INTRODUCTION

Healing diabetic foot ulcerations is a multi-faceted process engaging the interdisciplinary team approach (1). Preferably this approach heals the diabetic ulcer and obviates the need for off-loading surgery. In cases where the interdisciplinary team approach (i.e., excellent wound care, conservative off-loading, vascular, metabolic, nutritional, and infection management) fails to permanently resolve a diabetic ulceration, the healing approach shifts towards off-loading surgery that is curative, functional, and customized.

The core supposition of curative off-loading surgery (OLS) is that each ulcer location is uniquely and causally associated with pathological deformity and biomechanics. Accordingly, the definitive principle of curative OLS begins with ascertaining the pathomechanics inducive to diabetic ulcer formation. Following discovery of causative mechanical factors, OLS procedure selection is customized to the clinical condition of the patient. The presence of infection, metabolic factors, age, social support, activity level, and the ability to remain non-weightbearing affect both timing and selection of curative procedures. Consequently, the ultimate or preferred corrective procedures may not be indicated given the condition of the patient.

A common area requiring curative OLS is the great toe. Among neuropathic diabetic foot ulcer locations, the plantar hallux is one of the most commonly involved sites (2). In this study, which catalogued ulcer locations in 360 consecutive feet, nearly 60% of plantar diabetic foot ulcers occurred at the first ray wherein the plantar hallux was more frequently wounded (34.5%) than the plantar first metatarsal (25.3%).

The importance of healing hallux ulcerations has been well documented. Partial first ray resection at any level (partial hallux and/or metatarsal) appears to be associated with a 20-50% re-amputation rate at a more proximal level (3,4). These investigators questioned the reliability and durability of partial first ray amputations. Additionally, given that nearly one-half of the patients died during the follow-up period, these authors suggested that a transmetatarsal amputation be considered at initial presentation (3,4). These findings further validate the importance of preserving the hallux and first ray through curative OLS.

As mentioned previously, determining the causative overloading forces is essential to curative OLS of hallux ulcerations. A variety of intrinsic and extrinsic biomechanical overloading forces contribute to neuropathic hallux ulceration (Table 1). Intrinsic forces are those occurring at the level of the hallux and typically are healed by OLS at the hallux level. Extrinsic causative forces of hallux ulceration emanate from anatomic levels proximal to the hallux, and curative OLS is focused upon proximal pathomechanics (e.g., by correcting first metatarsophalangeal joint [MPJ] and equinus pathology).

In addition to evaluating overloading forces, the location of the hallux ulcer is a reliable indicator of causative pathomechanics. Taken as an isolated indicator, the site of hallux ulceration can clarify causation as intrinsic or extrinsic and further substantiate curative OLS options (Table 2).

The purpose of this article is to serve as a general procedure selection guide for curative healing of plantar hallux neuropathic diabetic foot ulcers. The forensic approach herein pursues etiologic pathomechanics by starting with ulcer location (the effect) and concludes with recognition of overloading force (the cause). A similar

<table>
<thead>
<tr>
<th>Intrinsic</th>
<th>Extrinsic</th>
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<tr>
<td>Hallux extensus</td>
<td>Equinus</td>
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<tr>
<td>Hallux malleus</td>
<td>Hallux valgus</td>
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<tr>
<td>Hallux limitus/rigidus</td>
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approach has been described for plantar first metatarsal ulcers (5). Ultimately, the goals of curative offloading hallux surgery are to preserve the first ray and permanently resolve the ulceration with a single surgery.

**HALLUX ULCER LOCATIONS**

This section reviews common hallux ulcer locations, etiology and surgical treatment options. Towards achieving good surgical outcomes the following principles are recommended:

1. The patient is medically optimized through the interdisciplinary team approach.
2. Whenever possible the ulcer is epithelialized at the time of surgery. The presence of an open ulcer is a risk factor for contaminating the surgical site, and typically limits procedure options. This is especially true for osseous procedures requiring internal fixation.
3. In cases where the ulcer is not healed preoperatively, the ulcer must be clean and uninfected.
4. All neuropathic patients undergoing curative OLS must be managed more closely and for a longer period postoperatively than sensate patients. The neuropathic postoperative patient lacks normal pain feedback and can be completely unaware of sabotaging outcomes.
5. Address equinus as indicated at the time of curative OLS. The author prefers the gastrocnemius recession to the tendoAchilles lengthening (TAL). Among gastrocnemius recession procedures the modified Strayer is most conducive to being performed in the supine position and offers reduced intraoperative complications (6).
6. To ensure discovery of overloading factors and globally assess causation a thorough lower extremity weightbearing and non-weightbearing biomechanical examination must be performed bilaterally. Discovery of proximal asymmetries and abnormalities may be addressed through physical therapy and other supportive measures. Equally interesting and instructive (for the forensic pathomechanics investigator) is determining why the opposite limb is not affected.
7. Overt patient specific deformities (e.g., Charcot) exhibit unique characteristics, which are beyond the scope of this article.
8. Combined intrinsic and extrinsic pathomechanics may contribute to hallux ulceration (i.e., distal translation of HL/HR forces may incite hallux extensus deformity, or hallux valgus and limitus deformities may combine to create a hallux ulcer). In these cases, curative OLS addressing multiple ulcerative etiologies may be indicated.

**Distal Hallux Ulcers**

Distal hallux tuft ulcers are most commonly caused by flexor hallucis longus contracture at the hallux interphalangeal (IPJ) joint level, creating a hallux malleus deformity. The pathomechanics are akin to tuft ulcers of lesser toes wherein muscle imbalance exists between the long digital flexors and extensors. Surgical treatment is directed towards rebalancing the IPJ and correcting the hallux malleus deformity. The ideal curative procedure is the hallux IPJ fusion. In fact this approach is likely the sole curative option for rigid, non-reducible, osseous hallux malleus deformity. This procedure durably corrects hallux malleus, stabilizes the hallux IPJ and redistributes distal pressure to the entire plantar surface of the hallux distal phalanx.

The simplest curative OLS for distal hallux ulcers is flexor hallucis longus (FHL) tenotomy. This tenotomy is

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**Table 2**

<table>
<thead>
<tr>
<th>Ulcer Location</th>
<th>Deformity</th>
<th>Curative OLS options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal hallux</td>
<td>Hallux malleus</td>
<td>FHL tenotomy</td>
</tr>
<tr>
<td>Plantar central hallux IPJ</td>
<td>Hallux hyperextension</td>
<td>Hallux IPJ fusion</td>
</tr>
<tr>
<td>Plantar medial hallux &amp;/or Plantar central-medial</td>
<td>Hallux valgus &amp;/or Hallux limitus</td>
<td>Keller arthroplasty/ 1st Met osteotomy</td>
</tr>
</tbody>
</table>

*Equinus is commonly associated with diabetic foot ulcer formation and must be addressed during curative OLS.*
performed a few millimeters proximal to the hallux IPJ. The author reserves the FHL tenotomy for flexible deformities in less propulsive, older, and sedentary patients. This procedure is a convenient solution in those cases where preoperative ulcer healing has failed. Although the FHL tenotomy is a simple solution allowing more rapid recovery than IPJ fusion, there are drawbacks. In addition to transfer lesions (e.g., to the distal tufts of adjacent toes), the hallux IPJ is destabilized and can incite pathology at this level (especially in more active patients). Following FHL tenotomy, the need for subsequent procedures is a common risk and should be mentioned within informed patient consent.

Plantar Central Hallux IPJ Ulcers
Skin erosion at the plantar central hallux IPJ is typically associated with hyperextension at this articulation (Figure 1). Hallux extensus is easily assessed through hallux IPJ passive range of motion and functional overload may be especially evident during unshod weightbearing and gait. While ulcer location may vary slightly in medial or lateral directions, hyperextension is typically the key etiologic factor. As mentioned earlier hallux ulcerations may be associated with coexisting overloading forces. Regarding hallux IPJ ulcers, hallux limitus needs to be assessed as a cause of hallux hyperextension.

The sole reliable procedure conducive to curative OLS for hallux extensus deformity is hallux IPJ arthrodesis. Unique to repair of this deformity is the crucial requirement to have healed the ulcer prior to joint resection and fixation (hallux limitus will be addressed below).

Presence of a Hallux IPJ Sesamoid
The presence of a hallux IPJ sesamoid is a common etiologic distractor to relevant pathomechanics. This ossicle may be immediately adjacent or deep to hallux ulceration, but overload at this area is reliably related to intrinsic or extrinsic forces. Consequently, isolated excision of a hallux IPJ sesamoid is typically a futile approach to curative OLS necessitating revisional surgery.

Similar futility is experienced when the hallux IPJ arthroplasty is selected as a curative OLS procedure. This procedure destabilizes the hallux IPJ and repairs neither intrinsic nor extrinsic pathomechanics. Postoperatively, etiologic forces continue to load the hallux with a new feature: an unstable IPJ. Consequently, the goal of a single curative OLS will not be met, and subsequent revisional surgery addressing the etiology will be required (Figure 2).

Undoubtedly, revision surgery is technically more difficult, poses higher risks for complications, and involves prolonged recovery. This is particularly true when the revision involves addressing previous hallux IPJ
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Figure 2A. Hallux interphalangeal arthroplasty creates instability at this joint.

Figure 2B. Note the elevated distal phalanx on lateral weight-bearing view.

Figures 2C-E. A few years later this patient developed 2 hallux ulcers (dorsal-medial and plantar-central) associated with an unstable IPJ.

Figure 2D. Note dorsal-medial bony prominence at the hallux interphalangeal joint.
arthroplasty in the presence of extrinsic hallux ulcerative forces. In this case pathology occurs at two sites: the surgeon will need to address both the etiologic extrinsic pathomechanics (e.g., hallux valgus or limitus) and the intrinsic hallux IPJ instability (caused by the arthroplasty). Unfortunately, following 2 surgeries and 3 procedures, higher rates of postoperative complications will likely be encountered through hallux shortening, transfer lesions, and delayed healing.

**Plantar Medial and Plantar Central-Medial Hallux Ulcers**

Ulcers at the plantar medial and plantar central medial hallux are associated with extrinsic overload from the first MPJ and the medial column. Both first MPJ instability (hallux valgus) and rigidity (HL/HR) can overload the plantar surface of the hallux. Further compounding these forces are the effects of equinus upon the medial column (Figure 3). During gait, these extrinsic pathomechanics transfer distally and incite skin breakdown at the plantar medial or plantar central medial surface of the hallux through increased abductory shear and ground reactive force.

The subtle shift in position of hallux ulceration from plantar medial to plantar central medial is a useful clinical sign to distinguish first MPJ pathology. Plantar medial breakdown is most commonly associated with the shearing effects of hallux valgus, and plantar central medial ulceration is typically associated with the ground reactive forces of HL/HR upon the hallux.

Whereas this ulcer location theory makes biomechanical sense, the presence of multiple overloading pathologies can alter hallux ulcer location. For example, features of both hallux valgus and limitus may coexist in the same deformity. Ultimately, in the absence of a hallux extensus deformity, the key etiologic factors of plantar hallux IPJ ulcers are hallux valgus and hallux limitus. Fortunately, curative OLS for both first MPJ pathologies are similar.

Several curative OLS options exist for hallux valgus and limitus. Among all options, the Keller first MPJ arthroplasty is a simple and reliable approach. This procedure is effective for both first MPJ deformities and is conducive to rapid postoperative recovery. This procedure is ideal for older sedentary patients. However, following Keller arthroplasty,
hallux malleus deformity and plantar second metatarsal transfer lesions are common postoperative conditions. In an attempt to minimize these complications, the author recommends minimal joint resection performed 2-3 mm distal to the concavity of the proximal phalanx base (5). Large gauge (2 mm) temporary percutaneous Kirschner wire fixation is maintained for 3 weeks postoperatively to allow for controlled fibrosis. As mentioned above equinus deformity is addressed through gastrocnemius recession. Similarly, partial first metatarsal head excision effectively off-loads hallux ulcers associated with hallux valgus and limitus deformity. However, this procedure is a poor choice for curative OLS of hallux ulcers as it is associated with profound postoperative malleus and second metatarsal transfer lesions.

Distal first metatarsal osteotomy may be utilized for hallux ulcers associated with hallux valgus. However this procedure is likely not indicated for hallux ulcers associated with HL/HR. The use of distal first metatarsal osteotomy for hallux rigidus is of questionable efficacy (7). This systematic review evaluated outcomes of periarticular osteotomy for hallux rigidus deformity (in sensate patients), and revealed a high incidence of postoperative complications associated with these procedures. Ultimately, when using isolated periarticular osteotomy for hallux rigidus, the author recommends proceeding with caution or not at all. Presumably, in the diabetic neuropathic patient, a higher incidence of complications would be likely, thereby rendering periarticular osteotomy procedures as unsuitable for this population.
Likewise, distal first metatarsal osteotomy may initially appear to be an ideal method of correcting hallux valgus, however, in the diabetic neuropathic patient the postoperative course is dangerously fraught with complications. In addition to delayed healing associated with diabetes, the neuropathic patient lacks ordinary pain feedback and subjectively feels comfortable and unrestricted to engage in vigorous activities in the absence of proper support. This is a recipe for potential disaster (Figure 4). If this procedure is selected as a curative OLS option for hallux ulceration rigorous patient selection, extensive patient education, and laborious postoperative management is indicated. This author does not recommend first metatarsal osteotomy as an OLS option for hallux valgus or limitus. Proximal first metatarsal osteotomy and Lapidus procedures are associated with higher rates of complications and are rarely (if at all) indicated for curative OLS.

CONCLUSION

A commonly wounded site, the plantar hallux accounts for nearly 35% of plantar diabetic foot ulcers. Repetitive peak ambulatory plantar pressures about the hallux are inducive to both ulcer formation and recurrence. Amputation of the hallux frequently leads to subsequent proximal amputation. Preservation of the great toe through long term curative healing is paramount to preventing sequential amputations.

The recommended limb preserving approach herein entails linking specific hallux ulcer locations to causative deformity and mechanics. Once off-loading surgery is indicated these causative factors should be repaired during a single, curative, functional, and customized surgery.
Figure 4D. View at 9 weeks postoperative, following pin removal.

Figure 4E. Postoperative view at 14 weeks. This patient missed a subsequent 4 week reappointment visit.

Figure 4F. Postoperative view at 14 weeks.

Figure 4G. At 18 months postoperative, this patient presents with neuropathic degeneration of this distal first metatarsal osteotomy. The chief concern during this visit was bilateral second toe pain, left greater than right.
REFERENCES


