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Management Of Planter Pressure Points in Diabetic Perpheral Neuropathy A Narrative Review

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Abstract—**Background: Diabetic peripheral neuropathy (DPN) leads to loss of protective sensation and altered biomechanical loading of the foot, predisposing to focal plantar pressure points, callus formation, and neuropathic ulceration. Concurrent peripheral arterial disease (PAD) further compromises tissue viability under load, and Charcot neuroarthropathy introduces an especially severe, deformity-driven pattern of midfoot overload. Aim: This narrative review synthesizes current evidence on the pathophysiology, assessment, and management of plantar pressure points in DPN, incorporating the 2023 International Working Group on the Diabetic Foot (IWGDF) guideline updates, the ankle-brachial index (ABI) as a vascular screening tool, the concept of a therapeutic "healing pressure" threshold, Charcot-specific considerations, and emerging wearable sensor technology. Methods: A narrative synthesis of peer-reviewed systematic reviews, meta-analyses, cohort studies, contemporary clinical guidelines (IWGDF 2023 offloading and peripheral artery disease guidelines), and recent technology-focused literature (2023–2025) addressing plantar pressure biomechanics, vascular assessment, and offloading interventions in diabetic foot disease. Summary: Peak plantar pressure is consistently higher at the forefoot in patients with DPN and a history of ulceration compared with those without, and is markedly elevated at the midfoot in Charcot deformity. An in-shoe mean peak plantar pressure below 200 kPa remains the most widely supported therapeutic target for both ulcer healing and recurrence prevention. The ABI remains the first-line vascular screen for PAD but has reduced sensitivity in the presence of arterial calcification; the updated IWGDF 2023 guideline now recommends combining ABI with toe-brachial index and Doppler waveform assessment, since no single modality reliably excludes PAD. The 2023 IWGDF offloading guideline formalizes a stepped approach beginning with non-removable knee-high devices, and wearable sensor-embedded insoles are emerging as adjuncts for continuous, real-time pressure feedback and adherence monitoring. Conclusion: Combining biomechanical offloading targets, updated vascular risk stratification, Charcot-specific vigilance, and emerging monitoring technology allows a more individualized and evidence-based approach to preventing and healing plantar pressure-related complications in diabetic peripheral neuropathy.**

I. INTRODUCTION

Diabetic peripheral neuropathy (DPN) affects approximately 30% of patients at the time of diabetes diagnosis and rises to roughly 50% in those with disease duration exceeding 20 years [1]. DPN causes progressive loss of cutaneous and proprioceptive sensation, beginning distally in the feet, which removes the protective sensory feedback that normally limits harmful mechanical loading during standing and gait [1]. In combination with elevated blood glucose and reduced tissue tolerance, abnormal plantar pressure and shear stress are key mechanical drivers of diabetic foot ulceration (DFU); approximately one in four people with diabetes will develop a DFU during their lifetime [1].

Vascular disease compounds this mechanical burden: approximately half of patients with diabetes and an active foot ulcer have concomitant peripheral artery disease (PAD), according to the 2023 intersocietal IWGDF, European Society for Vascular Surgery (ESVS), and Society for Vascular Surgery (SVS) guideline [2]. Superimposed PAD reduces the capacity of plantar soft tissue to tolerate mechanical load, delays wound healing, and increases amputation risk [3]. Plantar pressure points focal areas of elevated load, typically over the metatarsal heads, heel, and hallux arise from a combination of structural deformity, altered gait mechanics, and loss of protective sensation that permits repetitive unnoticed trauma, and are further exaggerated in the presence of Charcot neuroarthropathy, a deformity-driven midfoot overload syndrome discussed in detail below.

Management of plantar pressure points therefore requires an integrated approach: biomechanical offloading, vascular risk stratification using the ABI (now formally combined with toe-brachial index and Doppler assessment under the updated 2023 IWGDF guidance), recognition and specific management of Charcot deformity, and, increasingly, adjunctive use of wearable sensor technology for real-time monitoring and adherence support. This review addresses each of these dimensions in turn, with particular attention to the evidence underlying plantar pressure thresholds for healing and prevention, the updated role and limitations of ABI, and recent (2023–2025) developments in guidelines and monitoring technology.

II. METHODS

This is a narrative review. Relevant literature was identified through targeted searches for systematic reviews, meta-analyses, cohort studies, and clinical guidance addressing: (i) plantar pressure biomechanics and offloading interventions in diabetic peripheral neuropathy; (ii) diagnostic accuracy and clinical application of the ankle-brachial index in diabetes, including the updated 2023 IWGDF peripheral artery disease guideline; (iii) plantar pressure thresholds associated with ulcer healing and prevention; (iv) Charcot neuroarthropathy as a special case of plantar pressure pathology; and (v) recent (2023–2025) wearable sensor and smart insole technology for plantar pressure monitoring. No formal systematic search protocol, quality appraisal, or meta-analytic pooling was undertaken; findings are synthesized narratively to provide a clinically oriented, updated overview rather than a quantitative systematic review.

III. PATHOPHYSIOLOGY OF PLANTAR PRESSURE POINTS IN DIABETIC PERIPHERAL NEUROPATHY

Loss of somatosensory feedback in DPN alters gait pattern, center-of-pressure trajectory, and muscle activation timing, producing abnormal and often asymmetric loading across the plantar surface. Motor neuropathy contributes to intrinsic muscle wasting and toe deformity, which shifts load anteriorly onto the metatarsal heads, while autonomic neuropathy reduces sweating and predisposes to dry, fissured skin and callus formation, both of which further concentrate local peak pressure. Consistent with this mechanism, meta-analytic pooling of observational studies shows that both mean peak plantar pressure and pressure-time integral are significantly higher in patients with DPN who have a history of plantar ulceration compared with those with DPN but no ulcer history [4]. Plantar tissue hardness is also significantly correlated with peak plantar pressure and pressure-time integral in the forefoot, and increases further with greater daily weight-bearing load, suggesting a self-reinforcing cycle in which callus and tissue stiffening progressively concentrate pressure at already vulnerable sites [5]. Where DPN is accompanied by joint and bone destruction, as in Charcot neuroarthropathy, this pattern of forefoot-predominant overload is joined by a distinct and often more severe midfoot overload pattern, discussed separately in Section 7.

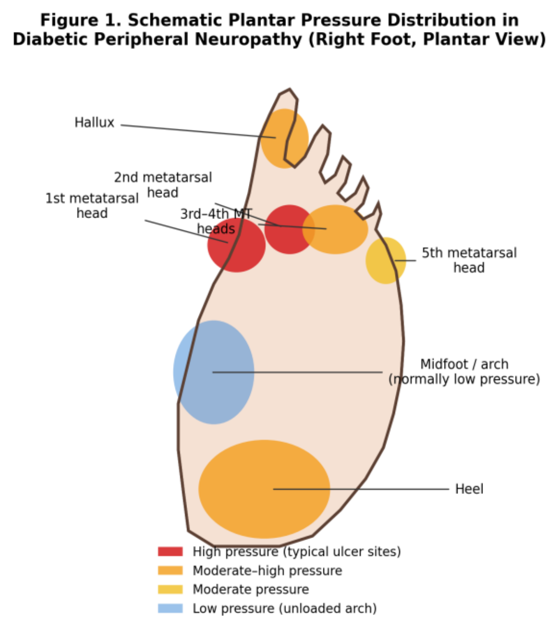

Figure 1. Schematic distribution of typical high-pressure zones on the plantar surface in diabetic peripheral neuropathy. Red/orange zones (metatarsal heads, hallux, heel) correspond to sites most frequently affected by neuropathic ulceration; the medial arch is normally unloaded (blue).

IV. VASCULAR ASSESSMENT: THE ROLE OF THE ANKLE-BRACHIAL INDEX

4.1 Ankle-Brachial Index (ABI)

The ABI is a simple, reproducible, and cost-effective ratio of systolic blood pressure at the ankle to that in the brachial artery, and is recommended as a first-line screening test for lower-extremity PAD in patients with diabetes [6]. An ABI in the range of 0.90–1.30 is generally regarded as normal, while values below 0.90 are diagnostic of PAD and are further stratified by severity: 0.70–0.89 mild, 0.50–0.69 moderate, and ≤0.50 severe arterial obstruction [3]. Values above 1.30 indicate non-compressible, calcified vessels and should not be interpreted using standard cutoffs [3].

Figure 3. Schematic of Ankle-Brachial Index (ABI) Measurement

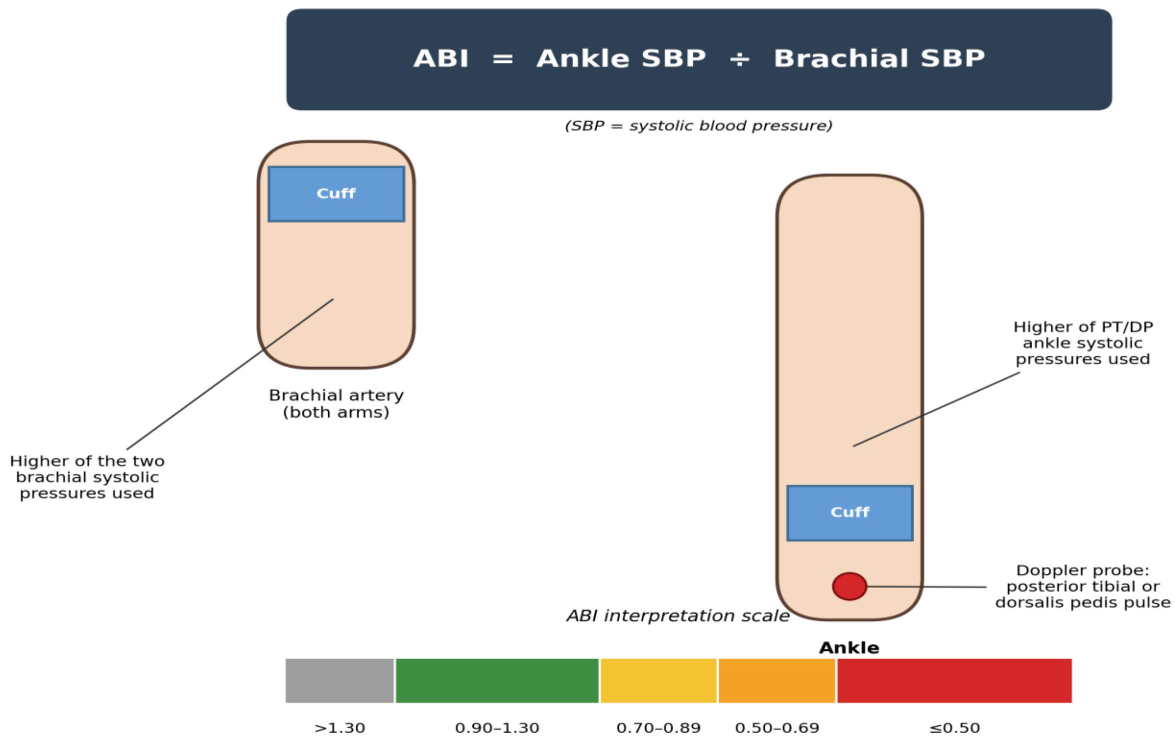

Figure 3. Schematic of ankle-brachial index measurement: cuffs are placed on the brachial artery and the ankle, with Doppler confirmation of the pedal pulse; the ratio of ankle to brachial systolic pressure is interpreted against the color-coded severity scale shown.

ABI Value	Interpretation	Clinical Significance
>1.30	Non-compressible / calcified vessel	Excluded from ABI interpretation; use toe-brachial index
0.90–1.30	Normal	Low likelihood of significant PAD
0.70–0.89	Mild PAD	Monitor; optimize risk factors
0.50–0.69	Moderate PAD	Vascular referral; caution with offloading pressure
≤0.50	Severe PAD	Urgent vascular assessment; high amputation risk

Table 1. Interpretation of the ankle-brachial index in patients with diabetes [3].

4.2 The Updated 2023 IWGDF/ESVS/SVS Approach to Vascular Diagnosis

The 2023 intersocietal IWGDF, ESVS, and SVS guideline on peripheral artery disease in people with diabetes and a foot ulcer represents an important update to prior practice: it explicitly states that no single bedside modality including ABI alone has been shown to be optimal for diagnosing PAD, and that there is no single value above which PAD can be confidently excluded [2]. Accordingly, the guideline recommends evaluating pedal Doppler waveforms in combination with both ABI and toe-brachial index (TBI) measurements in any person with diabetes and a foot ulcer or gangrene, noting that PAD is less likely when ABI is 0.90–1.30, TBI is ≥0.70, and Doppler waveforms are triphasic or biphasic [2]. For a person with diabetes, a foot ulcer, and evidence of severe ischemia defined as any one of an ABI <0.4, ankle pressure <50 mmHg, toe pressure <30 mmHg, transcutaneous oxygen pressure <30 mmHg, or a monophasic or absent pedal Doppler waveform the guideline issues a best-practice statement to urgently consult a vascular specialist regarding possible revascularization [2].

Finding	Threshold	Action
PAD unlikely	ABI 0.90–1.30, TBI ≥0.70, triphasic/biphasic Doppler	Routine risk-based follow-up
PAD present, non-severe	ABI <0.90 or TBI <0.70	Optimize risk factors; consider vascular referral
Severe ischemia (any one)	ABI <0.4; ankle pressure <50 mmHg; toe pressure <30 mmHg; $TcPO_2$ <30 mmHg; monophasic/absent Doppler	Urgent vascular specialist consultation for possible revascularization

Table 2. Summary of 2023 IWGDF/ESVS/SVS vascular diagnostic thresholds and recommended actions in people with diabetes and a foot ulcer [2].

4.3 Diagnostic Performance and Limitations

A low ABI is independently associated with both cardiovascular events and diabetic foot ulceration, supporting its dual role as a vascular and wound-risk marker [6]. However, its diagnostic accuracy is imperfect in people with diabetes: in a community-based cohort using duplex ultrasound as the reference standard, ABI demonstrated high specificity (97.6%) but limited sensitivity (35.5%) for PAD, meaning a normal ABI does not reliably exclude significant arterial disease, particularly where medial arterial calcification renders vessels poorly compressible [7]. In this context, the toe-brachial index (TBI), which relies on digital arteries that are relatively spared by calcification, has been shown to be non-inferior to ABI and is now recommended alongside it, rather than as a fallback, under the updated 2023 guidance [8]. Peripheral arterial disease itself is highly prevalent in patients with DFU (35–60%), delays wound healing through impaired nutrient and antibiotic delivery, and is an independent risk factor for lower-extremity amputation, with ulcer recurrence reportedly five-fold more likely in diabetic patients with PAD than in those without [3].

Practical implication: consistent with the updated 2023 guidance, ABI should not be relied upon in isolation; TBI and pedal Doppler waveform assessment should be obtained alongside it in all patients with diabetes and an active foot ulcer, and repeated periodically in those without an ulcer but with suspected PAD, before offloading intensity or surgical decisions are finalized [2,8].

V. PLANTAR PRESSURE ASSESSMENT AND THE CONCEPT OF “HEALING PRESSURE”

Beyond identifying at-risk anatomical sites, a substantial body of work has sought to define numerical plantar pressure thresholds below which ulcer healing is achieved and maintained, and recurrence risk is minimized sometimes referred to collectively as the “healing pressure” or therapeutic offloading target. The most extensively validated of these is an in-shoe mean peak plantar pressure of less than 200 kPa, a figure derived from cohorts of healed, non-recurrent patients who’s in-shoe pressures averaged approximately 207 kPa, subsequently rounded to a 200 kPa clinical target [9]. In a prospective follow-up study, maintaining in-shoe mean peak plantar pressure below 200 kPa, combined with adherence to therapeutic offloading footwear greater than 80% of the time, significantly reduced the odds of plantar ulcer recurrence [9].

A systematic review of plantar pressure thresholds identified this 200 kPa in-shoe target as the most widely used, appearing in roughly two-thirds of relevant studies, though several alternative thresholds are also applied where an absolute pressure target is impractical: a 25% or 40–80% relative reduction from an individual's baseline pressure, a sustained in-shoe pressure threshold of approximately 32–35 mmHg (≈4.3–4.7 kPa) maintained over a 15-minute walking period, reflecting the capillary perfusion pressure below which local tissue ischemia under load is avoided, and barefoot thresholds of 450–750 kPa for populations assessed without offloading footwear [10]. For barefoot assessment specifically, peak plantar pressures run up to four-fold higher than in-shoe measurements, and site-specific thresholds have also been proposed, such as a critical peak pressure exceedingly approximately 6 kg/cm² (≈588 kPa) at the metatarsal heads, which is associated with a 6.4-fold increase in the odds of prior ulceration at that site [11].

Measurement Context	Threshold	Purpose / Evidence Basis
In-shoe mean peak pressure	<200 kPa	Most widely adopted target for ulcer prevention and healing maintenance; derived from mean in-shoe pressure of 207 kPa observed in healed, non-recurrent patients
In-shoe, sustained loading (15-min)	4.3–4.7 kPa	Alternative sustained pressure-time threshold used in RCT-based offloading protocols
In-shoe, relative reduction	25% or 40–80% from baseline	Used when an absolute kPa target is not feasible due to severe deformity
Capillary perfusion pressure	32–35 mmHg	Threshold beyond which local tissue perfusion is compromised, contributing to pressure-induced ischemic injury
Barefoot peak plantar pressure	450–750 kPa	Screening threshold in populations without offloading footwear; barefoot values run up to 4-fold higher than in-shoe values
Barefoot, site-specific (metatarsal head)	>6 kg/cm² (≈588 kPa)	Associated with 6.4-fold increased odds of prior ulceration at that site

Table 3. Plantar pressure thresholds proposed for prevention and healing of diabetic foot ulceration [9–11].

These thresholds should be interpreted as clinical targets rather than absolute physiological cut-points: substantial heterogeneity exists across studies in measurement systems, number of plantar sub-regions assessed, and the number of steps sampled, and the 200 kPa figure in particular has stronger evidence as a recurrence-prevention target than as a validated healing endpoint for active ulceration [10]. Notably, the updated 2023 IWGDF offloading guideline itself now recommends that, where available, prescribed offloading footwear be validated against a plantar pressure threshold rather than fitted by clinical judgement alone, formally incorporating the biomechanical evidence summarized above into international practice recommendations [12].

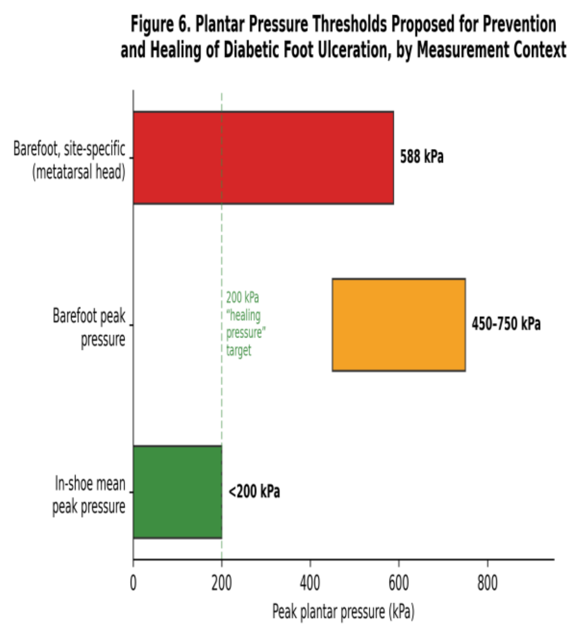

Figure 6. Comparison of plantar pressure thresholds by measurement context. The in-shoe 200 kPa target (green) is the most widely validated; barefoot thresholds (orange, red) run several-fold higher, reflecting the loss of footwear cushioning.

VI. THE 2023 IWGDF OFFLOADING GUIDELINE: A STEPPED APPROACH

The 2023 IWGDF evidence-based guideline on offloading interventions, developed using the GRADE methodology, updates the 2019 guideline and provides a formal stepped approach to healing neuropathic plantar ulcers [12]. For a neuropathic plantar forefoot or midfoot ulcer, the guideline strongly recommends a non-removable knee-high offloading device (e.g., a total contact cast or non-removable knee-high walker) as the first-choice treatment to promote healing [12]. Where a non-removable device is contraindicated or not tolerated, a removable knee-high or ankle-high offloading device is recommended as the second-choice treatment, with explicit encouragement for the person to wear the device during all weight-bearing activity [12]. If no offloading devices are available at all, appropriately fitting footwear combined with felted foam is recommended as a third-choice intervention [12]. For a neuropathic plantar rearfoot ulcer, the guideline similarly favors a non-removable knee-high device over a removable alternative, while non-plantar ulcers are managed with removable devices, footwear modification, toe spacers, orthoses, or digital flexor tenotomy depending on ulcer type and location [12]. Where non-surgical offloading fails to heal a recalcitrant plantar forefoot ulcer, the guideline recommends considering surgical offloading procedures, discussed further in Section 7 in the specific context of Charcot deformity [12]. In people with both moderate-to-severe infection or ischemia, the guideline recommends primarily addressing the infection or ischemia first, using a removable offloading intervention over no offloading at all based on individual patient factors [12].

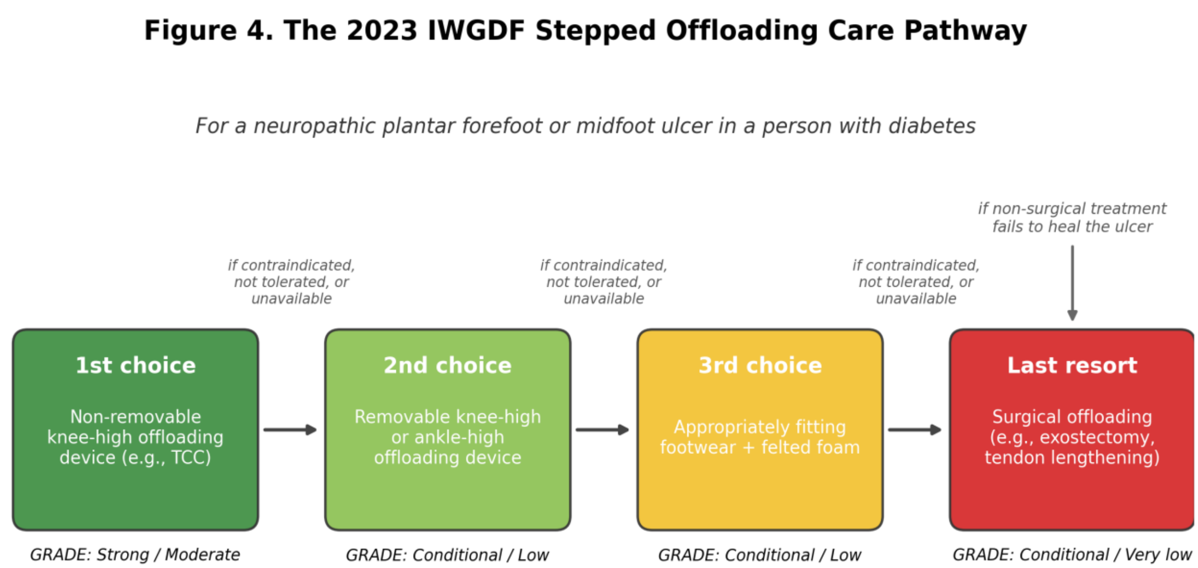

Figure 4. The 2023 IWGDF stepped offloading care pathway for a neuropathic plantar forefoot or midfoot ulcer, showing the GRADE strength and certainty of evidence for each successive step.

VII. CHARCOT NEUROARTHROPATHY: A SPECIAL CASE OF PLANTAR PRESSURE PATHOLOGY

Charcot neuroarthropathy (CN) is a progressive, often insidious complication of severe sensory and autonomic neuropathy characterized by joint dislocation, pathological fracture, and bony fragmentation, most commonly affecting the midfoot, and frequently producing a rocker-bottom deformity with a plantar bony prominence at the apex of the collapse. Because normal joint alignment and the longitudinal arch are lost, Charcot deformity concentrates extreme mechanical load on a small midfoot area that was never designed to bear primary weight, producing a pattern of plantar pressure pathology distinct from the forefoot-predominant pattern typical of uncomplicated DPN. In a cohort comparison, barefoot midfoot peak plantar pressures were dramatically higher in patients with Charcot midfoot deformity than in diabetic patients without Charcot foot (median 756 kPa, interquartile range 260–1267, versus 146 kPa, interquartile range 100–208; p<0.001), whereas in-shoe midfoot pressures did not differ significantly between groups when custom-made footwear was worn and adhered to — underscoring both the severity of unprotected Charcot loading and the protective potential of properly fitted, consistently worn offloading footwear [13]. In the same cohort, the risk of recurrent plantar ulceration over 18 months was substantial, consistent with a broader literature estimate that roughly 40% of patients with diabetic foot ulceration experience recurrence [13].

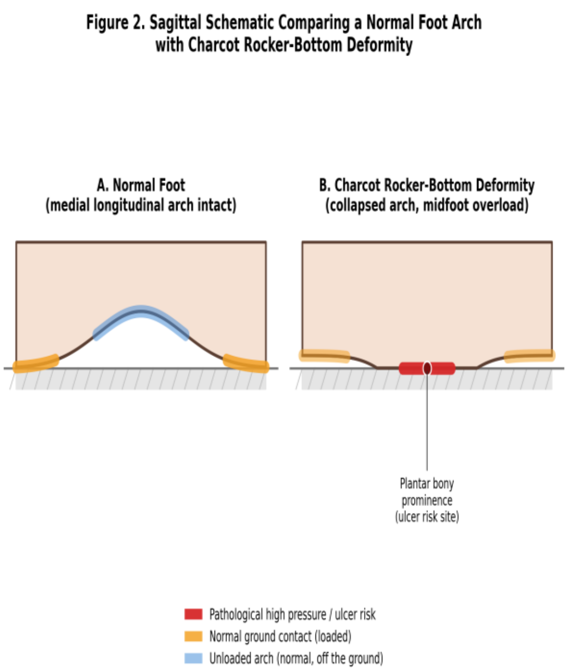

Figure 2. Sagittal schematic contrasting a normal foot, in which the medial longitudinal arch lifts the midfoot clear of the ground (heel and forefoot bear load), with Charcot rocker-bottom deformity, in which midfoot collapse creates a new plantar bony prominence that becomes the dominant, pathological pressure point.

Charcot foot carries a sobering prognosis: reported 5-year mortality is approximately 29% in patients with Charcot foot alone, rising to as high as 56.6% in those who additionally undergo major lower-extremity amputation, underscoring that Charcot deformity is a marker of substantial systemic vascular and metabolic burden and not merely a localized orthopedic problem [14]. The critical plantar pressure threshold widely used as a target for skin tolerance in Charcot management is the same in-shoe 200 kPa figure applied more broadly in DPN offloading, and comparative trials of prefabricated, relasted, and double rocker-modified therapeutic shoes (all paired with the same custom-made insole) have found no significant difference between shoe types in reducing or redistributing peak plantar pressure across high-risk midfoot and forefoot zones in chronic, nonulcerated Charcot feet, suggesting that the insole and overall offloading strategy may matter more than the specific outer shoe design [14].

Management of active Charcot deformity follows the same non-removable, knee-high offloading principle described in Section 6, most often via total contact casting until consolidation, with longer-term protection subsequently provided by a Charcot Restraint Orthotic Walker (CROW) boot. Where a fixed plantar bony prominence causes recurrent or non-healing ulceration despite optimized offloading, surgical offloading — such as exostectomy of the prominent bone, Achilles tendon lengthening to reduce forefoot loading, and, in complex cases, soft-tissue reconstruction — has been described as an effective limb-salvage strategy, illustrated by a reported case in which combined exostectomy, tendon lengthening, and flap reconstruction achieved complete healing without recurrence at six months, despite partial non-adherence to postoperative weight-bearing restrictions [15]. Because Charcot wounds are frequently excluded from standard diabetic foot ulcer clinical trials owing to the complexity of achieving reliable offloading, the evidence base specific to Charcot management remains smaller and more heterogeneous than that for uncomplicated neuropathic forefoot ulceration, and management is often extrapolated from the broader DPN offloading literature.

VIII. OFFLOADING STRATEGIES FOR MANAGING PLANTAR PRESSURE POINTS

Pressure offloading is the accepted standard of care for both healing active neuropathic plantar ulcers and preventing new or recurrent ulceration, and the gold-standard modalities described in Section 6 non-removable total contact casts (TCCs) and, where contraindicated, removable cast walkers (RCWs) remain central to active ulcer management because they most effectively redistribute load away from the wound site, with TCCs additionally enforcing adherence by preventing removal [16]. For ulcer prevention in patients with DPN and no active wound, therapeutic footwear, custom insoles, and orthotic modification are the mainstay of management.

A systematic review and meta-analysis of footwear and insole design features found that specific design elements produced measurable peak plantar pressure reductions: arch support/profile modification reduced peak pressure by a mean of 37 kPa, metatarsal pad or bar addition by 36 kPa, and comprehensive pressure-informed design (custom-molded, based on individual pressure mapping) by 75 kPa compared with standard footwear [17]. In a randomized comparison of footwear conditions, a purpose-designed pressure-relief shoe reduced peak plantar pressure by 51% relative to a control canvas shoe and by 43% relative to participants' own standard footwear [18]. At a population level, pressure-guided therapeutic footwear and custom orthotics have been associated with a 46–65% reduction in ulcer recurrence rates in randomized and cohort data [19], a magnitude of benefit consistent with the strong GRADE recommendation now given to pressure-validated offloading in the 2023 IWGDF guideline [12].

An important and often under-recognized trade-off is that offloading devices, while reducing plantar pressure, can adversely affect static and dynamic balance in patients who already have impaired proprioception from DPN, potentially increasing fall risk; this should be considered and mitigated (e.g., through gait training or assistive devices) when prescribing offloading interventions, particularly rigid or elevated devices such as RCWs [1].

IX. EMERGING TECHNOLOGY: WEARABLE SENSORS AND SMART INSOLES

A persistent challenge in offloading therapy is that measured benefit depends on sustained device adherence, which is difficult to verify by clinical observation alone. This has driven substantial recent (2023–2025) growth in wearable sensor-embedded insoles and footwear designed to continuously monitor plantar pressure, temperature, and adherence, and to provide real-time biofeedback that prompts patients to offload before tissue damage occurs. An early and influential example is an intelligent insole system, worn beneath a patient's own prescribed insoles, that continuously measures plantar pressure at six anatomically targeted sites the first metatarsal head, lateral metatarsal heads, hallux, lateral toes, lateral foot, and heel and delivers a vibratory alert via a connected smartwatch when sustained, low-magnitude pressure suggestive of harmful prolonged sitting or standing is detected, prompting the patient to offload [20].

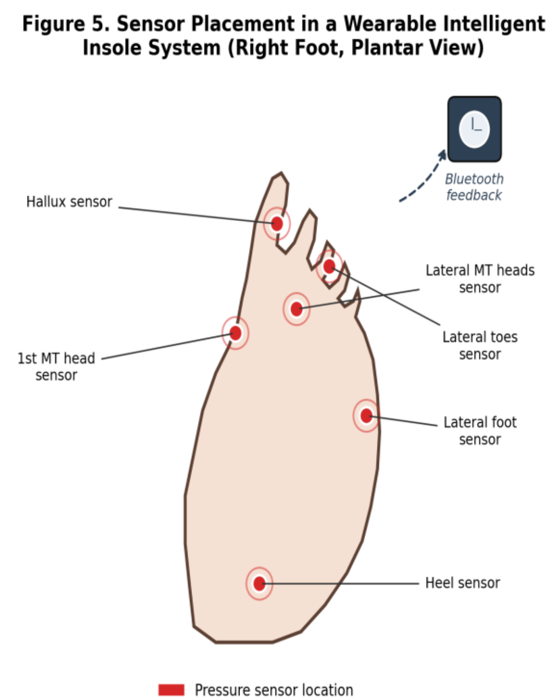

Figure 5. Sensor placement in a wearable intelligent insole system, targeting the six plantar sites most frequently affected by neuropathic ulceration, with wireless (Bluetooth) feedback to a connected smartwatch.

More recent multi-modal systems build on this concept: a prospective cohort study equipped patients with a history of diabetic plantar foot ulcers with custom sensory insoles tracking plantar pressure, plantar temperature, step count, and device adherence simultaneously, feeding this information back to patients as a cue for active offloading and to clinicians for remote patient monitoring when concerning trends emerged; in a randomized comparison of 46 patients with DPN and previous DFU, continued use of such an intelligent insole system was associated with a reduction in plantar pressure relative to controls [21]. A broader review of wearable technologies for diabetic foot ulcer monitoring and risk reduction, spanning studies published between 2021 and 2025, catalogued a heterogeneous but rapidly expanding landscape of device types insoles, socks, anklets, bandages, and boots measuring pressure, temperature, blood flow, shear stress, steps, cadence, and offloading adherence, transmitted variously via smartphone or smartwatch interfaces, though most individual studies to date remain small (often fewer than 30 participants) and short in follow-up duration (weeks to a few months) [22].

Taken together, this emerging evidence suggests that wearable, sensor-based feedback systems have genuine potential to close the adherence gap that limits the real-world effectiveness of otherwise well-validated offloading pressure thresholds (Section 5), by making the abstract 200 kPa "healing pressure" target visible and actionable to the patient in daily life rather than something assessed only intermittently in clinic. However, the current evidence base is still preliminary: sample sizes are small, follow-up periods are short, device types and outcome measures are highly heterogeneous, and few studies have yet demonstrated durable reductions in hard outcomes such as ulcer incidence or recurrence over multi-year follow-up [22].

X. INTEGRATED CLINICAL APPROACH

Effective management of plantar pressure points in DPN requires integration of the vascular, biomechanical, deformity-specific, and, increasingly, technological dimensions described above. In practice, this involves: (i) baseline and periodic vascular assessment combining ABI, TBI, and pedal Doppler waveforms per the updated 2023 IWGDF/ESVS/SVS criteria, with urgent vascular referral for any severe-ischemia threshold (Table 2), before aggressive offloading or debridement is undertaken [2,8]; (ii) barefoot and/or in-shoe plantar pressure assessment, where available, to identify focal pressure points exceeding the relevant threshold (Table 3) and to guide footwear or orthotic prescription, now formally recommended for footwear validation under the 2023 IWGDF guideline [10,11,12]; (iii) explicit recognition of Charcot deformity as a distinct, high-severity midfoot overload pattern warranting non-removable offloading and, where a fixed bony prominence persists, consideration of surgical offloading [13–15]; (iv) selection of an offloading strategy appropriate to wound status following the stepped 2023 IWGDF hierarchy non-removable knee-high device first, removable device second, pressure-informed therapeutic footwear or insoles third, surgical offloading if non-surgical treatment fails [12,16–18]; and (v) structured follow-up incorporating adherence monitoring increasingly supported by wearable sensor feedback since the protective benefit of an offloading device is contingent on both achieving the target pressure and sustaining device use over time [9,20,21].

XI. ECONOMIC AND PUBLIC HEALTH BURDEN

Beyond its clinical severity, diabetic foot ulceration carries a substantial and rising economic burden that reinforces the case for early, guideline-concordant offloading. Diabetes mellitus overall accounts for at least USD 966 billion in global annual health expenditure, an increase of 316% over the preceding 15 years, and diabetic foot disease is a disproportionate contributor to this burden: healthcare expenditure is approximately five-fold higher in people with diabetes who have a foot ulcer than in those without one, with average annual direct and indirect costs per affected person in Europe estimated at approximately USD 13,500 [23]. Regional estimates are similarly striking: in North America, the excess annual cost attributable to diabetic foot ulcers beyond routine diabetes care has been estimated at USD 9–13 billion, while prospective European cohort data project costs of approximately EUR 10 billion per year, with patients who have concurrent PAD and infection incurring substantially higher direct and indirect costs than those with uncomplicated ulcers [24].

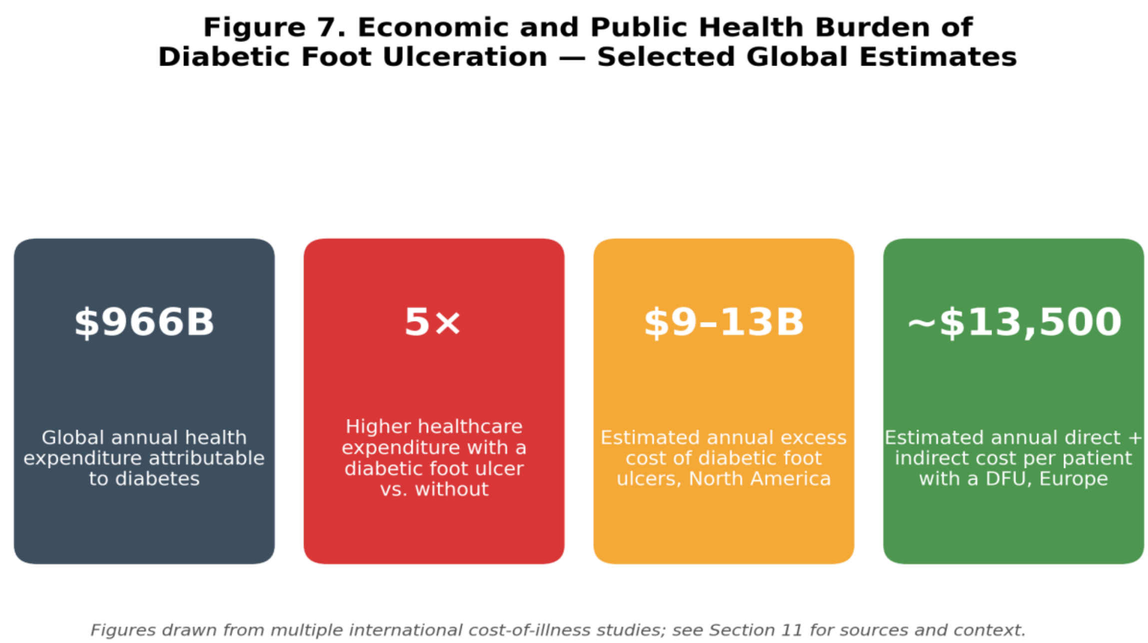

Figure 7. Selected global cost-of-illness estimates for diabetic foot ulceration, illustrating the disproportionate economic burden relative to diabetes care overall [23,24].

These figures underscore that effective plantar pressure management is not only a matter of individual limb preservation but also a significant lever for health-system cost containment: since costs escalate sharply with ulcer severity and amputation, interventions that keep pressure below the thresholds described in Sections 5 and 6 and that are implemented early, per the stepped 2023 IWGDF pathway are likely to be cost-saving as well as clinically beneficial, even though a formal cost-effectiveness analysis of threshold-based offloading is beyond the scope of this review.

XII. FUTURE DIRECTIONS AND RESEARCH PRIORITIES

- Adequately powered, multi-year randomized trials of wearable sensor-based offloading feedback are needed to determine whether the promising pressure reductions seen in early studies (Section 9) translate into durable reductions in ulcer incidence, recurrence, and amputation.
- Charcot-specific offloading and surgical-timing evidence remains disproportionately sparse relative to disease severity; dedicated Charcot cohorts and registries potentially enabled by wearable monitoring that tolerates the complex geometry of Charcot deformity would help close this gap.
- Prospective validation of the 200 kPa in-shoe threshold specifically as a healing endpoint (rather than only a recurrence-prevention target) would strengthen its use as a treat-to-target goal during active ulcer management, not only after healing.
- Standardization of barefoot plantar pressure measurement protocols (sensor systems, number of sub-regions, step counts) would improve comparability across studies and facilitate pooled analyses.
- Economic evaluations directly linking guideline-concordant, pressure-validated offloading to downstream cost savings would strengthen the case for health-system investment in offloading devices, footwear, and monitoring technology in resource-limited settings.
- Integration of ABI/TBI vascular data, plantar pressure mapping, and wearable adherence data into a single digital clinical decision-support tool could operationalize the integrated approach described in Section 10 at the point of care.

XIII. LIMITATIONS OF THE CURRENT EVIDENCE BASE

- Plantar pressure threshold data are derived predominantly from observational and small prospective cohorts with heterogeneous measurement systems, sub-region definitions, and step-sampling protocols, limiting direct comparability across studies [10].
- The widely cited 200 kPa in-shoe threshold has stronger evidence as a recurrence-prevention target than as a validated "healing pressure" for active ulceration [9,10].
- Even with the 2023 IWGDF update, ABI (and TBI) retain documented limitations in diabetic populations owing to arterial calcification, and the guideline itself acknowledges that no single bedside modality is optimal, meaning residual diagnostic uncertainty persists even with combined testing [2,7].
- Barefoot pressure thresholds are less standardized than in-shoe thresholds, and translation between the two is imprecise given up to four-fold differences in measured pressure [11].
- Evidence specific to Charcot neuroarthropathy is comparatively sparse and often drawn from small cohorts or single case reports, in part because Charcot wounds are frequently excluded from standard diabetic foot ulcer trials owing to the difficulty of achieving reliable offloading [14,15].
- Wearable sensor and smart insole studies to date are predominantly small, short-duration feasibility or pilot studies; robust, adequately powered trials demonstrating durable reductions in ulcer incidence or recurrence are still lacking [22].
- Most offloading efficacy data address pressure reduction or ulcer recurrence as surrogate or intermediate outcomes rather than long-term, hard outcomes such as amputation-free survival.

XIV. CONCLUSION

Plantar pressure points in diabetic peripheral neuropathy arise from a combination of sensory loss, motor and autonomic neuropathic changes, and structural deformity, are further compromised by coexisting peripheral arterial disease, and reach their most severe expression in Charcot neuroarthropathy. The updated 2023 IWGDF/ESVS/SVS guidance now recommends combining the ankle-brachial index with toe-brachial index and Doppler waveform assessment rather than relying on ABI alone, reflecting the persistent diagnostic limitations of any single bedside vascular test in this population. An in-shoe mean peak plantar pressure below 200 kPa remains the most widely supported therapeutic "healing pressure" target for both preventing and reducing recurrence of neuropathic plantar ulceration, now formally incorporated into the stepped, GRADE-based 2023 IWGDF offloading guideline. Charcot deformity requires specific recognition and, where fixed bony prominences persist, may warrant surgical offloading alongside standard non-removable casting. Emerging wearable sensor and smart insole technology offers a promising, though still early-stage, means of closing the long-standing gap between validated pressure thresholds and real-world offloading adherence. A combined approach updated vascular risk stratification, biomechanical assessment of plantar pressure, deformity-specific management, and individualized, guideline-concordant offloading, increasingly supported by continuous monitoring technology — offers the most current, evidence-based framework for preventing and healing plantar pressure-related complications in this high-risk population.

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