcer site. A split thickness skin graft is then used to cover the donor site in the medial arch (Fig. 17.13).

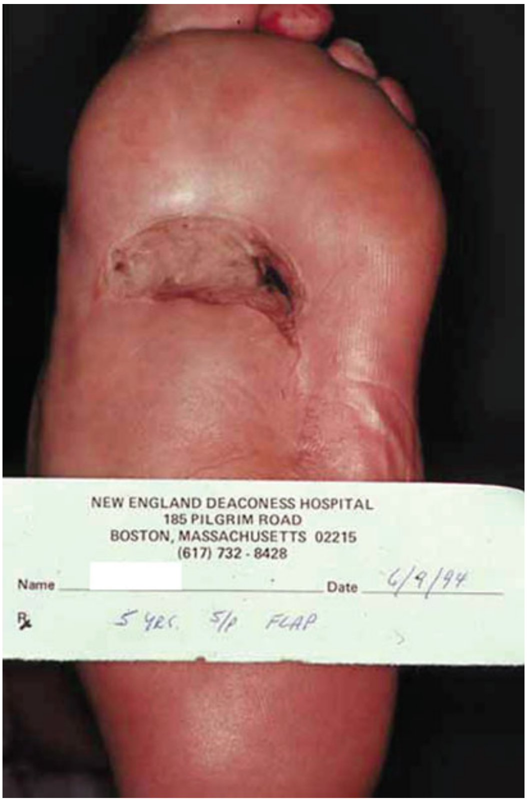

Fig. 17.13 A patient who is 5 years status post cuboid exostectomy with an interpositional muscle flap and a rotational fasciocutaneous flap

Six weeks of total non-weight bearing is required for adequate healing and incorporation of the flap. This is followed by an additional 2–4 weeks of protected weight bearing in a surgical shoe with a molded orthotic device. Long-term care will require the use of plastizote orthoses and modified shoe gear.

Advancement or rotational flaps of the foot have become relatively infrequent since the introduction of negative pressure wound therapy (NPWT), also referred to as vacuum assisted closure (VAC). Negative pressure wound therapy was first introduced in the United States in 1997 [42, 43]. Since its introduction, it has been extensively used in large circumference wounds with significant depth in order to promote granulation, decrease the number of dressing changes and avoid more extensive and morbid procedures. As a result, there has been a significant reduction in the number of rotational flaps or free tissue transfers needing to be performed. Newer designs of NPWT have concentrated on making the device smaller and more portable [44].

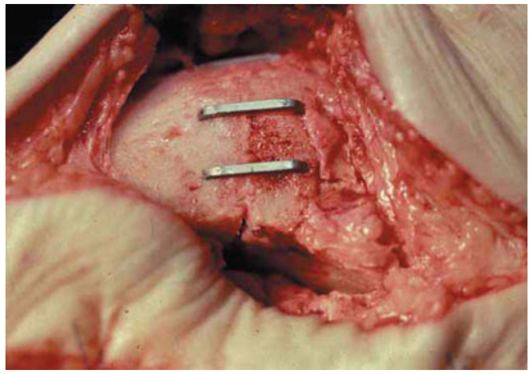

Fig. 17.14 Fusion of the 1st metatarsal-medial cuneiform joint for an unstable Charcot joint complicated by recurrent ulceration can be achieved by use of staples

Medial Column Fusion

When resolution of the Charcot joint process has resulted in significant bone loss such that there is significant instability at the 1st metatarsal-medial cuneiform joint, primary fusion of this joint should be considered. Simple exostectomy in the presence of instability will often fail due to continued collapse of this segment, resulting in a new bony prominence. Stabilization of this joint therefore is the better alternative.

The joint can be approached surgically through a direct medial incision. This will afford adequate exposure of the dorsum of the joint as well as the plantar surface. The articular cartilage on both sides of the joint is resected with a sagittal saw. It is recommended that the bone cut on the 1st metatarsal side be slightly angulated from dorsal-proximal to plantar-distal. This will plantarflex the 1st metatarsal slightly, restoring the weight-bearing function of the first ray. In addition, it is recommended that any plantar bony prominence also be resected from medial to lateral.

Fixation of the joint can be achieved in a variety of ways. While crossed .062 Kirschner wires and staples are acceptable means of fixation, the authors prefer either a medial plate with an interfragmentary screw or crossed screws to provide rigid internal fixation and compression (Figs. 17.14

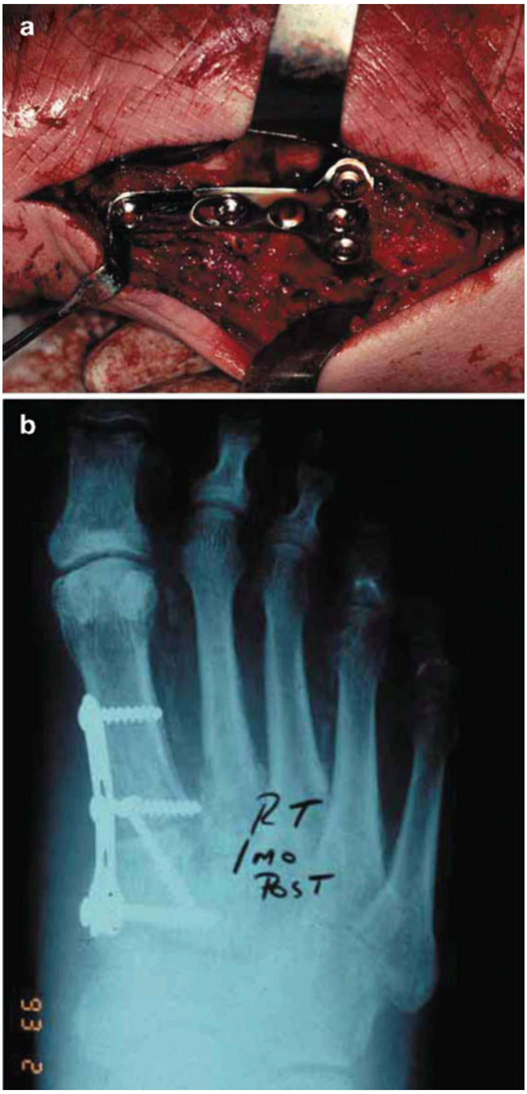

Fig. 17.15 (**a**) A T-plate with an interfragmentary screw is another acceptable form of fixation of the 1st metatarsal-medial cuneiform joint in the presence of unstable Charcot joint. (**b**) Radiograph of patient with T-plate and interfragmentary screw across the 1st metatarsal-medial cuneiform joint

and 17.15a, b). Recently we have been using an intramedullary rodding technique which will be described later in this chapter. It is advisable to insert a Jackson-Pratt drain to prevent the accumulation of an hematoma.

The postoperative course requires immobilization and non-weight bearing. While there is no standard length of immobilization and non-weight bearing, the patient can expect to be non-weight bearing on average 3 months. Partial weight bearing may begin when serial X-rays show early trabeculation across the 1st metatarsal-medial cuneiform joint. Continued resumption of weight bearing is allowed as long as both clinical and radiographic evaluations suggest continued healing of the fusion site.

Charcot joint disease can also affect the entire Lisfranc's joint complex, i.e., all five tarsometatarsal joints. This is commonly referred to as multiarticular involvement. In the case of severe midfoot instability stabilization of the entire midfoot may be necessary. These procedures will be covered below under hindfoot procedures.

Hindfoot Procedures

Surgical procedures of the hindfoot are most commonly performed for reconstruction of unstable Charcot joint disease and can be truly classified as limb salvage procedures. These include partial or subtotal calcanectomy, tendo-Achilles lengthening, triple arthrodesis, and pantalar arthrodesis. We will also include multisegmental midfoot arthrodeses.

Indications for these reconstructive procedures include chronic, nonhealing ulcerations with underlying hindfoot deformity or instability, severe instability of the hindfoot making ambulation difficult at best, or chronic heel ulcerations with underlying osteomyelitis. Because of the high-risk nature of these procedures, all conservative measures should be attempted prior to intervening surgically or when the only alternative is a major limb amputation.

Calcanectomy

Heel ulcerations a common event in patients with diabetes. Due to the comorbid conditions most diabetic patients display, periods of prolonged bedrest is not unusual. Without proper protection decubitus ulcerations can occur. However other causes for heel ulcers include blisters from shoe or cast irritation and heel fissures resulting from dry skin or puncture wounds. Regardless of the precipitating cause, the end result is prolonged disability and morbidity. In cases of bone involvement (i.e., osteomyelitis), below knee amputation can be the final outcome. Attempts to save this extremity to provide a limb capable of functional ambulation can involve excision of the ulceration and the calcaneus, either partial or subtotal.

The goals of the calcanectomy should include excision of all necrotic and infected soft tissue, resection of any and all infected bone and primary closure of the wound whenever possible. Additional bone resection may be necessary in order to achieve primary closure. Hindrances to primary closure can include the lack of mobility of the surrounding soft tissue and severe tissue loss from infection. In these cases, a more creative approach may be necessary. This can include rotational skin flaps, free tissue transfers, or NPWT.

The majority of times this procedure is performed for osteomyelitis. It is therefore critical that adequate bone is removed to eliminate the infection. It is also important that no plantar prominence be left behind which could serve as an irritant to the soft tissue and result in ulceration. In resecting the calcaneus the Achilles tendon is often encountered. Depending on the extent of infection, it may need to be debrided or even released. While one may be tempted to reattach the tendon, it is rarely advisable to do so. Advancement of the Achilles tendon would require the introduction of foreign materials such as screws or anchors which could serve as a nidus of recurrent infection. In those cases where the Achilles tendon is detached, it will often fibrose to the surrounding tissues and provide some degree of plantarflexion (Fig. 17.16a, b).

Fig. 17.16 (**a**) Osteomyelitis of the calcaneus with resultant soft tissue loss is a common cause of lower limb amputation. (**b**) Same patient following partial calcanectomy with excision and debridement of infected, necrotic tissue and primary closure. Successful eradication of infected bone resulted in limb salvage

Tendo-Achilles Lengthening

Over the past 5 years there has been increasing literature on the effect of a tight Achilles tendon on foot ulcerations and Charcot joint disease [45]. It has been well documented that patients with diabetes develop increased glycosylation of skin and soft tissue structures, including tendons [27]. This then leads to increased plantar foot pressures. A tight Achilles tendon from enzymatic glycosylation has been implicated as a contributing factor not only in forefoot ulcerations but in the development of Charcot joint disease. Whether the Achilles tendon becomes tight as a result of the Charcot joint disease or a tight Achilles tendon contributes to the development of Charcot joint disease remains a matter of great discussion. In the end, all authors agree that a tight Achilles tendon contributes to the recurrent nature of these problems and should be addressed via tendon lengthening [46–48].

There are several techniques to lengthen the Achilles tendon. These can be classified as either open or percutaneous [49]. The simplest and least morbid technique is the percutaneous approach. This technique uses three small stab incisions and minimal soft tissue dissection. However, this requires an understanding of the anatomy of the Achilles tendon and the ability to convert to the open technique when necessary. The Achilles tendon is formed by the end fibers of the gastrocnemius and soleus muscles and insert into the dorsal posterior aspect of the calcaneus. By virtue of its insertion, the Achilles tendon functions as a strong plantarflexor at the ankle joint and invertor of the subtalar joint. Its plantarflexion motion is opposed by the extensor muscles crossing the anterior aspect of the ankle joint and its inversion motion is opposed by the peroneal muscles laterally. When the calcaneus everts and the axis of the subtalar joint changes as occurs in patients who excessively pronate, the axis of pull of the Achilles tendon also changes [18]. It now creates a strong pronatory force of the foot. This can lead to excessive medial transfer of weight and midfoot collapse as seen in Charcot joint disease. It is for this reason that the Achilles tendon must be evaluated in every case of Charcot joint disease and reconstructive surgery. Failure to recognize this fact can lead to recurrence of the ulceration and failure of the reconstruction.

The percutaneous technique is the simplest, least morbid procedure to lengthen the Achilles tendon. The procedure can be approached with the patient lying either supine or prone. Three small stab incisions are made centrally on the Achilles tendon (Fig. 17.17). The incisions will be spaced approximately 1.5–2.0 cm apart with the distal most incision being 1.5 cm from the insertion of the Achilles on the calcaneus. The most proximal and most distal incisions will incise the Achilles centrally and exit laterally while the middle incision will incise the Achilles centrally and exit medially. Once the three incisions are completed, a gentle dorsiflexion force is exerted on the foot until a gentle stretch can be felt on the Achilles. Care should be taken not to stretch the Achilles beyond 10° of dorsiflexion. The skin incisions are then closed with suture of the surgeon's choice.

While the percutaneous technique provides adequate correction and is the least morbid technique, there are situations where greater degrees of correction are needed. This is where the open technique may be needed. This is best performed with the patient prone. An approximately 8–10 cm incision is made along the central portion of the Achilles tendon. The incision is deepened until the peritenon is visualized. The peritenon is incised longitudinally along the line of the skin incision exposing the Achilles tendon. While there have been several ways described to lengthen the tendon, our preferred method is to make one incision approximately 1.0 cm proximal to the insertion. The blade is inserted into the midsubstance of the Achilles all the way across and the anterior fibers are transected. Attention is then directed approximately 2.5–3.0 cm proximally where the blade is once again inserted into the midsubstance of the Achilles tendon. The posterior fibers of the tendon are now transected. Once completed, the foot is once again gently dorsiflexed until the tendon can be seen to lengthen along the central intact fibers (Fig. 17.18). In this fashion, the surgeon can visualize the amount of lengthening achieved and

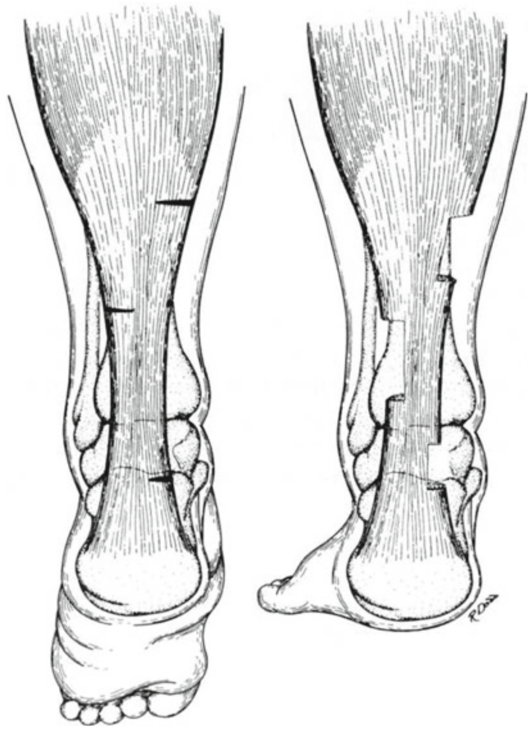

Fig. 17.17 The percutaneous technique uses two medial stab incisions and one lateral incision. The ankle is dorsiflexed to allow for lengthening of the Achilles tendon

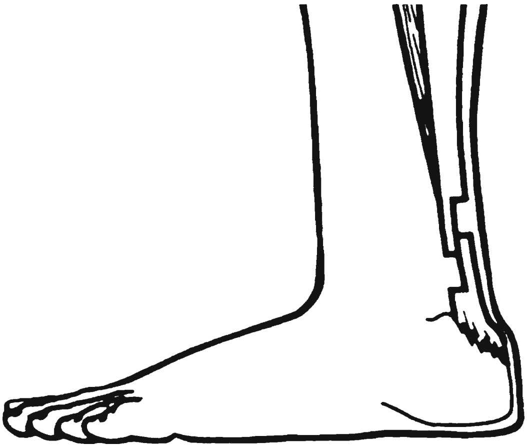

Fig. 17.18 The open Achilles tendon lengthening creates incisions proximally and distally. The tendon is then lengthened in the frontal plane

Fig. 17.19 The intramedullary rodding technique introduces large diameter screws through the metatarsals and across the hindfoot joints to achieve stability, primary fusion and deformity correction

"dial-in" more dorsiflexion if necessary and if feasible. Closure of the wound, including the peritenon, is performed in a layered fashion. The Achilles tendon lengthening is protected for approximately 6 weeks in a splint or brace that maintains the ankle joint at 90°.

Midfoot Arthrodesis

The most common location for Charcot joint disease is the tarsometatarsal joints, i.e., Lisfranc's joints. These are the joints formed by the metatarsal bases and the cuneiforms and cuboid bone. These joints are supported by several small ligaments that connect these bones to each other. While the inciting event for the development of Charcot joint disease remains unclear, in the majority of cases disruption of these ligaments with or without fractures is a common feature. Because of absence of pain, the patient continues to ambulate on this unstable foot resulting in further destruction, displacement and instability. The end result is a foot that is grossly misshapened, unstable to walk on, and at risk for ulceration, infection, and amputation. While initial treatment should consist of non-weight bearing, immobilization, and bracing, many feet are so unstable that bracing actually poses a risk to the patient. It is in these cases that surgical intervention should be contemplated.

Earlier in this chapter, we described one technique of surgical reconstruction consisting of medial column fusion, either with screws or plates. This works well when destruction is limited to the 1st metatarsal-medial cuneiform joint. However, when the entire midfoot is involved and there is dorsal displacement of the midfoot on to the hindfoot, a more aggressive approach relocating the entire midfoot is needed [50, 51]. Over the past 5 years we have employed a technique where the medial and lateral columns of the midfoot are rodded with large screws that are inserted through the intramedullary canals of the 1st metatarsal and the 4th metatarsal (Fig. 17.19). These screws cross the tarsometatarsal joints into the respective tarsal bones. In those cases where the talonavicular joint is also involved, a single long screw can be used to cross both the 1st metatarsal-medial cuneiform joint and the talonavicular joint as part of a triple arthrodesis. The screws are inserted following appropriate resection and realignment of the involved joints.

This intramedullary rodding technique has the advantage of providing adequate realignment and compression of the affected joints. This is a very stable construct. The other advantage is it avoids excessive dissection of the joints. With the introduction of cannulated screws and using intraoperative X-rays, these screws can be accurately placed through small stab incisions, avoiding large wounds and excessive stripping of the periosteum which can compromise healing of these fusion sites.

Triple Arthrodesis

The incidence of Charcot joint disease involving the tarsal joints—talonavicular, calcaneocuboid or subtalar—ranges from 1.8 to 37% depending on the reports [52–54]. Clinically, these feet may appear with a rocker-bottom deformity from plantar subluxation of the talonavicular joint or the calcaneocuboid joint. This can then lead to chronic ulceration. When faced with a significant degree of instability from this destructive process, the approach should include surgical stabilization of the involved joint or joints. This often requires fusion of the talonavicular joint, calcaneocuboid joint, and the subtalar joint, i.e., triple arthrodesis.

The goal of a triple arthrodesis is to stabilize the foot and to reduce the deformity, thereby reducing the risk of recurrent ulceration. The surgery should be delayed until the acute phase has resolved and the Charcot joint has entered the coalescent phase. If an open ulceration is present, surgery should be delayed until all signs of acute infection are resolved.

The triple arthrodesis is performed in a standard fashion [55]. The calcaneocuboid joint is approached through a lateral incision just inferior to the lateral malleolus and extending distally to the base of the 4th and 5th metatarsals. While it is possible to obtain adequate exposure of the talonavicular joint through this incision, one should not hesitate to make a separate incision medially if this affords better exposure.

The cartilage is resected off all joint surfaces until bleeding bone is exposed. The joints are then reapproximated. If significant deformity exists, wedge resections through the joints may be required to adequately reduce the deformity. Additionally, significant bone resorption may have occurred as a result of the destructive process. In these cases, bone graft may be necessary to fill the gaps between joint surfaces. This can be obtained from the iliac crest or from the bone bank.

The method of fixation is the surgeon's choice. Typically, the posterior subtalar joint is fixated with a 6.5-mm cancellous screw. This screw can be introduced from a dorsal approach through the talar neck or a small stab incision can be made on the plantar surface of the heel. The screw is then inserted from plantar to dorsal, across the subtalar joint into the body of the talus. This latter technique is preferred. While screws are preferred for the talonavicular and calcaneocuboid joints, staples can also be used. Recently, a small claw plate has been introduced to arthrodese the calcaneocuboid joint. Adequate apposition of joints and accurate placement of fixation devices is achieved by the use of intraoperative X-rays. The goal of surgery is correction of the deformity with good apposition of all joint surfaces. Minimal to no gapping should be present. This should always be confirmed with a final intraoperative X-ray to confirm the final position of all fixation devices, adequate joint apposition and appropriate foot position. The position of the calcaneus should be neutral to slight valgus.

Postoperatively, the patient is initially placed in a posterior splint to immobilize the fusion site. This is replaced with a below the knee fiberglass cast usually 4–5 days following surgery. Total non-weight bearing is maintained for a minimum of 3–4 months. Serial X-rays are obtained to evaluate bone healing and maintenance of postoperative correction and alignment. The patient is then advanced to gradual protected weight bearing when X-rays show signs of bone union. Case reports suggest that the likelihood and rate of fusion may be improved with the use of electrical bone stimulation, although prospective, randomized, double-blinded trials are needed to determine overall efficacy [56].

Pantalar Arthrodesis

The ankle joint that has undergone severe destruction from Charcot joint disease is

Fig. 17.20 Severe instability of the rearfoot due to Charcot joint often requires major reconstructive surgery of the hindfoot and ankle. A pantalar fusion was performed in this patient for severe cavoadductovarus deformity and chronic ulceration resulting from Charcot joint. Two 7.0 mm cannulated screws were used to fuse the subtalar and ankle joints

particularly problematic. This typically will result in an ankle joint so flail that it makes ambulation extremely difficult if not impossible. This deformity may result from total collapse of the talar body, fractures through the medial malleolus, lateral malleolus, or both. Patients with these types of fractures will often be found ambulating directly on either the medial or lateral malleolus. This inherent instability will result in the development of chronic ulcerations and are extremely difficult to control with conservative care alone. The prognosis for these deformities is poor. In order for limb salvage to be achieved, primary fusion of the ankle and subtalar joints is necessary.

The surgical approach depends on the level and degree of destruction. If the primary level of instability and destruction involves the tibiotalar joint, isolated fusion of this joint may be sufficient. However, if destruction of the other rearfoot joints is present, then fusion of the ankle, talonavicular, subtalar, and calcaneocuboid joints (i.e., pantalar fusion) should be performed. All surgical intervention should be delayed until all signs of acute Charcot joint disease have resolved. Attempted fusion during the active, hyperemic phase of this disorder not only will make fusion technically difficult but may also result in failure to fuse.

A lateral incision which begins approximately at the midfibula and extends to the tip of the lateral malleolus offers adequate exposure of the ankle joint. If a pantalar fusion is to be performed, this incision can be extended distally to the calcaneocuboid joint. The fibula is typically osteotomized just proximal to the ankle joint line. The anterior aspect of the fibula is dissected free and reflected posteriorly. This preserves the vascular supply to the fibula. This will allow the fibula to be used as a vascularized strut graft on the lateral side of the ankle joint. The ankle joint is now well visualized.

The articular cartilage is resected down to bleeding cancellous bone from the inferior surface of the tibia and the dome of the talus. The ankle joint is repeatedly manipulated so as to assess alignment of the foot. The joint surfaces are continually remodeled until optimal bone apposition and foot alignment is achieved. In cases where the talar body is deemed nonsalvageable, a femoral head allograft has been used to fill in any defect or accommodate for significant bone loss. If a pantalar fusion is being performed, the remaining hindfoot joints can be addressed at this time in the same manner as in a triple arthrodesis.

After all articular surfaces have been resected, the foot should be positioned so that all bone surfaces are in good apposition with minimal to no gapping. Care should also be taken to avoid any interposition of soft tissue. If the foot cannot be aligned properly or bone surfaces do not appose adequately, further remodeling of the bone should be performed. Once optimal alignment has been achieved, the ankle joint is ready for fixation. Internal fixation of the ankle joint can take one of two forms. This can be performed with the introduction of two 7.0 mm cannulated screws. These are typically inserted from a plantar to dorsal direction through the body of the calcaneus and across the resected ankle joint. This will also fixate the posterior subtalar joint (Fig. 17.20). Ideally, the tips of the screw should purchase the cortex of the tibia. An alternate technique is the use of a retrograde intramedullary nail which is also introduced across the ankle and subtalar joints from a plantar approach

Fig. 17.21 X-ray showing Charcot ankle reconstruction using an intramedullary nail and femoral head allograft

(Fig. 17.21). When bone quality precludes the use of internal fixation, external devices for fixation are appropriate alternatives. The use of intraoperative imaging is critical in the placement of guide wires and for final fixation. It is critical that the calcaneus be positioned either in neutral or in slight valgus position. Any degree of varus should be avoided. After fixation of the ankle joint, the remaining rearfoot joints can be fixated as previously described.

As with triple arthrodesis, the postoperative care is critical to successful limb salvage. Wound infection, dehiscence, and nonunion are the major complications seen with this procedure. Immobilization of the extremity immediately postoperatively can decrease the risks of these complications. Total non-weight bearing in a fiberglass below the knee cast is required for a minimum of 4–6 months. The fusion site must be protected with cast immobilization and casts must be changed frequently to prevent abrasions or cast irritations. Once it is felt fusion is sufficient to support weight bearing, this should be instituted in a gradual protected manner. A return to protected weight bearing will be dictated by serial X-rays. The use of adjunctive modalities to promote fusion, such as electrical bone stimulation, should be considered in this patient population as these patients and procedures are considered at high risk for nonunion.

Arthrodesis with External Fixation

The complex nature of these deformities has recently required utilization of recent advances in external fixation [57–60]. As previously stated, the degree of bone loss in these hindfoot deformities will often not allow for dependable use of internal fixation devices. In addition, the presence of an open ulceration and osteomyelitis makes the use of internal fixation contraindicated. It has therefore become necessary to use various external fixation constructs to achieve stabilization of these deformities without the inherent risks of internal fixation [61]. The most common construct utilizes a combination of multiplane fine wire ring fixators, half pins, and foot plate attached to the leg and foot at different levels [62]. If possible, this can be used in conjunction with internal fixation (Fig. 17.21).

Resection of joints and devitalized bone is performed as previously described. The resected joints can be wedged to allow for as near anatomic alignment as possible. The use of bone graft is often necessary for proper alignment and to make up for large defects. Once the foot is reduced into an anatomic alignment, a series of thin wires are placed proximal and distal to the osteotomy. The proximal wires are generally inserted through the calcaneus and the talus, while the distal wires are generally passed through the metatarsal shafts. These wires will then be connected to the foot plate and tensioned. This will provide compression across the osteotomy site, whether it is in the midfoot or hindfoot. Additional wires and ring fixators are inserted through the distal tibia. The rings are then connected to each other by a series of bolts. This configuration will then provide increased stability and rigidity of the lower extremity. There are times when the external fixator is used in combination with an intramedullary rodding technique. While this provides a very stable construct, the insertion of the skinny wires is more challenging and critical.

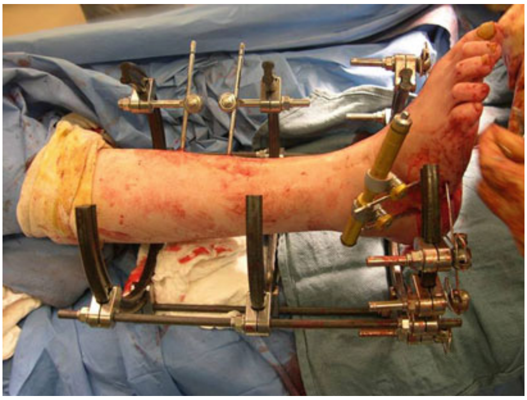

Fig. 17.22 Severe Charcot deformity with an open ulceration and osteomyelitis will require the use of external fixation to correct the deformity and to avoid the use of internal fixation at the site of ulceration and osteomyelitis

The advantage of this technique is that it allows the reconstruction of severe deformities where there have been large degrees of bone loss and large degrees of instability. The thin wires are used to bridge these defects and insert the fixation into better quality bone. This technique can also be used in the face of open ulcerations or where osteomyelitis had been present since the external wires can be inserted at sites remote from the site of ulceration and infection (Fig. 17.22).

The major disadvantages of this technique are the bulkiness of the device itself, the risk of pin tract infections and the inability to apply a compression dressing leading to significant edema. The external fixator is usually in place for approximately 3 months. During this time, the patient finds it difficult to be mobile. It can also interfere with sleep and irritate the contralateral extremity. Showering is also a problem. The exposed wires are also vulnerable to bending and irritation of the surrounding skin, making pin tract infections possible. It is therefore important that meticulous pin care is performed (Fig. 17.23a, b).

In spite of these disadvantages, many patients will choose to undergo this extensive procedure, as the only other option is a major limb amputation.

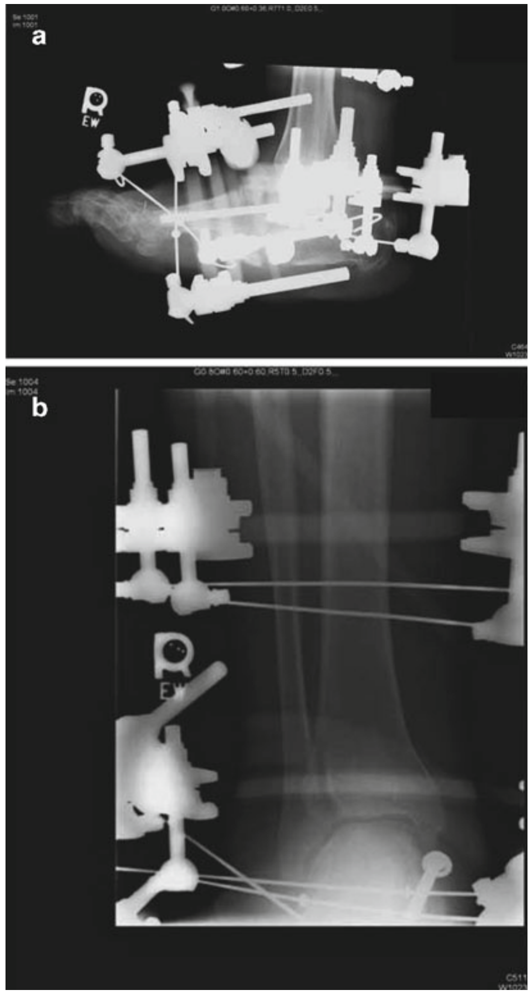

Fig. 17.23 (**a**) Charcot reconstruction demonstrating correction with combination of internal and external fixation to address midfoot and hindfoot deformities (*lateral view*). (**b**) External ring fixator using thin wire technique (*AP view*)

References

1. Jiwa F. Diabetes in the 1990s – an overview. Stat Bull Metrop Insur Co. 1997;78:2–8.
2. Center for Disease Control and Prevention. National diabetes fact sheet, 2007. Atlanta, GA: U.S. Dept. Health and Human Services; 2007.
3. Boyle JP, Thompson TJ, Gregg EW, Barker LE, Williamson DF. Projection of the year 2050 burden of diabetes in the US adult population: dynamic modeling of incidence, mortality, and prediabetes prevalence. Popul Health Metr. 2010;8(29):1–12.

4. Reiber GE, Boyko EJ, Smith DG. Lower extremity foot ulcers and amputations in diabetes. Diabetes in America. 2nd ed. Bethesda, MD: National Institutes of Health; 1995. p. 409–28. NIH Publication No. 95–1468.
5. Tennvall GR, Apelqvist J. Health-economic consequences of diabetic foot lesions. Clin Infect Dis. 2004;39 Suppl 2:S132–9.
6. Centers for Disease Control and Prevention. Diabetes surveillance, 1993. Atlanta, GA: U.S. Dept. Health and Human Services; 1993. p. 87–93.
7. Diabetes and chronic disabling conditions. In: Healthy people 2000. US Department of Health and Human Services, Public Health Service. DHHS publ. No. PHS 91-50212; 1991. p. 442.
8. Edmonds ME, Blundell MP, Morris HE, Maelor-Thomas E, Cotton LT, Watkins PJ. Improved survival of the diabetic foot: the role of the specialist foot clinic. Q J Med. 1986;232:763–71.
9. Thomson FJ, Veves A, Ashe H, Knowles EA, Gem J, Walker MG, Hirst P, Boulton AJM. A team approach to diabetic foot care – the Manchester experience. Foot. 1991;1:75–82.
10. Frykberg RG. Diabetic foot ulcerations. In: Frykberg RG, editor. The high risk foot in diabetes mellitus. New York: Churchill Livingstone; 1991. p. 151.
11. Caputo GM, Cavanagh PR, Ulbrecht JS, Gibbons GW, Karchmer AW. Assessment and management of foot disease in patients with diabetes. N Engl J Med. 1994;331:854–60.
12. Gibbons GW. Vascular evaluation and long-term results of distal bypass surgery in patients with diabetes. Clin Podiatr Med Surg. 1995;12:129–40.
13. Roseblum BI, Pomposelli Jr FB, Giurini JM, Freeman DV, Chrzan JS, Campbell DR, Habershaw GM, LoGerfo FW. Maximizing foot salvage by a combined approach to foot ischemia and neuropathic ulceration in patients with diabetes; a 5-year experience. Diabetes Care. 1994;9:983–7.
14. Gibbons GW. The diabetic foot: amputations and drainage of infection. J Vasc Surg. 1987;5:791–3.
15. Morton DJ. The human foot. New York: Columbia University Press; 1935.
16. Cavanagh PR, Rodgers MM, Iiboshi A. Pressure distribution under symptom-free feet during barefoot standing. Foot Ankle. 1987;7:262–76.
17. Ctercteko GC, Chanendran M, Hutton WC, Lequesne LP. Vertical forces acting on the feet of diabetic patients with neuropathic ulceration. Br J Surg. 1981; 68:608–14.
18. Root M, Weed J, Orien W. Normal and abnormal function of the foot. Los Angeles: Clinical Biomechanics Corp; 1977. p. 211.
19. Dannels E. Neuropathic foot ulcer prevention in diabetic American Indians with hallux limitus. J Am Podiatr Med Assoc. 1989;76:33–7.
20. Downs DM, Jacobs RL. Treatment of resistant ulcers on the plantar surface of the great toe in diabetics. J Bone Joint Surg Am. 1982;64:930–3.
21. Rosenblum BI, Giurini JM, Chrzan JS, Habershaw GM. Preventing loss of the great toe with the hallux interphalangeal joint arthroplasty. J Foot Ankle Surg. 1994;33:557–60.
22. Giurini JM, Chrzan JS, Gibbons GW, Habershaw GM. Sesamoidectomy for the treatment of chronic neuropathic ulcerations. J Am Podiatr Med Assoc. 1991;81:167–73.
23. Grayson ML, Gibbons GW, Balogh K, Levin E, Karchmer AW. Probing to bone in infected pedal ulcers: a clinical sign of underlying osteomyelitis in diabetic patients. JAMA. 1995;273:721–3.
24. Young MJ, Coffey J, Taylor PM, Boulton AJM. Weight bearing ultrasound in diabetic and rheumatoid arthritis patients. Foot. 1995;5:76–9.
25. Giurini JM, Rosenblum BI. The role of foot surgery in patients with diabetes. Clin Podiatr Med Surg. 1995;12:119–27.
26. Fernando DJ, Masson EA, Veves A, Boulton AJM. Relationship of limited joint mobility to abnormal foot pressures and diabetic foot ulceration. Diabetes Care. 1991;14:8–11.
27. Veves A, Sarnow MR, Giurini JM, Rosenblum BI, Lyons TE, Chrzan JS, Habershaw GM. Differences in joint mobility and foot pressures between black and white diabetic patients. Diabet Med. 1995;12:585–9.
28. Tillo TH, Giurini JM, Habershaw GM, Chrzan JS, Rowbotham JL. Review of metatarsal osteotomies for the treatment of neuropathic ulcerations. J Am Podiatr Med Assoc. 1990;80:211–7.
29. Trnka HJ, Gebhard C, Muhlbauer M, Ivanic G, Ritschi P. The Weil osteotomy for treatment of dislocated lesser metatarsophalangeal joints: good outcome in 21 patients with 42 osteotomies. Acta Orthop Scand. 2002;73(2):190–4.
30. McKittrick LS, McKittrick JB, Risley T. Transmetatarsal amputation for infection or gangrene in patients with diabetes mellitus. Ann Surg. 1949; 130:826.
31. Kates A, Kessel L, Kay A. Arthroplasty of the forefoot. J Bone Joint Surg. 1967;49B:552.
32. Hoffman P. Operation for severe grades of contracted or clawed toes. Am J Orthop Surg. 1912;9:441–9.
33. Marmor L. Resection of the forefoot in rheumatoid arthritis. Clin Orthop. 1975;108:223.
34. Clayton ML. Surgery of the forefoot in rheumatiod arthritis. Clin Orthop. 1960;16:136–40.
35. Jacobs RL. Hoffman procedure in the ulcerated diabetic neuropathic foot. Foot Ankle. 1982;3:142–9.
36. Giurini JM, Habershaw GM, Chrzan JS. Panmetatarsal head resection in chronic neuropathic ulcerations. J Foot Surg. 1987;26:249–52.
37. Giurini JM, Basile P, Chrzan JS, Habershaw GM, Rosenblum BI. Panmetatarsal head resection: a viable alternative to the transmetatarsal amputation. J Am Podiatr Med Assoc. 1993;83:101–7.
38. Hodor L, Dobbs BM. Pan metatarsal head resection: a review and new approach. J Am Podiatry Assoc. 1983;73(6):287–92.

39. Harris JR, Brand PW. Patterns of disintegration of the tarsus in the anesthetic foot. J Bone Joint Surg. 1966;48B:4–16.
40. Sanders LJ, Frykberg RG. Diabetic neuropathic osteoarthropathy: the charcot foot. In: Frykberg RG, editor. The high risk foot in diabetes mellitus. New York, NY: Churchill-Livingstone; 1990. p. 297–338.
41. Rosenblum BI, Giurini JM, Miller LB, Chrzan JS, Habershaw GM. Neuropathic ulcerations plantar to the lateral column in patients with Charcot foot deformity: a flexible approach to limb salvage. J Foot Ankle Surg. 1997;36(5):360–3.
42. Wu SU, Marston W, Armstrong DG. Wound care: the role of advanced wound-healing technologies. J Am Podiatr Med Assoc. 2010;100(5):385–94.
43. Capobianco CM, Zgonis T. An overview of negative pressure wound therapy for the lower extremity. Clin Pod Med Surg. 2009;26(4):619–31.
44. Lerman B, Oldenbrook L, Ryu J, Fong KD, Schubart PJ. The SNaP Wound Care System: a case series using a novel ultraportable negative pressure wound therapy device for the treatment of diabetic lower extremity wounds. J Diabetes Sci Technol. 2010;4(4):831–2.
45. Mueller MJ, Sinacore DR, Hastings MK, Strube MJ, Johnson JE. Effect of achilles tendon lengthening on neuropathic plantar ulcers. J Bone Joint Surg. 2003;85-A(8):1436–45.
46. Cunha M, Faul J, Steinberg J, Attinger C. Forefoot ulcer recurrence following partial first ray amputation. J Am Podiatr Med Assoc. 2010;100(1):80–1.
47. Willrich A, Angirasa AK, Sage RA. Percutaneous tendo achillis lengthening to promote healing of diabetic plantar foot ulceration. J Am Podiatr Med Assoc. 2005;95(3):281–4.
48. Armstrong DG, Stacpoole-Shea S, Nguyen H, Harkless LB. Lengthening of the achilles tendon in diabetic patients who are at high risk for ulceration of the foot. J Bone Joint Surg. 1999;81-A:535–8.
49. Downey MS. Ankle equinus. In: Banks AS, Downey MS, Martin DE, Miller SJ, editors. McGlamry's comprehensive textbook of foot and ankle surgery. Philadelphia, PA: Lippincott Williams & Wilkins; 2001. p. 715–60.
50. Sammarco VJ, Sammarco GJ, Walker Jr EW, Guiao RP. Midtarsal arthrodesis in the treatment of Charcot midfoot arthropathy. Surgical technique. J Bone Joint Surg. 2010;92-A(Supp 1):1–19.
51. Assal M, Stern R. Realignment and extended fusion with use of a medial column screw for midfoot deformities secondary to diabetic neuropathy. J Bone Joint Surg. 2009;91-A(4):812–20.
52. Miller DS, Lichtman WF. Diabetic neuropathic arthropathy of feet. Arch Surg. 1955;70:513.
53. Sinha S, Munichoodappa C, Kozak GP. Neuroarthropathy (Charcot Joints) in diabetes mellitus: clinical study of 101 cases. Medicine. 1972;52:191.
54. Cofield RH, Morison MJ, Beabout JW. Diabetic neuroarthropathy in the foot: patient characteristic and patterns of radiographic change. Foot Ankle. 1983;4:15.
55. Banks AS, McGlamry ED. Charcot foot. J Am Podiatr Med Assoc. 1989;79:213–35.
56. Bier RR, Estersohn HS. A new treatment for Charcot joint in the diabetic foot. J Am Podiatr Med Assoc. 1987;77:63–9.
57. Conway JD. Charcot salvage of the foot and ankle using external fixation. Foot Ankle Clin. 2008;13(1):157–73.
58. Burns PR, Wukich DK. Surgical reconstruction of the Charcot rearfoot and ankle. Clin Podiatr Med Surg. 2008;25(1):95–120.
59. Pinzur MS. The role of ring external fixation in Charcot foot arthropathy. Foot Ankle Clin. 2006;11(4):837–47.
60. Cooper PS. Application of external fixators for management of Charcot deformities of the foot and ankle. Foot Ankle Clin. 2002;7(1):207–54.
61. Saltzman CL. Salvage of diffuse ankle osteomyelitis by single-stage resection and circumferential frame compression arthrodesis. Iowa Orthop J. 2005;25:47–52.
62. Sayner LR, Rosenblum BI. External fixation for Charcot foot reconstruction. Curr Surg. 2005;62(6):618–23.