

Clinical Review

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Triangle of Wound Assessment Made Easy: Revisited

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ABSTRACT

Objective: We have provided evidence based critical review of the “Triangle of wound assessment made easy”.

Background: Wound assessment is a vital step in managing patients with acute and chronic wounds in daily clinical practice. The natural progression of most wounds is to heal naturally in a coherent and timely manner, although a minority of wounds will fail to heal. The aim of treating recalcitrant wounds that fail to heal is to control symptoms and delay or prevent complications. One vital step in managing wounds is proper and comprehensive evidence based wound assessment.

Methods: We have carried out literature review including systemic reviews, met analysis pertaining wound assessment including international guidelines.

Results: We have found few gaps in the proposed “Triangle of wound assessment made easy” which have been addressed accordingly. We have revised the concept of (infection) i.e. “raised white blood cell (WBC) count as a sign of infection” and argued that in up to one-half of patients, even with severe diabetic foot infection raised temperature, WBC count, or sedimentation rate are absent. A second point of concern is the omission of “probing to bone sign” in the diagnostic criteria of local signs of infection. Recent clinical studies have demonstrated that in the presence of a clinically infected ulcer, a positive Probe-to-Bone Test (PTB) test is highly suggestive of osteomyelitis, but a negative test does not rule out the diagnosis; conversely, in the situation of an apparently uninfected foot wound, a positive PTB test is not specific for osteomyelitis, but this diagnosis is unlikely if the PTB test is negative. Lastly, the authors have suggested reducing wound bio burden/manage infection through the use of topical antimicrobial therapy-including antiseptic agents. Topical antiseptics have inconclusive proof of efficacy in various etiology wounds and concern remain regarding their residual cytotoxicity when in contact with newly forming granulating tissues especially when highly concentrated rinse solutions is the common practice in developing countries of the world.

Conclusion: On the basis of these analysis, we propose revising the “Triangle of wound assessment made easy” in order to address these gaps and maximize its utility in clinical practice.

We read the article of Dowsett et al,¹ “Acute wounds, Assessment and diagnosis, Complex wounds” published in the May 2015 issue of Wound International Journal with interest.

A wound occurs as a result of the disruption of the normal structure and function of the skin and soft tissue structure secondary to a variety of etiologies and mechanisms.² Initial assessment of a wound starts by differentiating its etiology whether it’s acute or chronic in nature. The orderly physiologic cascade of inflammation, proliferation and maturation occur as healing is expected to occur in an acute wound,^{3,4} whereas in chronic wounds the cascade is impaired due to many reasons including impaired cellular mechanism, proliferation, migration, dysfunctional angiogenesis and impaired innervation among other reasons.⁵ Examples of chronic wounds include diabetic, arterial ulcers, venous ulcers, and infected wounds including surgical site infections.^{6,7}

Although, the majority of wounds heal without difficulty. Some wounds, however, will become chronic and non-healing. In these circumstances, the aim is to manage symptoms and delay or prevent complications.

Before initiating treatment of any wound a diagnostic hypothesis must be in place to ensure optimal healing outcome. A comprehensive clinical history of wound duration, history of trauma, previous ulceration, wound characteristics (site, size, pain, periwound area characteristics, odor, presence of infection or not and (discharge or exudate), family history of ulceration, skin temperature, current medical history (for example, diabetes mellitus, cardiovascular disease, neuropathy, autoimmune disease, venous insufficiency), previous surgery, smoking history, medications, radiation and allergic reactions to dressings and medications and quality of life (QoL).

Wound assessment is vital in formulating a management plan in order to achieve wound healing and patient well-being. The authors of the wound triangle made a considerable effort in devising a simple and practical assessment tool for clinician's worldwide. However, there are few shortcomings that need to be examined when utilizing this tool in daily practice and these include (Figure 1):

Under the section (infection) raised WBC are documented as a sign of infection.

Although this is true in most situations however in patients with diabetes the clinical diagnosis of diabetic foot ulcer infection poses a complex issue to most clinicians and the ideal method to identify localized diabetic foot ulcers (DFU) infection with certainty remains elusive. Many patients with diabetes may

not feel pain, do not suffer from fever, and will not have raised WBC count or raised erythrocyte-sedimentation rate.⁸⁻¹⁰

The presence of virulent pathogens and extensive tissue damage usually mount systemic signs or symptoms in the host, however in up to one-half of patients, even with severe diabetic foot infection raised temperature, WBC count, or sedimentation rate are absent. None the less worse clinical outcomes of treatment is predicted when these markers are elevated.¹¹

Two recent prospective clinical studies^{12,13} of patients with a diabetic foot ulcer have demonstrated that procalcitonin levels were more sensitive markers of clinical evidence of infection than levels of WBC, C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR). Clinically uninfected wounds can be differentiated accurately from those with mild or moderate infections based on the combined levels of CRP and procalcitonin.¹² Therefore, the inclusion of raised WBC count is insensitive marker and may delay the diagnosis of diabetic foot infections if it was taken into consideration as a sign or marker of infection in patients with diabetes.

A second point to concern is the omission of "probing to bone sign" in the diagnostic criteria of local signs of infection. There is high suspicion of osteomyelitis in a wound which fails to heal after at least 6 weeks of adequate wound therapy and offloading a patient with an adequate blood supply to the affected site. Both the presence of a deep wound area and any exposed bone increase the likelihood of osteomyelitis.¹⁴ Nonetheless, the likelihood of osteomyelitis is not influenced by either elevated WBC count nor the presence of signs of infection of the wound.^{14,15}

Baseline and serial measurements of the wound size (length, width or area, and depth), appearance and location, will help to establish a baseline for treatment and monitor any response to interventions.^{12,13} The method of measurement should be used consistently to aid meaningful tracking of changes over a specific number of days (e.g. 7-14 days).¹⁴ Problems identified in the wound bed may extend beyond the wound edge to the surrounding skin (e.g. maceration, erythema, swelling).

Record wound size: length ___ cm width ___ cm depth ___ cm

Record wound location

Tissue type		Exudate		Infection	
Please tick		Please tick all Δ that apply		Please tick all Δ that apply	
Necrotic	 Δ ___ %	Level	Type	Local	Spreading/systemic
Sloughy	 Δ ___ %	Dry Δ	Thin/watery Δ	\uparrow Pain or new onset Δ	As for local, plus:
Granulating	 Δ ___ %	Low Δ	Thick Δ	Erythema Δ	\uparrow Erythema Δ
Epithelialising	 Δ ___ %	Medium Δ	Cloidy Δ	Oedema Δ	Pyrexia Δ
		High Δ	Purulent (yellow/brown/green) Δ	Local warmth Δ	Abscess/pus Δ
			Pink/red Δ	\uparrow Exudate Δ	Wound breakdown Δ
				Delayed healing Δ	Cellulitis Δ
				Bleeding/friable granulation tissue Δ	General malaise Δ
				Malodour Δ	Raised WBC count Δ
				Pocketing Δ	Lymphangitis Δ

Figure 1: Using the triangle of wound assessment-wound bed.

According to a recent clinical study, independent risk factors for osteomyelitis in a patient with lower limb infection were, previous history of a wound, wounds that extended to bone or joint and multiple or recurrent wounds.¹⁶ Osteomyelitis can be differentiated from cellulitis by combining together laboratory data and clinical findings (ulcer depth > 3 mm or CRP > 3.2 mg/dL, ulcer depth > 3 mm or ESR > 60 mm/hour).¹⁷ Currently, there is no specific clinical criteria to diagnose osteomyelitis in the lower limb of patients with diabetes, although the presence of “sausage toe” (swollen, erythematous, and lacking normal contours) is highly suggestive of the diagnosis.¹⁸

Health care practitioners need to be aware that the true depth of a wound is often not clinically apparent, so a sterile blunt metal probe must be introduced into the wound at each visit (the PTB test). Any wound with a visible bone or with either a positive PTB test (i.e., palpable hard, gritty bone) is highly likely to be osteomyelitis until proven otherwise.¹⁷

The accuracy of the probe to bone test in predicting or excluding osteomyelitis is, however, directly related to the pretest likelihood (i.e., the prevalence in the population under study) of osteomyelitis. Recent clinical studies have demonstrated that in the presence of a clinically infected ulcer, a positive PTB test is highly suggestive of osteomyelitis, but a negative test does not rule out the diagnosis; conversely, in the situation of an apparently uninfected foot wound, a positive PTB test is not specific for osteomyelitis, but this diagnosis is unlikely if the PTB test is negative.¹⁹⁻²⁵

This clinical sign is of great importance since early reports in 1995, Grayson et al¹⁶ who explored the possibility of osteomyelitis in wounds by the use of a sterile blunt metal probe. They concluded that the PTB test had a positive predictive value of 89%. Later on in 2007, Lavery et al¹⁷ followed-up 247 patients with suspected osteomyelitis concluded that the positive predictive value was only 57% in a population with a lower prevalence of osteomyelitis.¹⁷ However, in 2014 in an outpatient setting, Morales Lozano et al¹⁸ followed-up 132 of patients with clinical suspicion of osteomyelitis over a 36 months concluded that the PTB test had an efficiency of 94%, sensitivity of 98%, specificity of 78%, positive predictive value of 95%, and negative predictive value of 91% ($p < 0.001$, κ 0.803); the positive likelihood ratio was 4.41, and the negative likelihood ratio was 0.02 (95% CI).¹⁸ Therefore, the inclusion of “probe to bone test (PTB)” is a vital component in assessing any chronic wound especially in patients with diabetic wounds of more than 4 week duration where the suspicion of the possibility of osteomyelitis is high.

The third point which is worth considering is mentioned under “setting treatment goals” (reduce wound bioburden/manage infection (e.g. topical antimicrobial therapy-including anti-septic agents-may be used for local infection and combined with antibiotic therapy for spreading or systemic infection).

The term topical antimicrobial is a vague term and may imply the use of topical antibacterial creams and ointments. The use of topical antibiotics among patients with diabetic wounds is not justified since most wounds in patients with diabetes have polymicrobial colonization.

Topical antiseptics have inconclusive proof of efficacy in various etiology wounds and concern remain regarding their residual cytotoxicity when in contact with newly forming granulating tissues especially when highly concentrated rinse solutions is the common practice in developing countries of the world. Some authors have even warned against the routine use of antiseptic solutions due to their cytotoxicity and lack of demonstrated benefit over saline irrigation.³¹ Furthermore, recently the International Working Group on Diabetic Foot Management (IWGDF) has warned against the use of topical antimicrobial dressing with the goal of improving wound healing or preventing secondary infection.²⁰ They went even further by saying that “currently supporting data are too limited to recommend topical antimicrobial therapy”.²⁰⁻²² The use of antimicrobial dressings for DFUs was recently assessed *via* a systemic review which concluded that current evidence base is too weak to suggest any specific antimicrobial dressing.²³ The management of diabetic foot ulcers pose a challenge to attending clinicians and some advanced and expensive topical antimicrobials have been in use through the last few decades.²⁴ On the other hand, alternative modalities such as natural honey have been in use for a millennia and recently a new interest by clinicians worldwide have surfaced through the publications of case series and case studies highlighting its efficacy in various types of wounds.²⁵⁻³² This is quite important since natural honey offers an efficacious and cost effective alternative option characterized by its ability to provide moisture, antimicrobial cover and anti-inflammatory properties.³² This is quite important especially in developing countries where diabetes has reached epidemic proportions and the incidence of diabetic wounds is likely to rise too thereby posing a huge economic burden on already stretched out economies. Additionally, health practitioners worldwide have over utilized antimicrobials, particularly topical antiseptics as a result of coining the controversial idea of excess wound bio burden in the medical literature, despite little evidence substantiating any benefit of these dressings over conventional therapy.³³⁻³⁷ In addition to their potential for causing local reactions and expense, bacterial resistance may emerge as a consequence to the arbitrary use of these antimicrobials.^{38,39} Due to these inherent risks, and a lack of sound clinical evidence of any advantages, the use of topical antimicrobials for clinically uninfected wounds is not recommended.³³ Furthermore, current data does not support any benefit to using advanced wound products such as silver-based dressings for clinically infected wounds.⁴⁰

We believe there is an urgent need for head to head large scale multicenter randomized controlled trials to clarify the role of antimicrobial therapies in various etiology wounds and we believe that this knowledge is imperative before universal suggestions regarding wound assessment and management

could be advised.

HIGHLIGHTS

- A revised version of the “Triangle of wound assessment made easy” is urgently needed
- Specific areas of improvement of the “Triangle of wound assessment made easy” is proposed.
- The mechanism relies on stabilization changes of anionic ubiquinone intermediates.
- The validity and reliability of “Triangle of wound assessment made easy” should be tested through double-blinded randomized controlled trials controlled during turnover.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

REFERENCES

1. Dowsett C, Gronemann M, Harding K. Taking wound assessment beyond the wound edge. *Wounds International*. 2015; 6(1): 6-10.
2. Atiyeh BS, Ioannovich J, Al-Amm CA, El-Musa KA. Management of acute and chronic open wounds: The importance of moist environment in optimal wound healing. *Curr Pharm Biotechnol*. 2002; 3(3): 179-195.
3. Schultz GS, Sibbald RG, Falanga V, et al. Wound bed preparation: A systematic approach to wound management. *Wound Repair Regen*. 2003; 11(Suppl 1): S1.
4. Broughton G 2nd, Janis JE, Attinger CE. The basic science of wound healing. *Plast Reconstr Surg*. 2006; 117: 12S.
5. Golinko MS, Clark S, Rennert R, et al. Wound emergencies: The importance of assessment, documentation, and early treatment using a wound electronic medical record. *Ostomy Wound Manage*. 2009; 55: 54-61. Web site. http://s3.amazonaws.com/academia.edu.documents/42003584/Wound_emergencies_the_importance_of_asse20160203-6042-131xx49.pdf?AWSAccessKeyId=AKIAJ56TQJRTWSMTNPEA&Expires=147652265-2&Signature=GFFV4ChLqvqq1ddfwYxt48dl2w4%3D&response-content-disposition=inline%3B%20filename%3DWound_emergencies_the_importance_of_asse.pdf. Accessed August 21, 2016.
6. Brem H, Balledux J, Bloom T, et al. Healing of diabetic foot ulcers and pressure ulcers with human skin equivalent: A new paradigm in wound healing. *Arch Surg*. 2000; 135: 627.
7. Brem H, Jacobs T, Vileikyte L, et al. Wound-healing protocols for diabetic foot and pressure ulcers. *Surg Technol Int*. 2003; 11: 85.
8. Eneroth M, Apelqvist J, Stenstrom A. Clinical characteristics and outcome in 223 diabetic patients with deep foot infections. *Foot Ankle Int*. 1997; 18: 716-722. doi: [10.1177/107110079701801107](https://doi.org/10.1177/107110079701801107)
9. Edelson GW, Armstrong DG, Lavery LA, Caicco G. The acutely infected diabetic foot is not adequately evaluated in an inpatient setting. *J Am Podiatr Med Assoc*. 1997; 87(6): 260-265.
10. Armstrong DG, Perales TA, Murff RT, Edelson GW, Welchon JG. Value of white blood cell count with differential in the acute diabetic foot infection. *J Am Podiatr Med Assoc*. 1996; 86(5): 224-227. doi: [10.7547/87507315-86-5-224](https://doi.org/10.7547/87507315-86-5-224)
11. Lipsky BA, Sheehan P, Armstrong DG, Tice AD, Polis AB, Abramson MA. Clinical predictors of treatment failure for diabetic foot infections: Data from a prospective trial. *Int Wound J*. 2007; 4: 30-38. doi: [10.1111/j.1742-481X.2006.00274.x](https://doi.org/10.1111/j.1742-481X.2006.00274.x)
12. Jeandrot A, Richard JL, Combescure C, et al. Serum procalcitonin and C-reactive protein concentrations to distinguish mildly infected from non-infected diabetic foot ulcers: A pilot study. *Diabetologia*. 2008; 51: 347-352. doi: [10.1007/s00125-007-0840-8](https://doi.org/10.1007/s00125-007-0840-8)
13. Uzun G, Solmazgul E, Curuksulu H, et al. Procalcitonin as a diagnostic aid in diabetic foot infections. *Tohoku J Exp Med*. 2007; 213: 305-312. doi: [10.1620/tjem.213.305](https://doi.org/10.1620/tjem.213.305)
14. Newman LG, Waller J, Palestro CJ, et al. Unsuspected osteomyelitis in diabetic foot ulcers. Diagnosis and monitoring by leukocyte scanning with indium in 111 oxyquinoline. *JAMA*. 1991; 266: 1246-1251.
15. Armstrong DG, Harkless LB. Outcomes of preventative care in a diabetic foot specialty clinic. *J Foot Ankle Surg*. 1998; 37: 460-466. doi: [10.1016/S1067-2516\(98\)80022-7](https://doi.org/10.1016/S1067-2516(98)80022-7)
16. 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: 721-723.
17. Lavery LA, Armstrong DG, Peters EJG, Lipsky BA. Probe-to-bone test for diagnosing diabetic foot osteomyelitis. *Diabetes Care*. 2007; 30(2): 270-274. doi: [10.2337/dc06-1572](https://doi.org/10.2337/dc06-1572)
18. Morales Lozano R, González Fernández ML, Martínez Hernández D, Beneit Montesinos JV, Guisado Jiménez S, Gonzalez Jurado MA. Validating the probe-to-bone test and other tests for diagnosing chronic osteomyelitis in the diabetic foot. *Diabetes Care*. 2010; 33(10): 2140-2145. doi: [10.2337/dc09-2309](https://doi.org/10.2337/dc09-2309)
19. White RJ, Cutting K, Kingsley A. Topical antimicrobi-

- als in the control of wound bioburden. *Ostomy Wound Manage.* 2006; 52: 26-58. Web site. <http://europepmc.org/abstract/med/16896238>. Accessed August 21, 2016.
20. Bakker K, Apelqvist J, Lipsky BA, Van Netten JJ. The 2015 IWGDF guidance documents on prevention and management of foot problems in diabetes: Development of an evidence-based global consensus. *Diabetes Metab Res Rev.* 2016; 32(Suppl 1): 2-6. Web site. http://www.iwgdf.org/files/2015/website_development.pdf. Accessed August 21, 2016.
21. Gottrup F, Apelqvist J, Bjansholt T, et al. EWMA document: Antimicrobials and non-healing wounds. Evidence, controversies and suggestions. *J Wound Care.* 2013; 22: S1-89. doi: [10.12968/jowc.2013.22.Sup5.S1](https://doi.org/10.12968/jowc.2013.22.Sup5.S1)
22. Vermeulen H, van Hattem JM, Storm-Versloot MN, Ubbink DT. Topical silver for treating infected wounds. *Cochrane Database Syst Rev.* 2007; 24: CD005486. doi: [10.1002/14651858.CD005486.pub2](https://doi.org/10.1002/14651858.CD005486.pub2)
23. Storm-Versloot MN, Vos CG, Ubbink DT, Vermeulen H. Topical silver for preventing wound infection. *Cochrane Database Syst Rev.* 2010; 17: CD006478. doi: [10.1002/14651858.CD006478.pub2](https://doi.org/10.1002/14651858.CD006478.pub2)
24. Roeder B, Van Gils CC, Maling S. Antibiotic beads in the treatment of diabetic pedal osteomyelitis. *J Foot Ankle Surg.* 2000; 39: 124-130. doi: [10.1016/S1067-2516\(00\)80037-X](https://doi.org/10.1016/S1067-2516(00)80037-X)
25. Nelson EA, O'Meara S, Golder S, et al. Systematic review of antimicrobial treatments for diabetic foot ulcers. *Diabet Med.* 2006; 23: 348-359. doi: [10.1111/j.1464-5491.2006.01785.x](https://doi.org/10.1111/j.1464-5491.2006.01785.x)
26. Al-Lenjawi B, Mohamed H, Al-Ali A, Kherallah B. Are all wound products created equally? The re-emergence of natural honey. *The Journal of Diabetic Foot Complications.* 2015; 7(2): 26-41. Web site. <http://jdfc.org/spotlight/are-all-wound-products-created-equally-the-re-emergence-of-natural-honey/>. Accessed August 21, 2016.
27. Mohamed H, Abu Salma M, Al lenjawi B, et al. The efficacy and safety of natural honey on the healing of foot ulcers: A case series. *Wounds.* 2015; 27(4): 103-114. Web site. <http://europepmc.org/abstract/med/25855854>. Accessed August 21, 2016.
28. Mohamed H, El Lenjawi B, Abu Salma M, Abdi S. Honey based therapy for the management of a recalcitrant diabetic foot ulcer. *J Tissue Viability.* 2014; 23(1): 29-33. doi: [10.1016/j.jtv.2013.06.001](https://doi.org/10.1016/j.jtv.2013.06.001)
29. Mohamed H, Abu Salma M, Al Lenjawi B, et al. Enhancing primary healing post ray amputation in a diabetic patient: Efficacy of natural honey. *J Diabetic Foot Complications.* 2014; 6(1): 13-18. Web site. <http://jdfc.org/spotlight/enhancing-primary-healing-post-ray-amputation-in-a-diabetic-patient-efficacy-of-natural-honey/>. Accessed August 21, 2016.
30. Mohamed H, Abo Salma M, Al Lenjawi B, et al. The efficacy of natural honey in the management of second degree burn complicated by acute dermatitis in a diabetic patient. *J Diabetes Metab.* 2014; 5: 373.
31. Mohamed H, Abo Salma M, El-Lenjawi B, Hassan I. Using honey in post-excision malignant melanoma ulcers. *J Lymphoedema.* 2012; 7(1): 41-45.
32. Al-Lenjawi B, Mohamed H, Abu Salma M, Abo Gouda Z. Natural honey in the management of thermal burn of the foot in a type 2 diabetic patient: A case report. *Dermatol Open J.* 2016; 1(1): 14-18. doi: [10.17140/DRMTOJ-1-105](https://doi.org/10.17140/DRMTOJ-1-105)
33. Lipsky BA, Hoey C. Topical antimicrobial therapy for treating chronic wounds. *Clin Infect Dis.* 2009; 49: 1541-1549. doi: [10.1086/644732](https://doi.org/10.1086/644732)
34. Bergin SM, Wraight P. Silver based wound dressings and topical agents for treating diabetic foot ulcers. *Cochrane Database Syst Rev.* 2006; 1: CD005082. doi: [10.1002/14651858.CD005082.pub2](https://doi.org/10.1002/14651858.CD005082.pub2)
35. Hinchliffe RJ, Valk GD, Apelqvist J, et al. A systematic review of the effectiveness of interventions to enhance the healing of chronic ulcers of the foot in diabetes. *Diabetes Metab Res Rev.* 2008; 24(Suppl 1): S119-S144. doi: [10.1002/dmrr.825](https://doi.org/10.1002/dmrr.825)
36. Hilton JR, Williams DT, Beuker B, Miller DR, Harding KG. Wound dressings in diabetic foot disease. *Clin Infect Dis.* 2004; 39(Suppl 2): S100-S103. doi: [10.1086/383270](https://doi.org/10.1086/383270)
37. Cutting K, White R, Edmonds M. The safety and efficacy of dressings with silver-addressing clinical concerns. *Int Wound J.* 2007; 4: 177-184. doi: [10.1111/j.1742-481X.2007.00338.x](https://doi.org/10.1111/j.1742-481X.2007.00338.x)
38. Chopra I. The increasing use of silver-based products as antimicrobial agents: A useful development or a cause for concern? *J Antimicrob Chemother.* 2007; 59: 587-590. doi: [10.1093/jac/dkm006](https://doi.org/10.1093/jac/dkm006)
39. Percival SL, Woods E, Nutekpor M, Bowler P, Radford A, Cochrane C. Prevalence of silver resistance in bacteria isolated from diabetic foot ulcers and efficacy of silver-containing wound dressings. *Ostomy Wound Manage.* 2008; 54: 30-40.
40. Health Technology Inquiry Service. Silver dressings for the treatment of patients with infected wounds: A review of clinical and cost-effectiveness. *Canadian Agency for Drugs and Technologies in Health.* 2010. Web site. <https://www.cadth.ca/silver-dressings-treatment-patients-infected-wounds-review-clinical-and-cost-effectiveness-0>. Accessed August 21, 2016.