

WCHM

WOUND CARE AND HYPERBARIC MEDICINE

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EDITORIAL STAFF

Best Publishing Company
Jaclyn Mackey, Publisher
jmackey@bestpub.com

MAGAZINE PRODUCTION

Lorraine Fico-White
lorraine@bestpub.com

Diana Robinson
diana@bestpub.com

ADVERTISING/SPONSORSHIPS

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In This Issue...

This robust issue of *WCHM* includes something for everyone.

Puzzled over coding diabetic foot ulcers? Gretchen Dixon offers a solution with a peek into her webinar to help you figure out how to locate and arrange the pieces for accurate coding as well as what needs to be included in the clinical documentation.

In the field of hyperbaric medicine, read about Life Support Technologies' (LST) research concerning the pivotal role of oxygen in pressure injury etiology and prevention. UHMS shares with us their plans to publish a supplemental issue of the *Undersea and Hyperbaric Medicine* journal to discuss results and findings for traumatic brain injury (TBI) studies. And our Clinic in Focus section features Catholic Health Advanced Wound Healing Centers in Buffalo, New York. If you are a part of an exceptional hyperbaric or wound care center, contact us today at info@bestpub.com to be our next featured clinic.

Great news for divers in Guanaja, Honduras. Villa on Dunbar Rock funds the first hyperbaric chamber and clinic in Guanaja. Read about this chamber and clinic in the dive medicine section of this issue.

In the field of wound care, Heather Hettrick authors a three-part series addressing the clinical importance of recognizing edema for differential diagnosis. The first article appearing in this issue concentrates on recognizing edema and performing appropriate clinical tests to help with clinical diagnosis.

Longtime contributor Dr. Michael Strauss returns to *WCHM* with the first article in a series of five that expounds on the prevention of new and recurrent wounds in the lower extremities. This initial paper discusses myths and misconceptions about healing of the foot wound — especially in the diabetic — and introduces the four preventive measures for new and recurrent wounds, each of which will become a subsequent article.

Dr. Jayesh Shah continues his popular Q & A sessions on wound care, this time addressing atypical wounds. Dr. Terry Treadwell explains why continuing education is a big deal and provides a review of *Wound Care Certification Guide, 2nd edition*, which is now available for purchase.

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Lorraine Fico-White
Managing Editor, *WCHM*

Puzzled Over Coding Diabetic Foot Ulcers?

Gretchen Dixon, MBA, RN, CCS, CPCO, AHIMA Approved ICD-10-CM/PCS Trainer

Diabetic Foot Ulcer ICD-10-CM Coding and Wound Care Documentation Webinar is the answer to your puzzle. This webinar will help you figure out how to locate and arrange the pieces for accurate coding as well as what needs to be included in the clinical documentation.

This two-part webinar series brings together many puzzle pieces.

- Objectives
 - Compare provider's clinical documentation with the details required for an accurate ICD-10-CM code selection.
 - Define measurable success factors, and monitor the impact of improved clinical documentation.
 - Emphasize the need to capture accurately the level of acuity and intensity of services with complexity of care clinically documented.
 - Identify common diagnoses selected in reporting wound care services.
- Documentation principles review
- How clinical documentation components support acuity levels
- Clinical documentation required to select the ICD-10-CM code representing the highest level of specificity supporting acuity level
- The future role of selected unspecified codes and reimbursement
- Practicum involves real wound-care scenarios with how to select specific ICD-10-CM codes. Slides will take you in steps from the ICD-10-CM coding manual on how to arrive at the most accurate code and why.
- Question and answer self-assessment to measure your new knowledge and for continuing education credits

The following are excerpts from a few slides that share the depth of information you will learn and be able to use in real time on your next wound-care clinical documentation improvement and coding opportunity.

The six elements in Slide 1 are vital to the selection of an ICD-10-CM code with the highest level of specificity. Each element not documented weakens the specificity of the ICD-10-CM code selection, which has the potential to jeopardize the medical necessity of patient care and possible reimbursement.

SLIDE 1

NOW Identify the Elements of CDI

Increase details of clinical documentation must include specific elements:

- ▶ Acuity (acute / chronic)
- ▶ Anatomical site / location
- ▶ Laterality of ulcer
- ▶ Etiology
- ▶ Was it a non-pressure or a pressure ulcer?
- ▶ Severity of the ulcer depends on the layer of tissue affected

Does your clinical documentation support a high level of acuity?

Another example of weak clinical documentation is the actual description of nonhealthy wound tissue. Clinicians need to document their professional observation of each individual patient's wound. Often the point-click options with an electronic health record does not provide this level of specificity describing the appearance of the nonhealthy tissue, noted in Slide 2. The more details clinically provided demonstrates a higher level of acuity supporting the intensity of services and complexity of care provided to the patient. Speak with the EHR developers to ensure you have adequate space to document in free text your professional observation, thus providing an individuality of information into the patient's medical record.

Unspecified ICD-10-CM codes may cause a risk to financial stability of your department.

There is a place for unspecified diagnosis codes; however, the wound-care environment should not be the place where they are selected. Unspecified codes can cause a risk and a liability for what service is billed because they don't accurately reflect the level of acuity, the intensity of services and the complexity of care provided to the patient. An unspecified code may not support medical necessity of services such as a debridement.

Be proactive and THINK-N-INK about selecting the most descriptive ICD 10-CM code supported with your clinical documentation. HINT: Most third-party payers have been monitoring and continue to monitor the submission of unspecified codes on claim forms. It is anticipated that

WOUND CARE CERTIFICATION STUDY GUIDE

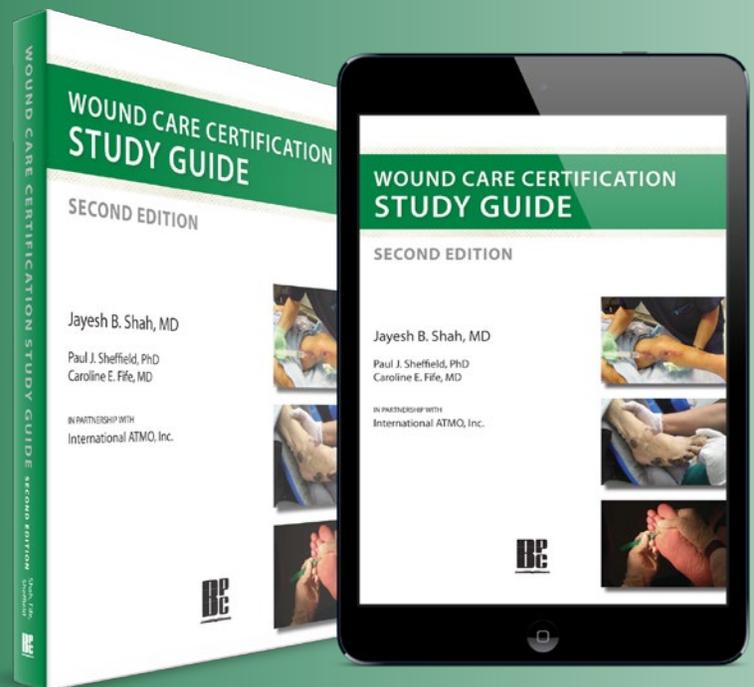
SECOND EDITION

DR. JAYESH SHAH, in partnership with **DR. PAUL SHEFFIELD** of International ATMO and **DR. CAROLINE FIFE** of Intellicure, has created the perfect tool for anyone studying to take a wound certification exam — AAWM, APWCA, CWCN, NAWC, and more.

Now in its second edition, the *Wound Care Certification Study Guide* is fully updated with the latest clinical practices and regulatory and reimbursement information. Drs. Shah, Sheffield, and Fife, along with numerous contributing authors who are considered experts in the field of wound care, have collaborated to create the best possible study resource for these important examinations. The content focuses on key information that wound care certifying agencies consider important in their examinations, with self-assessment questions at the conclusion of each chapter to help participants identify areas of comprehension and concepts that require further study.

This all-inclusive study guide includes:

- Thirty-three informative chapters that review the core principles candidates need to know to obtain wound care certification
- New chapter on hyperbaric oxygen therapy by Yvette Hall, Patricia Rios, and Jay Shah
- Added section on PQRS and quality reporting by Dr. Caroline Fife
- A full-length post-course exam complete with answers and explanations
- Comprehension questions with detailed answers at the end of each chapter
- More than 200 color photos, tables, and diagrams
- Clinical pathways with best practice recommendations for the practitioner
- New chapter on hyperbaric oxygen therapy and added section on PQRS and quality reporting
- Guidance on how to choose the certification



“It was my pleasure to review the second edition of the Wound Care Certification Guide. I found the chapters to be well written and organized, building upon the science of wound healing while including practical clinical applications and sample questions. This text should be useful to all wound care professionals, including the novice and expert alike. It will certainly be an important adjunct for anyone preparing for board examinations.”

— Robert J. Snyder, DPM, MSc, CWS; Professor and Director of Clinical Research, Barry University SPM; Past President, Association for the Advancement of Wound Care; Past President, American Board of Wound Management

“The manuscript is the result of a monumental amount of work. I congratulate all involved.”

— Terry Treadwell, MD, FACS; Medical Director, Institute for Advanced Wound Care



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SLIDE 2

Wound Tissue Characteristics

Tissue Type	Healthy Tissue	Non-healthy Tissue
Subcutaneous	Shiny, yellow color, soft, resilient	Gray pallor, hard, not pliable
Fascia	Hard, white, glistening appearance	Soft, dull, stringy, in process of liquefying
Tendon	Shiny, hard, firm	Dull look, soft, partially liquified
Muscle	Bright red, shiny, resilient appearance, contracts when touched	Pale, possibly yellowish color, some tone Dead: dull, swollen, grainy when touched / palpated, when pinched falls apart
Bone	White, shiny, hard	Dull, grayish color, soft

routine billing of unspecified ICD-10-CM codes may result in a reduced reimbursement rate once the data has been messaged and like medical specialties compared in the future. Additionally, a third-party payer could exclude a physician, practice or service from their contract if they feel the information data does not accurately provide the supporting evidence of medical necessity of patient care.

What are the day-to-day risk liability issues? Where does your practice or department fit in to this activity? Can you afford the risk? Slide 3 provides some of the top identified issues.

Learn more by joining the 60-minute informative webinar at <http://woundeducationpartners.com/online-courses/browse-course-list.html>. You will challenge yourself on code selection for diabetic foot ulcers in ICD-10-CM. The user-friendly information will improve your understanding of coding applications, provide answers to many of your coding questions, and reinforce your current knowledge of this topic.

At the end, you should have a clarified understanding of the value this session offers for those working in the wound-care department. Mitigating possible denials or claim holds through accurate, clear, detailed and cohesive clinical documentation is vital to ensure there is an even flow of revenue for services provided. Also, clinical documentation provides a level of granularity and specificity for an accurate ICD-10-CM diagnosis(es) code(s), which supports the reason

for services provided to the patient. This involves not only selecting the diagnoses codes but also sequencing the primary code that represents the reason for the patient's visit.

There is a must for understanding the specific application of the current year's Official Guidelines for Coding and Reporting Outpatient Services as it relates to the bottom line of your department's operation. References are included for your personal library. ■

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SLIDE 3

Unspecified Codes Risk

RISK liability issues may include:

- ▶ Unspecified codes may not support the Level of Acuity with Intensity of Services & Complexity of Care
- ▶ Medical necessity not supported (e.g. debridement procedures)
- ▶ Increase in held or denied claims
- ▶ Increase of re-work of claims by staff
- ▶ Decrease of revenue
- ▶ Overuse may result in being removed from various 3rd party payer plans

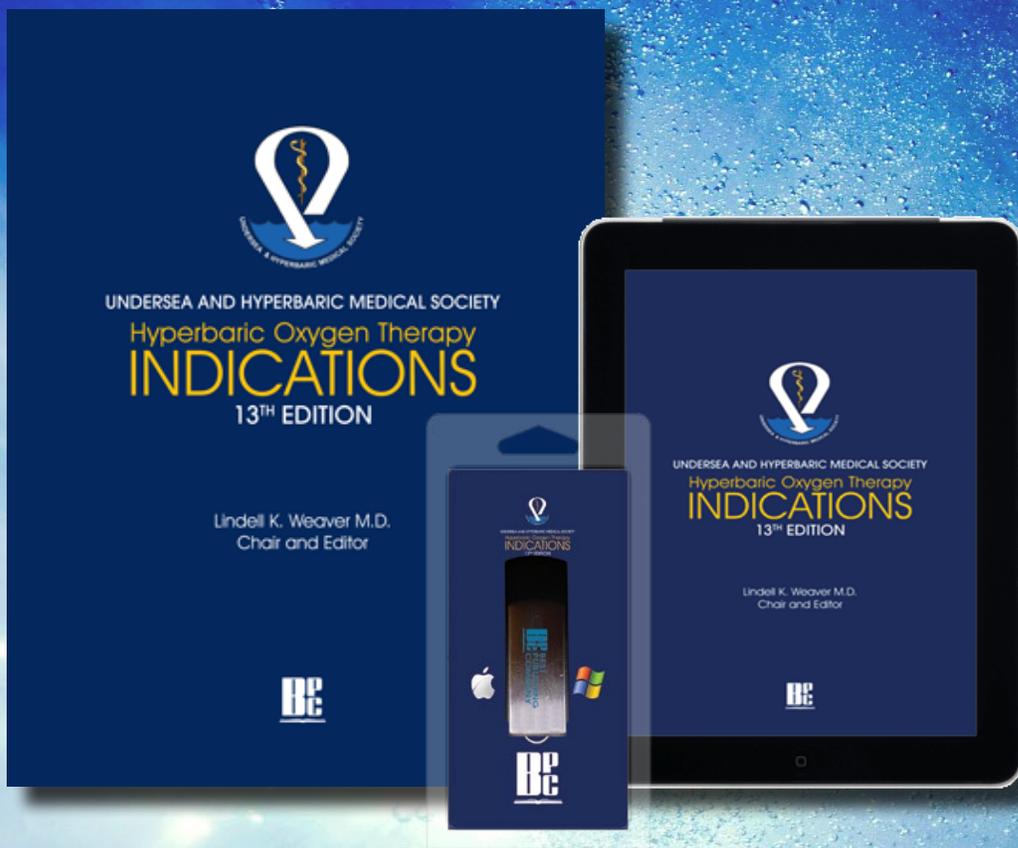
About the Author

GRETCHEN DIXON, MBA, RN, CCS,

is the owner of Professional Compliance Strategies (PCS), LLC, and consults on outpatient departments and physician services. She has provided revenue-cycle compliance audits of services with a focus on wound care department operations for more than 10 years. She holds several credentials including an MBA in healthcare management, an RN with a practicing license in New York, and 23 multistate licensure from Virginia, AHIMA Certified Coding Specialist (CCS), AHIMA ICD-10-CM/PCS Approved Trainer, and is an AAPC Certified Professional (Healthcare) Compliance Officer. A longtime internal healthcare auditor, Dixon identifies issues through audits of D, C, B (documentation, coding and billing) of provided services. The outcome of each audit determines the topics of education to be provided to the staff and physicians, as she proactively believes education is the key to having complete, accurate, and consistent documentation supporting reimbursement for billed services. If you are interested in her services, contact Dixon at gmdixon@cox.net or call 1-615-210-7476.



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- John J. Feldmeier, D.O., FACRO, FUHM and President of the UHMS



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The Pivotal Role of Oxygen in Pressure Injury Etiology and Prevention

By Glenn Butler, CHT; Jody C. DiGiacomo, MD, UHM, FACS; Edward Golembe, MD, UHM, FACCWS; Scott Gorenstein, MD, UHM, FACEP; Bok Lee, MD, FACS

In an effort to safely provide adjunctive hyperbaric oxygen therapy to a larger population of sick and postsurgical patients with compromised flaps/grafts, Life Support Technologies (LST) developed a research program to better understand the etiology of pressure injury (ulcers). (Note: The National Pressure Ulcer Advisory Board has elected to use the term “injury” rather than “ulcer” to describe the initial postischemic injury phase that may lead to an ulcer.)

During hyperbaric therapy, patients must lie on special hyperbaric chamber mattresses that are not as pressure relieving as hospital-provided bed systems that are powered, alternating, or low-air-loss system designs.

To better understand the biomechanical and biochemical mechanisms relating to pressure injury, LST developed systems that combined mattress interfacial pressure mapping with spectrographic oximetry. This new technology provided simultaneous real-time pressure and deep-tissue blood perfusion as measured by multiple, near-infrared sensors for tissue hemoglobin saturated oxygen or rSO_2 . We utilized this technology to evaluate 16 powered and nonpowered clinical mattress systems dominating the medical market. The human subject research portion of the program to date has involved more than 200 clinical evaluations using male and female subjects ranging in age from 18 to 65 years old.

Off-Loading Technologies, Inc. (OLT) then utilized this technology to design, evaluate, and patent the OXY-MAT™ series of offloading systems engineered to naturally optimize patient blood-perfusion and deep-tissue oxygen tensions over a wide range of patient/mattress interface pressures.

Traditional Pressure Mapping

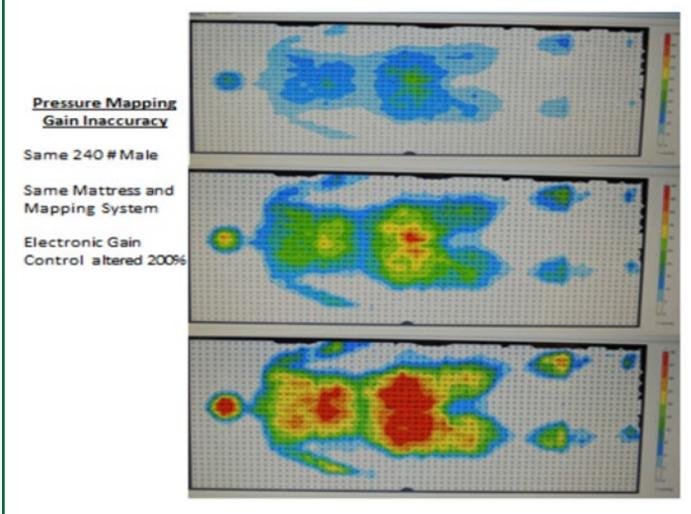
In developing the system, we first turned to the industry standard of pressure-mapping systems available on the market. This basic technology has been used over the past 50

years to design and promote commercially available clinical and retail mattress systems.

Commercial pressure-mapping systems often contain transient data variables that make them a concern for the reliable evaluation of pressure-offloading measurement. As Figure 1 shows, we placed a 240-pound male on a nonpowered mattress equipped with a conventional commercial pressure-mapping system. In only 30 seconds, we inadvertently adjusted the electronic gain up 200 percent and noted the changes in recorded interface pressure. This event represented an unacceptable opportunity for experimental error. Our modified pressure-mapping system has been designed to eliminate any ability to change adjustments between baseline and serial measurements.

Traditionally, to evaluate mattress systems, pressure data have been extrapolated over time using empirical algorithms (Hunt et al.) to estimate time-to-tissue ischemia and to predict

FIGURE 1. Effect of Gain



pressure-injury (ulcer) development. In an effort to better understand and measure internal tissue pressure changes, several invasive or indirect methods have been used.

Invasive pressure-sensing catheters are unsuitable for use in human volunteers. Indirect methods include ultrasonic and magnetic resonance with chemical tagging where the displacement of tag lines allows one to calculate internal tissue pressure deformation.

Classical teaching states approximately 30 mmHg tissue pressures would result in capillary blood vessel collapse, a reduction of blood flow carrying nutrients (i.e., oxygen), eventually leading to ischemia. Using these new evaluation technologies, however, we often see much higher interfacial pressures in basically healthy individuals without developing any pressure injury, while many compromised patients with comorbidities begin to have ischemic changes almost immediately despite best efforts to offload the patient. Clearly, a lot more is going on than we fully understand.

Deep-Tissue Oxygen Tensions

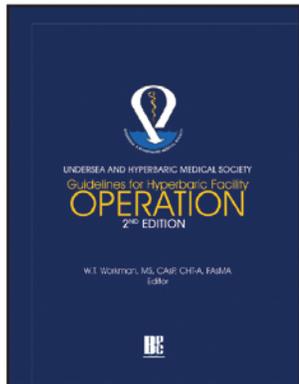
We soon realized measuring pressure alone was not a meaningful predictor of tissue ischemia — reperfusion (I/R) injury leading to a pressure injury. Deep-tissue blood perfusion as measured by oxygen tensions is the only reliable real-time indicator of relative tissue ischemia leading to a true reperfusion injury, pressure injury and ulcer development.

Colin, Loyant, et al. measured transcutaneous oxygen tensions on the sacrum of 20 healthy individuals positioned on 5 different mattress types. That study demonstrated lower oxygen tensions as compared with a control as subjects were exposed first to a standard mattress and then several other mattresses. Oxygen tensions improved on foam and maximized on a water-equalizing mattress.

Pressure is only one of the contributors to the pathophysiology and relative risk of pressure ulcer development. Factors such as microvascular disease, blood perfusion, BMI, nutrition, and comorbidities affecting nitric oxide (NO) autoregulation/

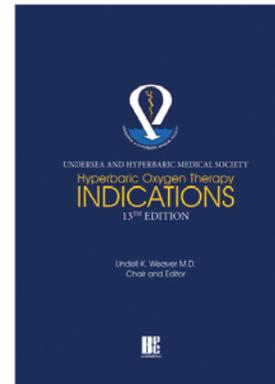
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vasodilatation and the physiological management of reactive oxygen species (ROS) all play an important part in relative pressure injury risk.

Do Age and Comorbidity Affect Risk?

Our test-data trending demonstrates young and healthy individuals are able to sustain higher tissue pressures longer and still demonstrate normal active hyperemic response and blood-oxygen tensions above the ischemic threshold of 40 mmHg and with a reliable return to baseline oxygen tensions. Conversely, some older patients with comorbidities that can compromise blood autoregulation appear to lose tissue-oxygen tensions faster under moderate pressure (60 mmHg) and do not demonstrate a normal active hyperemic response. They tend to maintain lower pressure tissue-oxygen levels than starting tensions. Research continues to evaluate this trend.

Pathophysiology of Pressure Ulcer Development

The prime causal factor for the development of pressure injury and subsequent ulcers consists of excessive tissue pressure-loading sustained for time periods sufficient to cause pressure-prone tissue to become ischemic, then hypoxic, leading to reperfusion injury.

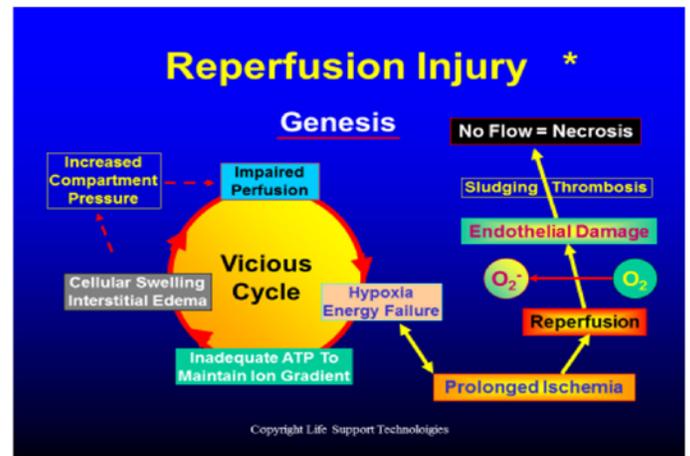
Since nearly all patients are in bed for eight hours or more, the mattress system selected for clinical use becomes a significant variable in the reduction and/or relief of pressure on the patient's body, particularly over bony prominences. Any increase in mechanical stress (pressure and shear) further affects the availability of nutrients, such as oxygen, to susceptible tissues.

Ischemia leading to hypoxic injury is the result of decreased blood flow to cutaneous tissue after prolonged periods of elevated tissue interface pressure. The resulting reperfusion injury causes neutrophil capillary endothelium adherence, cell rolling, sludging, and clotting that inhibits/occludes blood nutrient and oxygen supply.

This ischemia-reperfusion injury cycle (Figure 2) decreases blood flow and increases the hypoxic biochemical cascade that forces tissue cells to use anaerobic pathways to produce ATP energy. This causes more lactic acid to accumulate, resulting in greater acidosis, as well as increased quantities of hydrogen ions and potassium around the cell.

In normal individuals, this biochemical cascade of metabolites and oxygen radicals should lead to nitric oxide release and up-regulates other vessel vasodilators (active hyperemia) that promote increased fresh blood flow with oxygen and other nutrients to the tissues.

FIGURE 2



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A review of these physiological mechanisms helps us to appreciate why deep-tissue oxygen tensions are the only meaningful real-time indicator for pressure injury risk and prevention.

The Pressure/Oxygen Relationship

It is generally understood there is a close correlation between an increase in tissue pressure and a reduction in blood flow, with approximately 30 mmHg pressure resulting in capillary vessel collapse.

Our testing has demonstrated an increase in tissue pressure and that tissue's blood flow as measured by oxygen saturation often did not inversely correlate. A high interface pressure often did not result in a lower tissue-oxygen saturation value leading to ischemia and a lower tissue interface pressure did not always result in better blood perfusion and higher oxygen saturation.

Simultaneous Pressure and Oxygen Measurements – A More Complete Picture

Simultaneous measurement of both pressure and deep-tissue oxygen tensions provide a more complete picture of the effects of pressure and blood-flow reduction resulting in lower oxygen tensions leading to ischemia. To develop a dependable system, we modified and integrated near infrared spectrographic oximetry used to measure brain oxygen during anesthesia (tissue-oxygen saturation) into our existing, improved pressure-mapping system. This served as an indicator of blood perfusion by the direct measurement of tissue-oxygen tension. The combination of simultaneous pressure and oxygen tension has permitted us to evaluate offloading mattress system designs and to safely determine a given patient's tolerance to pressure without risking pressure injury development.

After substantial evaluation and human testing, we have demonstrated that tissue pressure and oxygen tensions are not necessarily inversely proportional and near normal perfusion can exist under high interfacial tissue pressures (>100 mmHg) as could be clinically experienced in healthy individuals.

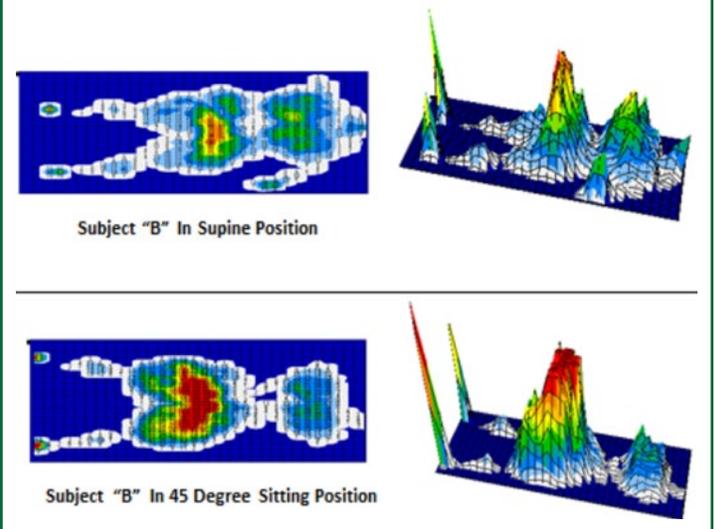
Weight Redistribution Analysis

Figure 3 is an example of an LST pressure-only weight distribution map on the same subject and mattress system lying down and then sitting with the bed raised to a 45-degree position.

These two maps depict the weight transfer from the torso in the supine (laying down) position and weight transfer down to the sacrum and ischium when in a 45-degree sitting position. With many bedded and wheelchair patients spending a majority of time in a sitting or upright position, the sacrum, ischium and heels are a primary concern.

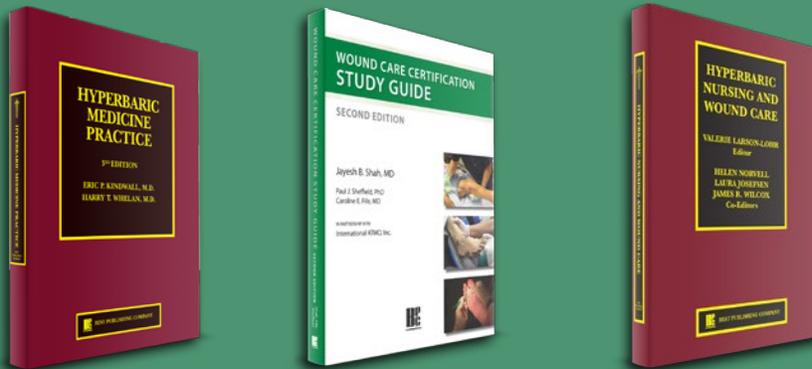
But what is actually happening to the blood perfusion and/or any ischemic changes taking place in these at-risk tissues? Is there any difference in blood perfusion values and pressure injury risk between a 45-year-old brittle diabetic motorcycle rider (smoker) with a broken hip and a healthy 86-year-old who slipped on ice and broke a hip?

FIGURE 3



Simultaneous Pressure/Oxygen Measurements in Humans

Figure 4 shows an example of an LST simultaneous interface pressure/tissue oxygen analysis. For clarity in this graph, we are looking only at ischium pressure and oxygen values. Our standard studies simultaneously include scapula, ischium, sacrum, trochanter, and heels.



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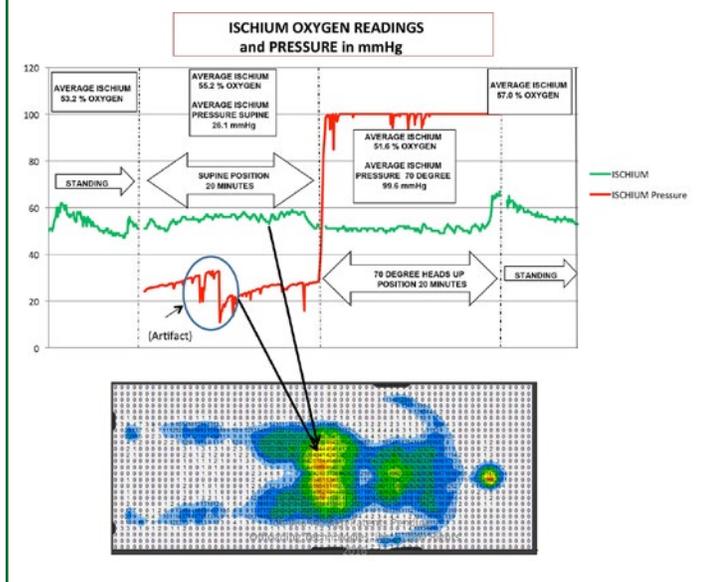
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FIGURE 4



In Figure 4, the subject goes from a standing position to supine for a 20-minute period, then is elevated to a 70-degree reclining position for an additional 20 minutes and then returns to a standing position.

Note that in both standing and supine positions, ischial tissue oxygen averages 55 percent, while ischial pressure averages 26 mmHg in the supine position. In the 70-degree position, the subject's weight transfers to the ischium and the average interface pressure rises to 99 mmHg, while the ischium oxygen tension only decreases to 51 percent.

The net pressure increase from the supine to sitting position is more than 280 percent, but the oxygen tension only decreases by 6.5 percent from the supine position. This is an effect of the human body's ability to autoregulate blood perfusion as measured by oxygen saturation.

This is a typical example of how pressure and blood perfusion are not inversely proportional in healthy subjects able to carry out normal autoregulation/active hyperemia. Conversely, it helps us better understand how age and comorbidities compromise blood perfusion autoregulation, hyperemia, I/R, and pressure injury development.

Active Hyperemia

Active hyperemia is a normal physiological process that automatically compensates for reductions in blood flow due to transient vessel occlusions or increased tissue-interface pressures, such as prolonged sitting in healthy persons. As we sit, receptors sense our muscles becoming ischemic and congested with metabolic byproducts. We then unconsciously shift our weight to allow normal active hyperemia to vasodilate the muscles and flush the tissues

with fresh blood. We can repeat this process hundreds of times a day without long-term effect.

As persons become older, develop comorbidities such as diabetes, associated neuropathy, paraplegias or compromised mentation, they become less able to initiate normal autonomic active hyperemia vasodilatation and become more susceptible to pressure-related ischemia leading to hypoxia, reperfusion injury and necrosis.

Reactive Hyperemia

Reactive hyperemia occurs after the normal physiological mechanisms of active hyperemia are exhausted. Reactive hyperemia is the transient uncontrolled increase in blood flow that occurs following some prolonged period of ischemia as might occur after the removal of a tourniquet.

The only meaningful variables seem to be patient comorbidities that down-regulate autonomic vasodilation and tissue-oxygen tension-recovery times that allow tissues to become ischemic long enough to induce a true reperfusion injury.

When Repetitive Hyperemia Leads To Ischemia

If the patient's position is changed often enough after a mild ischemic incident (two-hour nursing repositioning), some focal tissue pressure will be released and there will be moderate active hyperemia and blood-vessel dilation.

This increased blood flow flushes out metabolites/free radicals, and a normal blood flow due to autoregulation will resume. This is a normal process.

Excessive and repetitive ischemia, hypoxia, and then repetitive reactive hyperemia will lead to a true ischemic / reperfusion injury and neutrophil adherence to the capillary endothelium that then result in cell rolling, sludging and progressive blood flow reduction/occlusion.

This ischemic/reperfusion injured tissue becomes increasingly compromised and more susceptible to pressure injury/ulcer development upon repeating this pressure reinjury cycle.

For patients with comorbidities, the anatomical areas most susceptible to pressure and shear are the scapula, sacrum, ischium, trochanter and heel. For seated and wheelchair patients, the areas most impacted are the buttocks and the ischium.

The role of shear forces developed in sitting with respect to tissue trauma in the region of the ischial tuberosities may be significant in pressure ulcer causation. Prior study results have shown that cutaneous pulsatile flow measured at the

buttocks of the geriatric hospitalized patient and seated paraplegics is considerably reduced compared with that of healthy subjects.

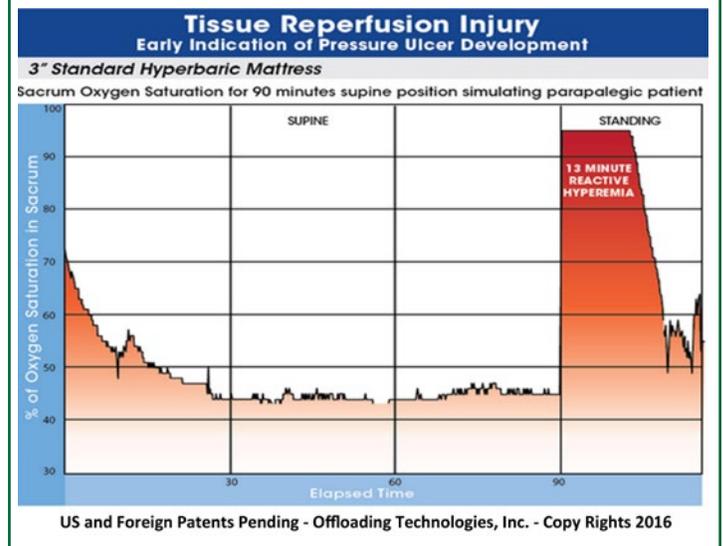
Average skin shear values developed by a geriatric hospitalized group were three times that of a young, healthy group. It also has been shown that the sitting shear force developed by paraplegics is considerably greater than corresponding measurements of normal subjects. We think this is due in part to neuropathy effecting muscle tone and normal active hyperemia vasodilation.

Reactive Hyperemia Demonstrated

Using our simultaneous Near-Infrared Spectrographic Tissue Oximetry/Interface Pressure System, we have noted that about 80 percent of normal test subjects demonstrated some reactive hyperemia (RH) of the sacrum, in particular, upon standing after being supine on a mattress surface for a 90-minute test period.

As an extreme example of this phenomenon, the LST lab group tested a standard, first-generation, three-inch, monolithic memory foam mattress designed for use in monoplace hyperbaric chambers. This testing was initiated because of LST's clinical concerns regarding this mattresses' ability to

FIGURE 5



adequately offload compromised patients receiving hyperbaric oxygen therapy over a two-hour supine period. (Figure 5)

The degree of reactive hyperemia was sometimes significant. For example, Subject #4 of eight subjects tested in this series went from a pretest sacral area oxygen saturation value of 76 percent, then down to an averaged value of 45 percent over

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a 90-minute period while the subject remained supine and as immobile as possible to simulate a paraplegic/insensate patient. Many subjects (70 percent) experienced ever-increasing to eventual pain/spasms in the sacral area during this 90-minute supine and immobile test period.

Upon standing, Subject #4's sacral oxygen levels went up immediately to more than 95 percent oxygen saturation (instrument full scale) and remained in that fulminant reactive hyperemic level for a 13-minute period before normalizing (autoregulation) back to 65 to 68 percent (below test-start oxygen baseline). On standing, the subject noted an extreme sensation of heat and interruption of pain. The sacral oxygen tension eventually stabilized at 62 percent. This was typical of tests repeatedly demonstrating lower baseline oxygen and is considered the hallmark of a true ischemic reperfusion injury.

Compromised Patients are at Greater Risk

During this same test series, a paraplegic subject's sacrum became so ischemic we had to interrupt the test after only a 30-minute period. We again attribute this to neuropathy affecting muscle tone and normal active hyperemia vasodilation. This subject had no pain sensation. This further demonstrates that neurologically compromised and paraquadriplegic patients are physiologically incapable of functional active hyperemia beyond any sensation of ischemic pain. We postulate that this magnitude reactive hyperemia is a hallmark of reperfusion injury and an early indicator of pressure injury development.

Repetitive Ischemia/Reperfusion Injury Syndrome

We theorize that significant and repetitive changes (>30 percent) in oxygen tensions can induce a reactive hyperemia

and will likely — over time — result in a repetitive ischemic/reperfusion injury syndrome that forces tissues into anaerobic cell respiration pathways, pressure injury and eventual necrosis. Figure 6 represents a theoretical progression of I/R over patient turning cycles.

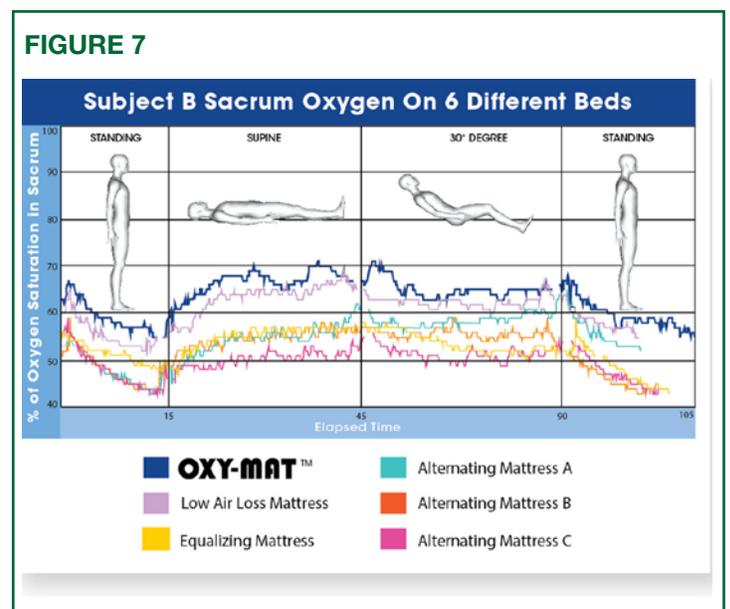
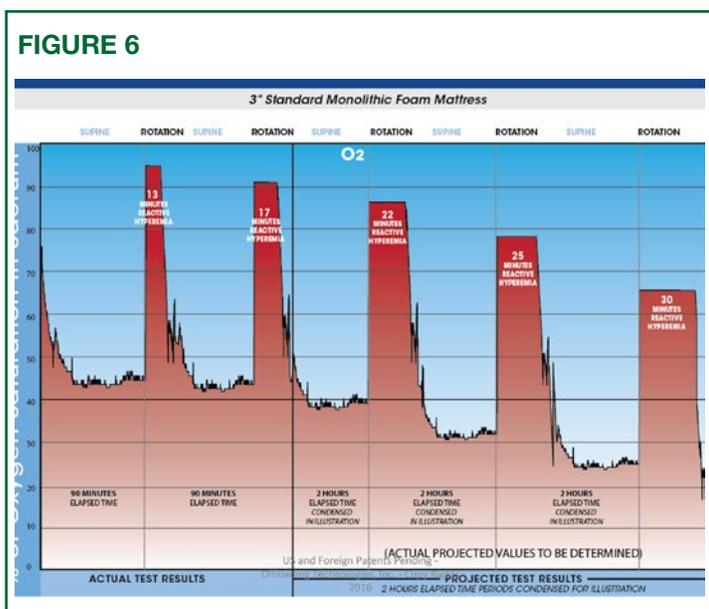
The three peaks on the right side of the graph are extrapolated from actual test results depicted in the two graph peaks at the left side of the graph. These two events (the first peak is from Figure 5) were 90 minutes apart with a 13-minute and then a 17-minute uncontrolled reactive hyperemia of the subject's sacrum.

Based on our other test results to date, we believe that this extrapolated data is representative of the actual progression of repetitive ischemia leading to significant I/R injury and neutrophil adherence to the capillary endothelium. The authors are presently developing an animal model to further demonstrate and research this phenomenon.

Whole Body Active Hyperemia

Another test program was designed to compare sacral oxygen tensions on the same subject while lying on six different mattress systems. Each test subject demonstrated a unique pattern of somatic active hyperemia (SAH) when going from a standing to a supine position, then sitting in a 70-degree position and back to standing.

This pattern was unique to each subject, and the autoregulation wave pattern results were also constant, regardless of the bed surface. Only the oxygen-tension shifted based on the mattress type being tested. Note the unique signature waveform regardless of the mattress type being tested.



Example: Subject B's sacral oxygen saturation has the same pattern signature on each of six different types/manufacturer mattress surfaces. Figure 6 shows standing time before the 20-minute supine test measurement, then test measurements over a 20-minute period while in the 30-degree position, and then again when standing.

Conclusions

1. The preponderance of the literature and our research support our conclusion that pressure alone has demonstrated not to be a reliable real-time indicator of mattress design superiority or to measurably reduce pressure injury risk or ulcer incidence.
2. The pathophysiology of pressure ulcer development is just beginning to be understood. The true dynamics of repetitive ischemia/reperfusion injury as they relate to deep-tissue oxygen/nutrient supply and cell metabolite management are critical to pressure injury prevention, ulcer development and wound care.
3. Reactive hyperemia must be avoided. The time and tissue interface pressures required to induce an ischemic/reperfusion (I/R) event vary significantly from patient to patient with age, comorbidities and functional circulatory autoregulation.
4. Repetitive I/R injury syndrome data support the hypothesis that repetitive reactive hyperemia inducing I/R produces ever-increasing neutrophil adherence to capillary endothelium that progressively reduces tissue-perfusion and tissue-oxygen tensions. This time and pressure is very variable and requires additional study.
5. Repetitive ischemic/reperfusion injury syndrome is a term the authors developed to describe the cyclic changes in blood flow in an immobile patient's tissue contact area under repeated pressure and offloading cycles when a patient is periodically turned. This phenomenon can also be induced by a misadjusted alternating mattress.
6. Medical institutions are being sold an ever-growing array of increasingly complicated and costly powered mattress systems ostensibly developed to further reduce pressure injury ulcer risks with each new design. New mattress designs are still based on interface pressure mapping and have not been able to produce measurable improvements in patients' deep-tissue, oxygen-saturation levels, improved patient comfort or reduced insomnia.
7. All types of mattress systems must be designed to either permit patients to induce normal active hyperemia by patient movement or to simulate movement in insensate/nonmoving patients to induce normal active hyperemia for them. It is estimated that more than 90 percent of

hospital and long-term-care facility patients are capable of normal active hyperemia and should be placed on nonpowered equalizing mattress systems.

8. A unique somatic active hyperemia signature was observed in each of our test subjects as they were placed in supine, sitting and standing positions. This could be repeated one year later and the subject identified solely by the shape of their oxygen saturation curves.
9. Powered mattress systems were compared with the Oxy-Mat™ in independent clinical trials. Oxy-Mat™ has been credited with improved patient sleep, some reduction in pain medication and the improved ability to participate in P/T rehabilitation.
10. The overutilization of powered mattress systems likely represents a significant unnecessary cost in health care and may be a contributing factor in clinical outcomes and longer length of stay due to sleep deprivation. ■

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About the Authors

GLENN BUTLER (Founder/CEO LST) has more than 45 years of experience in military and commercial diving and clinical hyperbaric operations. He has developed and/or operated more than 20 major hospital-based hyperbaric medicine programs internationally.



The Life Support Technologies group (LST) provides hospital-based elective and emergency wound care and hyperbaric medicine services around the New York Tri-State area. Butler was the principal consultant when LST was sole-sourced by NASA's Johnson Space Center to engineer zero-gravity simulation breathing systems and develop new oxygen safety standards for the astronaut training program. He continues to lead the group's efforts in its ongoing role with NASA.

He was a founding member of the National Board Certification for Hyperbaric Technologists and is a member of the UHMS Hyperbaric Safety Committee. He has authored 12 HBO₂-related U.S. patents and published more than 45 related documents. He is a founding partner of Off-Loading Technologies Inc, the design/manufacturing group for the OXY-MAT products.

DR. BOK LEE (Chairman of Clinical Research and Development) is a distinguished, internationally recognized vascular surgeon with more than 200 published scientific articles and 9 books to his credit. His latest books are *The Wound Management Manual* (McGraw-Hill, 2005) and *The Spinal Cord Injured Patient* (Second Edition, Demos, 2002).



Lee served on the Board of Governors of the American College of Surgeons for 1993 to 1999. He has been professor of surgery (NY Medical College, Valhalla, NY), director of Surgical Research (Sound Shore Medical Center, New Rochelle, NY) and adjunct professor (Rensselaer Polytechnic Institute, Troy, NY). Lee is actively involved in conducting hyperbaric research with the LST team.

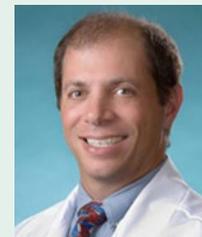
EDWARD GOLEMBE (Skin Integrity Service, Westchester Medical Center, NY) is board certified in plastic surgery and hyperbaric medicine. He belongs to the American Society of Plastic Surgeons, New York Regional Society of Plastic and Reconstructive Surgeons, Medical Society of the County of Kings, Undersea and Hyperbaric Medical Society, and the New York State Hyperbaric Medical Society. He is also scuba certified and a DAN referral physician.



JODY CHRISTOPHER DIGIACOMO (director of Surgical Critical Care, associate director of Trauma, Nassau University Medical Center, NY) has been board certified in hyperbaric medicine since 2004. He has published more than 33 medical articles. DiGiacomo's academic appointments include associate professor of surgery at St. George's University School of Medicine (Grenada); associate professor of surgery at State University of New York, Stony Brook; adjunct clinical associate professor of surgery at the New York College of Osteopathic Medicine; and assistant professor of surgery at the University of Pennsylvania School of Medicine (Philadelphia, PA). He is also a Fellow of the American College of Surgeons.



SCOTT GORENSTEIN (clinical director, Division of Regenerative Medicine Winthrop University Hospital Mineola, NY) is board certified in emergency medicine and hyperbaric medicine. He is clinical director, Division of Regenerative Medicine at Winthrop University Hospital in Mineola, NY, and assistant clinical professor of surgery at Stony Brook University in Stony Brook, NY. He is a Fellow of the American College of Emergency Physicians, president-elect of the Northeast Chapter of the Undersea and Hyperbaric Medical Society (UHMS) and a Fellow of the American Professional Wound Care Association.



Undersea and Hyperbaric Medicine Journal **Will Publish Special Issue on TBI Studies**

By Renee Duncan

The Undersea and Hyperbaric Medical Society will publish a supplemental issue of its member publication, the *Undersea and Hyperbaric Medicine* journal, to discuss results and findings in baseline publications for studies on traumatic brain injury (TBI). With publication scheduled for summer 2016, author teams are making final refinements to the texts. The issue will consist of 10 papers plus a summary of recommendations.

The manuscripts document work from the Brain Injury and Mechanisms of Action (BIMA) of HBO₂ for Persistent Postconcussive Symptoms after Mild Traumatic Brain Injury (mTBI) study. One manuscript describes interventions in mild traumatic brain injury without the use of hyperbaric oxygen (HBO₂).

The BIMA study, sponsored by the U.S. Department of Defense and held under an investigational new drug application by the Office of the Army Surgeon General, is one of the largest and most complex clinical trials of hyperbaric oxygen for postconcussive syndrome (PCS) in a military population. With the increase of improvised explosive devices, PCS has become a growing concern in the military. While body armor can help shield the body, explosions can cause a range of concussions and brain injuries.

“Compared to previously published HBO₂ trials for PCS, this randomized, double-blinded, sham-controlled exploratory study has a longer duration of follow-up

and more comprehensive battery of assessments,” notes Dr. Lindell K. Weaver, study director and lead principal investigator.

The study looks at active-duty military and veteran personnel with mild TBI sustained while on active duty, who remain symptomatic three months to five years later. Assessment tools include testing of vestibular, audiologic, neuropsychological, autonomic, neurological and ocular functions; neuroimaging; sleep studies; physical exams; and laboratory testing.

Primary objectives of the BIMA study are as follows:

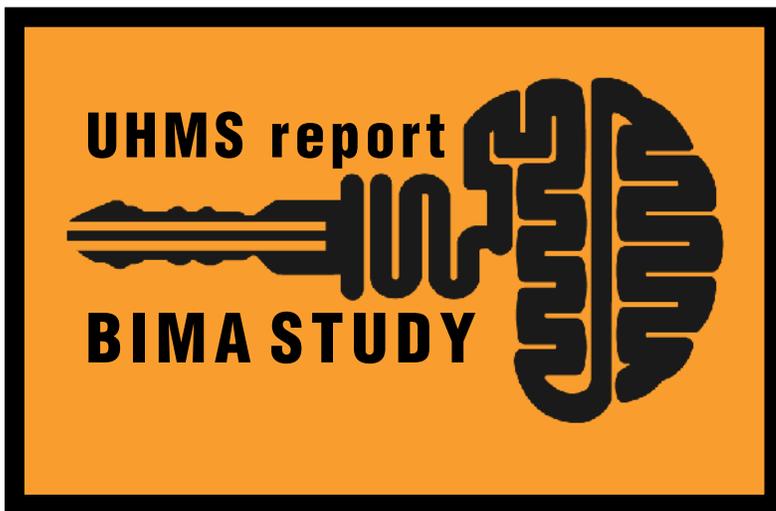
- To identify endpoints for future trials of potential treatments for PCS
- To describe brain function and anatomy of the military population with PCS at baseline and across time
- To explore potential associations between changes in brain function, anatomy, and participant-reported outcomes



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properties of potential outcome measures for the design of future studies,” Weaver said, adding several outcomes have been identified in these baseline publications that show as candidate outcomes for PCS. These will be the focus of further investigation following the close of the study.

“Having these BIMA specialty papers published will facilitate manuscript preparation immensely when study results become available,” Weaver said. As with all papers that appear in the *UHM* journal, these TBI-related texts received peer review.

Study results of HBO₂ vs. sham treatments in the BIMA study are not yet available.

Secondary objectives are to evaluate the safety of HBO₂ and sham interventions and to identify practical issues in instituting study assessments.

The study has identified outcomes that suggest baseline abnormality or deficit. “The outstanding adherence to interventions and study visit completion through 12 months positions the BIMA study to provide Class I evidence on the potential mechanisms of action of HBO₂ as well as the

Editor-in-Chief Dr. Enrico Camporesi notes, “We plan to follow with another issue discussing hyperbaric oxygen therapy trials for TBI in approximately a year.”

The issue will be available through the UHMS website under the subheading “Publications.” UHMS will announce availability in its member newsletter, *Pressure*, available to the public on the UHMS website at www.uhms.org, and via email to the membership. ■

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Dr. Lee Ruotsi and Laura Dewey in the St. Joseph Campus Clinic

CLINIC IN FOCUS

Catholic Health Advanced Wound Healing Centers

Continuing our series of interviews featuring outstanding hyperbaric and wound care centers around the world, we spotlight in this issue the Catholic Health Advanced Wound Healing Centers in Buffalo, New York.

If an accredited facility, how has seeking UHMS accreditation affected your clinic?

In 2010, Catholic Health opened its first Advanced Wound Healing Center under the medical direction of Dr. Lee Ruotsi. Although we had a consulting agreement with a management company, our model differed from other programs in our geographical area. In our program, each center is staffed by system-employed medical directors, RNs, CHTs, office staff and a director. Our second center opened in early 2011, when Dr. William Lagaly was recruited from Little Rock, Arkansas. Over the next four years, the staff and medical directors worked to build a reputation for quality wound care and high patient satisfaction, boasting strong clinical outcomes.

During 2014, Catholic Health submitted our application, and over the following year, we worked diligently to prepare for the survey. In late 2015, we achieved UHMS accreditation. The process was educational not only for us but also for our health system as a whole. It forced us to examine our business and clinical processes from a unique viewpoint: the hyperbaric patient perspective. During our preparation for the survey, we found that although our previous practices often met the minimum criteria required by UHMS, there was typically room for improvement. The survey also allowed the Advanced Wound Healing Centers to gain more visibility in our very busy, robust health system. Our quality, safety and senior leadership teams gained a better perspective into the challenges our department faces, and the accreditation fostered a sense of unity within our organization. The accreditation process gave senior leadership an in-depth understanding of how complex hyperbaric oxygen therapy is and what a strong program we had developed.

What are the most common indications treated at your clinic?

The most common indications treated at our clinic are diabetic foot ulcers, late effect of radiation, and failed or compromised skin grafts.

What is the most memorable treatment success story that has come out of your clinic?

“Of many memorable treatment successes, one comes immediately to mind: a 67-year-old female who was in our care in 2014,” Dr. Lee Ruotsi said. “She originally presented with typical chronic venous leg ulcers and was treated with multilayer compression wrapping to closure. During her initial consultation, a history of gynecologic malignancy in 1995 was noted; however, there was no further discussion of this at that time.

On one of her last visits for her leg ulcers, further history-taking and conversation led to a diagnosis of hemorrhagic radiation cystitis secondary to extensive external beam radiation therapy. She related her problems began about 8-9 years after the radiation treatments, and initially the symptoms consisted of mild discomfort, urgency, and occasional bleeding. As the years went on, however, the discomfort turned to pain, the urgency became incontinence, and the occasional bleeding became daily frank bleeding with clots. On a scale of 1-10, the adverse impact on her life was a 15. She had not left her house in years to attend social events or to go out for dinner, and she would not even sit on her own furniture unless she put down pads first.

We initiated hyperbaric oxygen therapy at 2.5 ATA for 90 minutes 5 days per week for a total of 40 treatments. As the weeks progressed, she became less and less symptomatic. The incontinence slowly resolved as did the pain, and the bleeding declined to occasional spotting. On her last visit she said the adverse impact was now a 2 out of 10. She was back to going out for dinner and sitting unprotected on her furniture. She thanked us, a bit tearfully, for what she felt had been a miraculous recovery. For us, it was a most gratifying reminder of the impact of hyperbaric oxygen therapy.

Do you work with a management company? If so, which one?

Catholic Health manages all operations internally, but we do have a consulting contract with Precision Healthcare.

If you had to pick one thing to attribute your clinic's success to, what would it be?

As a healthcare system, Catholic Health is dedicated to the needs of the community we serve. A lack of accessible wound care services was identified in the Western New York area. In response to this, we opened the Advanced

Wound Healing Centers. Being a part of such a large, yet community-centered, healthcare system allows our program to remain patient focused while being able to offer access to a broad network of collaborative services. We believe these are the key factors to providing the best possible patient care, thus achieving the highest level of successful outcomes.

What is one marketing recommendation that you can make to help clinics increase their patient load?

Our largest barrier to new patient referrals is a lack of wound care and hyperbaric oxygen knowledge within the medical providers in our area. Therefore, the best source of marketing for our clinic has been to utilize a wide range of educational activities. Our physicians speak to the medical community multiple times each year, including presenting to residents, nurses, and home-care clinicians. In addition, Catholic Health hosts an annual wound symposium — a full day, continuing-medical-education event that we market to all levels of caregivers on the latest advances in wound care. We also present relevant wound-care topics to the general public in support-group settings. Every educational event is an opportunity to market the services offered in our clinics. ■

CLINIC DETAILS

Clinic Name: Catholic Health Advanced Wound Healing Centers

Location: Buffalo, N.Y. (two locations)

Website: www.advancedwoundcenters.com

Phone: Sisters Hospital, St. Joseph Campus: 716-891-2570; Mercy Ambulatory Care Center: 716-828-2330

How long in business: Opened in 2010

How many chambers: 3 at Sisters Hospital, St. Joseph Campus; 2 at Mercy Ambulatory Care Center

Chamber types: Sechrist Monoplace 3600

How many physicians/nurses/CHTs: 2 full-time physicians, 1 nurse practitioner, 5 RNs, 2 LPNs, 3 CHTs

Medical directors: Dr. Lee Ruotsi at Sisters Hospital, St. Joseph Campus; Dr. William Lagaly at Mercy Ambulatory Care Center

Date of UHMS accreditation: November 2015



From left: Villa on Dunbar Rock Vice President of Business Development and Operations Mark Walker, Dr. Matias Nochetto, Director of Medical Programs at DAN, Villa on Dunbar Rock General Manager Denis Midence, Villa on Dunbar Rock CEO and owner Kirk Laney

Villa on Dunbar Rock Funds Hyperbaric Chamber Installation in Guanaja, Honduras

March 8 was a proud day for people of Guanaja and the Bay Islands when dignitaries gathered for the dedication ceremony of Guanaja's first hyperbaric chamber and clinic, Clinica Hiperbarica.

This project began two years ago when Dr. Matías Nochetto, director of medical programs for Divers Alert Network (DAN), proposed the idea for a chamber to Guanaja's Mayor Spurgeon Miller, Villa on Dunbar Rock owner and Chief Executive Officer Kirk Laney, and Vice President of Business Development and Operations Mark Walker.

The clinic, now officially open, was made possible through a grant from DAN and generous contributions from Laney

and Delia Cornett, whose late husband, Donald Cornett, was Laney's longtime business partner. The dedication plaque inside the clinic building reads, "In honor of Don Cornett, whose drive for excellence in life, thoughtfulness for others, and selfless acts provided the inspiration for donation of this hyperbaric chamber to the people of Guanaja."

"Our long-term goal is to educate our staff and neighboring resorts about the benefits of continuing to give back to Guanaja," Walker said. "One of the best ways we can do this is by taking care of its people and promoting a healthier life for them, beginning with the Clinica Hiperbarica. Through the combined efforts of many people and agencies, your

municipal government can now operate and offer hyperbaric oxygen therapy not only to the people of Guanaja but those of the Bay Islands and the nearby Honduran mainland coast as well. Equally important is the positive impact that knowledge of an accessible chamber will have in promoting dive tourism to the island from around the world.”

Funding of the clinic is just one of the many ways that the Villa on Dunbar Rock is giving back to the community and the people of Guanaja. The Villa is also responsible for establishing, with the assistance of many supporters, Guanaja’s first Library and Learning Center in Bonacca, which has been stocked with more than 10,000 books donated by guests from the Villa.

Most recently, they dedicated countless hours reestablishing the dive site mooring ball system throughout the national park system. More than 50 dives sites have been set up on the system to protect the reefs.

The Villa on Dunbar Rock dive vacation resort is located approximately 7 miles east of Roatan and 36 miles north of the mainland of Honduras. Perched on a cay with 360-degree views of Sandy Bay, Guanaja, and surrounding cays, this unique resort blends beauty and serenity in a laid-back,



inviting atmosphere. The Villa covers 15,000 sq. ft. and includes a PADI/SSI dive center that offers concierge dive services, two spacious dive boats, and a reef system you can practically call your own. Amenities include 8 ocean-front rooms and 2 ocean-front suites, a white-sand beach, infinity pool and pool bar, cardio room, great room for dining and lounging, and porch swings and hammocks throughout. The resort offers 7-night packages for Saturday to Saturday stays that include accommodations, hearty meals, diving, and more. For more information, visit www.dunbarrock.com. ■



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About the Author:
Dr. Neil Hampson, a Seattle native, is a retired pulmonary, critical care, and hyperbaric medicine physician. He has an international reputation in hyperbaric medicine, specifically in the area of carbon monoxide poisoning. During his clinical career, he treated more than 1,000 patients with carbon monoxide poisoning and published numerous papers in medical journals about the condition.

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Not All Swelling Is Equal: Differential Diagnosis of Edemas

By Heather Hettrick PT, PhD, CWS, CLT, CLWT

This article is the first in a series of three that will address the clinical importance of recognizing edema for differential diagnosis. The focus of this article will be on recognizing edema and performing appropriate clinical tests to help with clinical diagnosis. The second article will focus on the pathophysiology of lymphedema, and the third article will cover common complications seen with lymphedema.

Edema is a common condition many of us have experienced. It is seemingly benign and self-limiting. By definition, edema is the presence of abnormal amounts of fluid in the extracellular tissues. Typically, an equilibrium is maintained through a delicate balance between hydrostatic and osmotic pressure inside and outside the blood vessels.

Generally speaking, hydrostatic pressure is determined by blood pressure and the effects of gravity, whereas osmotic pressure is determined by the concentration of protein inside and outside the vessels. Under normal circumstances, the hydrostatic pressure that pushes fluid out of the veins is slightly higher than the osmotic pressure that keeps fluid in. This results in a slight loss of fluid into the interstitial space. Subsequently, this fluid is taken up by the lymphatic capillaries and returned to the venous circulation as lymphatic fluid. Daily fluctuations exist, but for the most part, the human body does an exceptional job maintaining fluid balance.

This may seem straightforward, but it isn't. There are more than 30 medical causes of edema, which can range from mild dependent edema to swelling from a minor trauma such as an ankle sprain, to specific disease-related edemas to complex swelling associated with concomitant comorbidities. To help accurately identify the type of edema and determine the underlying pathophysiology and associated complications that patients may have, healthcare providers need to be aware of the clinical presentation of edema and the subtle variations that can exist. Early

recognition of the type of edema is essential because not all edemas are managed the same nor benefit from standard interventions. This article will focus on how to clinically assess edema to assist with differential diagnosis and to direct the plan of care by providing the appropriate interventions based on the type of edema presenting.

Initially, it is important to examine the characteristics of the edema and involved tissue structures. The texture of the tissues as well as pitting and rebound qualities of the swelling can assist in differential diagnosis. Edema can present as pitting or nonpitting and can be determined by palpating the involved area or extremity. Pitting edema can be determined by applying firm, yet gentle pressure to the swollen area by depressing the skin with the finger. If the pressing results in an indentation that persists for some time after the release of the pressure, the edema is considered pitting edema. Rebound is the time it takes for the indentation to disappear and can be helpful in determining the severity of the edema. In nonpitting edema, the pressure applied to the skin does not result in an indentation, or the indentation may be difficult to induce and long to rebound. Pitting is indicative of fluid in the tissues, whereas rebound is indicative of fibrotic changes in the tissues.

Pitting edema is graded on a scale of one to four and is determined by the depth of the indentation as well as time to rebound or return to normal. Variations do exist. Table 1 is a culmination of the various pitting scales.

Nonpitting edema is typically indicative of lymphedema due to the fibrotic changes associated with this condition. Fibrosis leads to a hardening of the skin, which renders the tissues unyielding to indentation. In addition to determining the pitting or nonpitting quality of the edema, texture must also be assessed. Tissue texture, in the context of edema, can present on a spectrum or continuum as described in Table 2.

Appreciating the pitting and texture characteristics of the edema will provide valuable insight into the potential

underlying cause(s) of the edema. In addition, the tissue temperature should be assessed in the edematous area and compared to contralateral and/or noninvolved area(s) to ascertain whether the temperature is normal, elevated, or cooler to touch. Any deviation from normal should be further investigated and may warrant referral to a physician. Elevated temperatures could be indicative of active inflammation or infection. Cooler temperatures could be related to underlying circulatory impairments or a local ischemia.

Temperature can be assessed by using one of three methods in clinical practice. The first method is manual assessment

TABLE 1. Edema Pitting Scale

GRADE	DEFINITION
1+	2 mm, barely detectable, pitting rapidly disappears, immediate rebound
2+	4 mm, rebound varies from a few seconds to 10-15 seconds
3+	6 mm, rebound varies from 10-12 seconds to 30 seconds to 1 minute; extremity appears fuller and swollen
4+	8 mm, rebound varies from >20 seconds to 2-5 minutes; extremity is grossly edematous and distorted

by placing the back of the hand on the area in question and comparing it to the contralateral and/or noninvolved area(s) to detect temperature discrepancies. Two other methods involve simple, handheld clinical tools. A thermistor measures surface temperature with a probe that requires surface contact. Thermistors are appropriate on skin that is intact and not denuded or weeping. A radiometer measures surface temperature by infrared radiation. It does not require

TABLE 2. Tissue Texture

Normal	Supple, pliable, elastic
Watery Edema	Palpation manually displaces fluid; palpable pockets of fluid under thin, translucent skin
Soft Pitting Edema	Soft; boggy; feels like dough when palpated or manipulated
Fibrotic	Skin thickened, difficult to pinch or tent; difficult to induce pitting or pitting remains >30 seconds; dense connective tissue; less fibrotic — feels like a tube of toothpaste; more fibrotic — feels firms and leathery; skin may have a cobblestone or lumpy, bumpy appearance
Hard / Noncompressible	Nonpitting; nonpliable; appearance akin to alligator skin or tree bark; typical of advanced or long-standing lymphedema

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TABLE 3. Common Clinical Presentations of Swelling

	Phlebopolymphedema			
	Phlebolymphe ^d ema			
			Lipolymphe ^d ema	
	CVI	Lymphedema	Lipedema	CHF
Etiology	Failure of valves in deep, perforating or superficial veins	Lymph transport failure	Abnormal fat deposition and metabolism (not obesity)	Heart failure
Protein Content	Low	High	Low	Low
Stemmer Sign	Negative	Positive	Negative	Negative
Symmetry	Symmetrical	Asymmetrical	Symmetrical	Symmetrical
Appearance	Edema in gaiter area	Edematous legs with square-shaped toes, deep folds, loss of contours	Large hips, thighs, feet spared; disproportionately small trunk and arms	Buffalo hump on dorsal feet
Texture	Brawny	Lumpy, bumpy or hard, crusty; early pitting, long-standing nonpitting	Loose, lobular	Soft, doughy, deeply pitting
Progression	Distal, below knee	Distal to proximal	Distributed evenly hips and thighs	Distal to proximal
Response to Elevation	Reduces	Persists	Unaffected (not a fluid problem)	Reduces rapidly
Onset	Slow	Slow	Slow; primarily affects females	Rapid
Pain	Achy, worse at end of day	Rarely painful	Painful to palpation	Distention discomfort
Wounds	Weeping, blistering, shallow, uncomfortable, venous ulcers common	Ulcers uncommon, lymphorrea ^h with skin denudement common	Bruise easily; no ulcers or weeping	Distention and weeping, watery edema, blisters
Skin Changes	Hemosiderin staining, strophe blanche, lipodermatosclerosis, brawny or taut skin, varicose veins, dermatitis	Progressive fibrosis, lichenification, hyperkeratosis, papillomatosis	Bruises easily, weak connective tissue, loose and lobular fatty tissue	Cyanosis, jugular distention, shortness of breath
Infection	Rarely cellulitis, can be polymicrobial	Recurrent cellulitis and fungal infections	No cellulitis	No cellulitis
Tests and Measures	ABI, venous duplex ultrasound	ABI, venous duplex ultrasound	N/A unless concurrent CVI or lymphedema	ABI r/o arterial disease and venous duplex ultrasound to r/o DVT
Treatment	Multilayered compression bandaging to reduce, day compression garment to maintain, 30-40 mmHg, wound care	Complete decongestive therapy, diuretics are contraindicated (unless other medical condition warrants use)	Supportive compression garment 20-30 mmHg, diuretics not indicated, may progress to CVI and/or lymphedema; require those interventions	Medical management, diuretics, thigh high compression 20-30 mmHg

NOTE: Created by Robyn Bjork, International Lymphedema and Wound Training Institute. Reprinted with permission. Modified by author.

surface contact and therefore can be used on tissue that is disrupted, impaired, or weeping.

Another clinically relevant test is the Stemmer Sign. A thorough physical examination is considered the gold standard for the diagnosis of lymphedema. A complete history, systems review, inspection, and palpation can assist in determining whether the edema is lymphedema. At present, the only clinical test that has been proven reliable and valid to clinically diagnose lymphedema is the Stemmer Sign. The fibrotic changes associated with lymphedema can lead to a thickening of the skin over the proximal phalanges of the toes or fingers. If the clinician is unable to tent or pinch the skin on the involved extremity, this indicates the presence of lymphedema (positive Stemmer Sign as shown in the photo to the right). A negative finding (where the tissue is still pliable and soft), however, does not rule out the presence of lymphedema. It just may be the lymphedema is still in the early stages before tissue proliferation and fibrosis has occurred.

In clinical practice, the most common forms of edema seen are due to congestive heart failure (CHF), deep vein thrombosis (DVT), chronic venous insufficiency (CVI), and



lymphedema. Patients with lipedema often are thought to have edematous limbs, even though this condition is not fluid-related but rather a pathological deposition of adipose tissue. An excellent resource to learn more about lipedema can be found at <http://www.lipedema-simplified.org/>. It is important to note that lymphedema is often complicated by other conditions leading to combination forms of complex swelling. For example, lymphedema with concomitant CVI is *plebolymphe*ma. Lymphedema in patients with

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lipedema is *lipolymphedema*. Patients can also present with lymphedema, CVI, and lipedema, which is known as *phlebolipolymphedema*. A detailed article on lymphedema will follow in the next issue of *WCHM* magazine. Please refer to Table 3 for a comprehensive overview comparing various edemas.

A thorough patient history should be conducted on any patient presenting with edema of unknown origin. Reviewing the medications the patient is taking is of utmost importance, as many medications can induce or exacerbate swelling.

It is also important to perform noninvasive vascular testing on patients with lower-extremity edema. It is vital to appreciate the health of patients' venous and arterial systems, as compression is often the cornerstone therapy for edema management. To safely and effectively use compression, healthcare providers must understand if a patient's body can tolerate compression.

The Ankle Brachial Index (ABI) is a noninvasive vascular exam to screen for arterial insufficiency. It compares blood-flow pressure in the lower leg to blood-flow pressure in the upper arm. By dividing ankle pressure by brachial pressure, a ratio is determined, which is the ABI value. Variations exist in the literature, but the interpretation shown in Table 4 is widely accepted in clinical practice.

TABLE 4. ABI Values and Interpretations

ABI VALUE	INTRPRETATION	RECOMMENDATION
1.0-1.4	Normal	None
0.9-1.0	Acceptable	None
0.8-0.9	Some Arterial Disease	Treat risk factors
0.5-0.8	Moderate Arterial Disease	Refer to vascular specialist (do not compress)
< 0.5	Severe Arterial Disease	Refer to vascular specialist (do not compress)

Realize, however, that ABI screening may not be accurate in diabetic patients whose vessels are often calcified. Their values tend to be falsely elevated (often > 1.2) and may often be mistaken for normal. Transcutaneous oxygen, tcpO₂ or TCOM, and toe pressures are more reliable vascular screens for patients with diabetes.

Comprehensive skin assessment, including a thorough review for edema, should be part of every patient encounter. Recognizing and appreciating the qualities and characteristics of edema will assist healthcare providers with differential diagnosis to promote appropriate interventions and improve patient outcomes. ■

About the Author

HEATHER HETRICK, PT, PHD, CWS, CLT, CLWT, is an associate professor

in the Physical Therapy Program at Nova Southeastern University in Ft. Lauderdale, Florida. As a physical therapist, her expertise resides in integumentary dysfunction with clinical specialties in wound, burn and lymphedema management. Hettrick has three certifications including: Certified Wound Specialist by the American Board of Wound Management, Certified Lymphedema Therapist by the Academy of Lymphatic Studies, and is dual certified internationally as a Certified Lymphedema and Wound Therapist through the International Lymphedema Wound Training Institute.



Hettrick has diverse work experience in academia and the private sector. Her more recent employment history includes Assistant Professor and Director of Clinical Education at the University of New Mexico; Vice President of Academic Affairs and Education for Gordian Medical, Inc. dba American Medical Technologies; Clinical Assistant Professor in the Department of Physical Therapy at New York University; Adjunct Professor at Drexel University; Program Coordinator for Burn Rehabilitation Research at the William Randolph Hearst Burn Center at NY Presbyterian Hospital and a Master Clinician at the Hospital for Joint Diseases at the Diabetic Foot and Ankle Center.

Hettrick is a past president of the American Board of Wound Management and served on the Executive Committee and Board of the Association for the Advancement of Wound Care. She was recently appointed to the Board of the World Alliance of Wound and Lymphedema Care, and she is helping to establish a lymphatic filariasis morbidity plan for Haiti. She is actively involved in numerous professional organizations, conducts research and publishes, presents and teaches nationally and internationally on integumentary-related issues.

Prevention of New and Recurrent Foot Wounds

Part 1: Introduction and Misconceptions

By Michael B. Strauss, MD; Anna M. Tan, DPM; Lientra Q. Lu

In our previous articles in *Wound Care and Hyperbaric Medicine*, we discussed the evaluation and management of “problem” wounds and the roles of hyperbaric oxygen for them (Figure 1).¹⁻¹² Regardless of immediate results, what really counts is the durability of the healed wound and the restoration of function. This makes the prevention of new and recurrent wounds an essential component of the care provided to the patient. The adage “An ounce of prevention is

worth a pound of cure” is nowhere more true and appropriate than in the healing of the diabetic foot wound.

Often during hospitalization and subsequent skilled nursing care, the patients’ activities are so controlled that healing is achieved. When the patients return to their home environments and the restoration of their prewound activities, however, the wound recurs. This paper is the first in a series of five for *Wound Care and Hyperbaric Medicine* that expounds on the prevention of new and recurrent wounds in the lower extremities. This initial paper discusses myths and misconceptions about healing of the foot wound — especially in the diabetic — and introduces the four preventive measures for new and recurrent wounds, each of which will become a subsequent article.

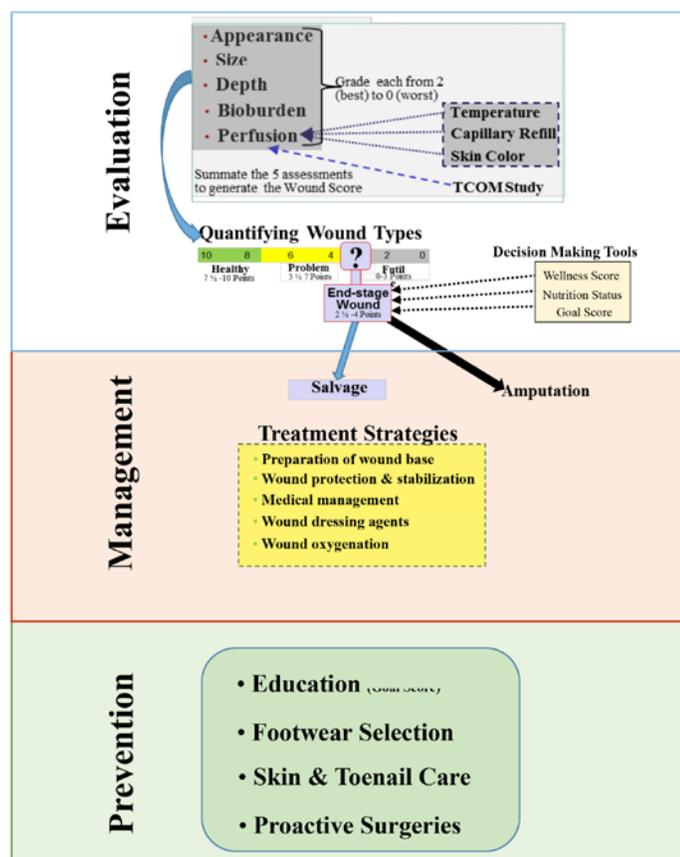
Although this paper and the subsequent papers in this series are primarily directed at diabetic foot wounds (DFWs), it is equally applicable to problem foot as well as other lower-extremity wounds in patients who are not diabetic. This concept was appreciated by Dr. William Wagner, for whom we are indebted because of using his nearly universally accepted diabetic foot ulcer grading system. In his initial paper on the subject, he limited his grading system to diabetic foot ulcers.¹³ Two years later he revised his algorithms to include foot ulcers in nondiabetics as well.¹⁴

Foot wounds in diabetics and patients with peripheral artery disease are common — 10 to 100 times more so than those without these problems. About 1 in 15 diabetics will develop a foot wound sometime during their lifetime.¹⁵ Approximately 85 percent of lower-limb amputations are preceded by nonhealing foot wounds.¹⁶ The majority of foot wounds are easy to manage and resolve completely in their incipient stages. We computed the costs to prevent a foot wound to be 1/50th of the costs to treat one.

Our Observations

Once a foot wound is healed, the majority — that is, more than 90 percent of the cases in our experiences — of new

FIGURE 1. Evaluation, Management and Prevention of Problem Wounds



Legend: Important elements of the evaluation management and prevention of wounds are depicted above. Almost all of the evaluation and prevention items have been published as separate articles (References 1-12). The remainder of this paper will introduce the subject of prevention measures.

foot wounds occur because of one or more confounding factors, which include the following: 1) underlying deformity (bone, bursa and/or cicatrix), 2) deep infection (especially of the deformity-related structures just mentioned) and/or 3) ischemia-hypoxia.¹⁷

These are labeled confounding factors because they are often not appreciated to the extent necessary to prevent new or recurrent wounds, overlooked or outright discarded. This latter consideration is especially important in the noncompliant patient; and/or the patient lost in the exigencies of eponymic health maintenance organizations (HMOs) and insurance payers authorization; and increasingly onerous documentation requirements. Each confounder has defined techniques for diagnosis and specific interventions for management and has been mentioned in the first author's previous publications.^{1,18}

It has been reported that 60 percent of patients with a diabetic foot wound will develop a recurrent wound.^{19,20} Problems such as malnutrition, adequate glycemic control, matrix metalloproteinases, collagen vascular diseases, lymphedema, requirement for steroids/immune-suppressors, anemia, chronic kidney disease, liver failure and immune deficiency disease that interfere with healing are usually adequately managed during hospitalizations, but for the reasons discussed in the preceding paragraph often lack adequate posthospitalization follow-up care (Table 1). Succeeding articles in this series of wound-prevention articles will further elaborate on the four prevention strategies.

Misconceptions Regarding Prevention of New and Recurrent Diabetic Wounds

1. Once a foot wound heals, the wound will inevitably recur after activity is resumed.

TABLE 1. Why Wounds—Especially of the Feet—Arise or Recur

Factor	Effect/Problem	Management	Comment
Confounding Factors, i.e. the “Troublesome Triad”			
Deformity	Pressure sores	Off-load, surgical correction	} One or more of these factors found, in 90% of the patients hospitalized with diabetic foot ulcers we studied ¹⁴
Deep Infection	Inflammation, cell death	Antibiotics, debridement	
Ischemia-hypoxia	Impairment of metabolic processes, cell death	Revascularization, hyperbaric O ₂	
Other Explanations			
Malnutrition	Inhibits generation of substrates; lowers resistance	Supplements, G-tube, hyperalimentation	Quantified with albumin & prealbumin measurements
Diabetes	Hastens atherosclerosis fosters infection,	Insulin, medications, weight loss	Quantified with blood glucoses & HbA1c
Matrix metallo-proteinases	Enzymatic degradation of wound healing precursors	Debridements, hygiene, tetracyclines, Promulgran®	Postulated to stall wound healing; but questionable
Collagen vascular diseases	Vasculitis, primary endothelial dysfunction	Wound care, live with chronic-stable wounds	Distal toe & foot amputations often fail; lead to BKAs
Lymphedema	O ₂ diffusion distance through tissue fluids	Elevation, elastic wraps, diuretics	O ₂ diffuses from capillary to cells through tissue fluids
Steroids / Immunosuppressors	Hinders healing & infection defenses	Vitamin C, antibiotics,	Interferes / halts inflammatory responses
Anemia	Decreased O ₂ delivery / availability to tissues	Iron supplements, erythropoietin, transfusion	May benefit perfusion; less cell mass in sluggish flow
Chronic kidney disease	Unhealthy environment for metabolism	Medications, dialysis, renal transplant	Edema & anemia; immuno-suppressors (transplants)
Liver disease	Hampers generation of proteins; eliminates wastes	Diet, medications, transplants	As above for chronic for chronic kidney disease
Immune deficiencies	Uncontrolled infections; life / limb threatening sepsis	Antibiotics, transfusions, vasopressors	Associated with adrenal shutdown, splenectomy

Fact #1: The reasons for this misconception are the high recurrence rates observed when activity is resumed after wounds heal with hospital management, rest and offloading and as is especially observed with malperforans ulcers after total contact casting.²¹ Without attention to the underlying deformities and the other wound confounders, new and recurrent foot wounds are likely to occur.

Fact #2: With attention to managing deformities, muscle imbalances, wound hypoxia and infection — and initiation of prevention strategies (as will be discussed in subsequent articles), new or recurrent wounds can be prevented.

Fact #3: Furthermore, once a wound is healed, the metabolic demands to maintain the wound site in a healthy condition are but a small fraction of what was required for healing and infection control. As much as a 20-fold increase in perfusion and metabolic activity compared to the resting, healed state may be required.²² The increased perfusion arises from redistributing the human body's limited blood volume (about 5 liters) from noncritical — in terms of perfusion — to the wound site in response to sympathetic nervous system activity and cytokines.

Even in the presence of peripheral arterial disease, the approximately 1/20th reduction in perfusion and metabolic activity is usually sufficient to meet the minimal metabolic demands of the noncritical tissues. Consequently, with the prevention strategies, healed wounds, especially of the feet and ankles, remain healed regardless of marginal perfusion to these areas.

In our region, and it is probably no different in other regions of the USA, once the HMO patient is discharged from an emergency hospitalization because of a foot wound and the wound is nearly healed and/or markedly improved, follow-up care is often delayed or the patient is returned to one of the HMOs contracted providers who lacks the expertise in optimizing wound care. The consequences often are worsening of the wound after discharge from the hospital.

If authorizations for follow-up care with wound care specialists are eventually provided, often the delay in follow-up care reveals absence of appropriate wound care in the interim, worsening of the wound, inappropriate use of protective footwear or the need for rehospitalization with additional surgeries. Other consequences include failure to recognize new or continuing confounding factors that need to be addressed to prevent new or recurrent wounds.

Fact #4: Patient compliance is a crucial factor in avoiding new and recurrent foot wounds. Although much is written about the importance of compliance, little information is available on how to quantify it. Our next article for *Wound Care and Hyperbaric Medicine*, which will focus on patient education, will offer much information on measuring patient compliance and how to use this information for managing patients.

2. Preserving the foot has little functional significance in the marginal ambulatory.

Fact #1: A lower-limb amputation in the minimal ambulator who had been independent with activities of daily living may increase the energy demands for walking with a prosthesis to the point that the patient requires assisted living.

Fact #2: Energy demands for walking with a prosthesis, as measured by oxygen consumption, essentially double with a below-knee amputation and more than triple with an above-knee amputation.^{23,24}

Fact #3: Additionally, weakness and arthritic changes in the upper extremities may make donning and removing the prosthesis difficult, if not impossible. With such comorbidities, the use of crutches or front-wheel walkers may be markedly restricted.

Fact #4: Rehabilitation and confidence in using a prosthesis may take a year or more to maximize strength, balance and endurance. In patients with cardiac and pulmonary comorbidities, oxygen-consumption demands for walking with a prosthesis may exceed the patients' maximal aerobic capabilities.

Fact #5: Although an amputation may appear to be immediately cost-effective by expediently moving a patient from the acute hospital setting to a lower level of care, the total costs for managing the wound may be overall cost beneficial. For example, expenses for surgery, prostheses and rehabilitation exceed \$50,000 during the initial 18 months after an amputation.^{25,26}

Fact #6: Challenges for fitting the prosthesis are anticipated in the patient with cardiac and renal impairments where marked variations in stump volume occur. This can be further compounded by stump tissue atrophy, knee joint contractures, short stump lengths and lack of soft tissue padding over the end of the tibia.

3. Once a leg is amputated, amputation of the other limb will soon follow.

Fact #1: If the lower limb amputation is due to severe, diffuse, bilateral peripheral artery disease that cannot be

revascularized, this statement may be true. With wound-prevention measures (which will be subsequently presented in future editions of *Wound Care and Hyperbaric Medicine*), however, contralateral lower-limb amputations can usually be avoided. This observation is supported by our previous discussion of the great differences in perfusion and metabolic activity to heal a wound in contrast to the steady state, nonwound, noninfection state.

Fact #2: If the lower-limb amputation is due to wounds associated with uncontrollable deformities such as Charcot neuroarthropathy or distal leg-ankle fracture nonunions, subsequent amputation of the other extremity is highly unlikely in the absence of new problems in the remaining extremity.

Fact #3: In the situations in which a lower-limb amputation becomes necessary, be it unilateral or for the opposite limb in the patient who is already an amputee, one or more criteria for amputation are invariable present. Undoubtedly, one of the most important is uncontrollable pain. Other reasons for the lower-limb amputation include gangrene of the foot, deformity severe enough that functional use of the extremity is not possible, nonhealing wounds secondary to vasculitis in patients with collagen vascular diseases and a subset of diabetic patients with foot wounds infected with methicillin-resistant *Staphylococcus aureus*. In this latter situation we have observed that as long as the patient remains on antibiotics, the wound appears healed, but once the antibiotics are discontinued — even after months of therapy — the wound recurs, usually moving proximally up the foot and leg. It appears this subset of diabetic patients is deficient in host factors needed to eradicate residual occultly infected tissues at the original wound site.

4. Care of the limb-threatening wound in the diabetic is different than in the patient without diabetes.

Fact #1: The severity of the wound is the overriding consideration for making decisions about management of the wound. Our Wound Score, which integrates information from the four most commonly used wound-grading systems, objectively grades wounds in a user-friendly format that determines the severity of the wound and provides the basis for management (Table 2). If the wound is in the “healthy” or “problem” wound categories, management using the strategies of 1) optimal wound base management, 2) appropriate protection and stabilization, 3) medical management including antibiotics, 4) suitable wound dressing agents and 5) adequate perfusion-oxygenation from our previous reviews resulted in more than a 90 percent positive predictive value for wound healing.^{18,27,28} It is

irrelevant whether the patient is diabetic or not with use of our wound grading and management strategies.

Fact #2: If the wound is serious enough that a lower-limb amputation becomes an option for management, then additional patient information — such as their health status and their goals — is required. Like the Wound Score, these scores are each based on five assessments graded from 2 (best) to 0 (worst) and have been previously described in *Wound Care and Hyperbaric Medicine* (Tables 3 and 4).^{1,18} These scores help to justify the decision whether to salvage the foot or recommend a lower-limb amputation and apply equally well to the diabetic as well as the nondiabetic.

Fact #3: The majority of patients with venous stasis disease ulcers are not diabetic. Again, management that utilizes the five strategies as described previously is employed. With venous stasis ulcers, the protection and stabilization strategy with compression dressings is a key element. Management of the wound base is also essential. When the ulcers are chronic and seemingly refractory to treatment, they require operating-room debridement to remove an impervious

layer of cicatrix between the ulcer base and the underlying healthy vascularized tissue that may extend through the subcutaneous tissue to the underlying fascia (Figure 2). Once a healthy granulating wound base develops, split-thickness skin grafting is almost uniformly successful.

5. Neuropathy is the primary reason foot wounds fail to heal.

Fact #1: Neuropathy with absence of sensation is an indirect cause of wounds. It, in itself, does not cause wounds. Foot wounds are caused by stresses such as shear, pressure concentrations and/or trauma that exceed the skin's ability to mitigate the aggravating factors. Contributing causes as previously mentioned include underlying deformities and ischemia-hypoxia.

The main concern of neuropathy-associated wounds is that it delays diagnosis. In the absence of pain, the wound may go unnoticed or be totally disregarded until others complain of odor, ascending infection develops or the patient becomes septic.

TABLE 2. The User-Friendly 0 (Worst) to 10 (Best) Wound Score to Objectively Categorize Wounds

Assessment	2 points	1 point	0 points
Appearance Of the wound	Red	White/Yellow	Black
Size	< thumb print	Thumb print to fist	> Fist
Depth	Skin coverage	SC* Muscle/tendon	Bone/ joint
Infection/Bioburden	Colonized	Osteomyelitis, maceration or cellulitis	Sepsis
Perfusion	Palpable pulses	Doppler pulses	Imperceptible Pulses

Summate the 5 assessments

Grade each from 2 (best) to 0 (worst) with objective parameters

Wound Categories:
Healthy = 7 ½ - 10 Points
Problem = 3 ½ - 7 points
Futile = 0-3 points

SC* = Subcutaneous tissue

TABLE 3. Wellness Score Quantity Patients' Health Status

Assessment	2- Points	1 Point	0-Points
ADLs Activities of daily living	Full	Some	None
Ambulation	Community	Household	None
Comorbidities Except neurological	Normal	Impaired	Decompensated
Tobacco/Steroid Use Which gives lower score	None	Past	Current
Neurological Deficits	None	Some	Severe

Summate the Grades of the 5 assessments
Scores of $\geq 4 \frac{1}{2}$ support the decision for limb salvage



TABLE 4. Goal Score Quantifies Patients' Desire to Avoid Lower Limb Amputation

Assessment	Full	Some	None (0 Points)
Comprehension	2 Points	1 Point	0 Points
Motivation			
Compliance			
Insight			

Summate the Grades of the 5 assessments
Scores of $\geq 4 \frac{1}{2}$ support the decision for limb salvage

Other neuropathy contributing factors to wounds include 1) muscle imbalances (such as those leading to clawed toes) from motor neuropathy (these concentrate pressure stresses at the deformity sites); 2) excessive shear stresses from impaired proprioception (wounds occur with ambulation and shoe wear); 3) increased vulnerability of the skin to breakdown due to autonomic nervous system dysfunction (this leads to skin dryness, atrophy, loss of elasticity and wasting away of underlying soft tissue padding).

Fact #2: Neuropathy is not the reason a wound fails to heal. Paradoxically, neuropathy may facilitate wound healing by increasing blood flow through loss of autonomic nerve function controlling vasoconstriction. This leads to hyperperfusion as is so often in association with Charcot neuroarthropathy. Another reason there may be increased blood flow is that the blood vessels are calcified (atherosclerotic) and do not constrict in response to sympathetic nervous system stimulation.

Fact #3: Diabetic foot wounds in the presence of sensory neuropathy are typically the easiest to manage because dressing changes and wound debridements can be optimized since they do not lead to discomfort for the patient.

Fact #4: Even more convincing evidence to dispel the misconception that neuropathy is a direct cause of foot wounds is the observation that once the wounds are healed in patients with sensory neuropathy, recurrences are the exception in the well-motivated patient. This is attributed to compliance of the patient for the prevention measures (e.g., education, foot skin and toenail care, protective footwear and proactive surgeries), which each will be discussed in subsequent articles in *Wound Care and Hyperbaric Medicine*.

6. It is difficult to predict which patients are prone to wound development in their feet.

Fact #1: Consensus workshops uniformly agree that five risk factors — especially in diabetics — need to be recognized and appropriately addressed to prevent new and recurrent wounds in patients prone to develop ulcerations in their feet.²⁹⁻³² The risk factors are: 1) deformity, 2) peripheral vascular disease, 3) history of previous wound, 4) previous amputation and 5) neuropathy

Fact #2: Other risk factors such as obesity, diabetes mellitus, malnutrition, smoking, myopathies, loss of proprioception (as in Charcot neuroarthropathy), collagen vascular diseases, inappropriate activities, improper footwear and compliance can also be contributing factors to new and recurrent foot wounds. Many of these can be recognized with an appropriate evaluation of the patient; several are remedial and manageable with appropriate care and counseling

such as diabetes, malnutrition and smoking. Smoking and compliance will be discussed in subsequent articles in this series. Myopathies and loss of proprioception are factors rarely mentioned as causes of foot wounds. Because of abnormal gait mechanics leading to disproportionate shear stresses with ambulation, foot wounds are prone to develop in patients with these problems.

7. The patients' physicians and other caregivers have little to offer in terms of preventing new or recurrent foot wounds.

Fact #1: It is crucial that appropriate medical follow-up be done to prevent new and recurrent foot wounds. Four items are essential for the follow-up evaluations (Figure 3): They include 1) patient education, 2) foot skin and toenail care, 3) proper protective footwear and 4) proactive surgeries. Each needs to be an element of follow-up evaluation and will be the subject for future articles in this series.

Fact #2: The more risk factors for foot wounds (i.e., deformity, previous wound, previous infection, peripheral artery disease and neuropathy) that are present in a patient, the more important follow-up evaluations are.

Fact #3: Follow-up intervals need to be tailored to the patient's needs. For some patients, follow-ups may need to be done only once or twice a year, whereas in others they may need to be done at biweekly intervals to prevent new or recurrent foot wounds. As expected, there is an inverse relationship between the recommended frequency of patient follow-ups and patient compliance.

Discussion

Preventive medicine is assuming increasingly important roles in patient management. We previously published

FIGURE 3. The Four Strategies to Prevent New and Recurrent Foot Wounds



Legend: Prevention strategies are fundamental for preventing new and recurrent foot wounds. Each complements the others. Each evaluation and management of a patient with one or more of the risk factors predictive for foot wounds (namely deformity, neuropathy, peripheral artery disease, previous foot wound, or partial foot/toe amputation) should always address all of the four strategies. The prevention of a wound is highly cost-effective versus dollars and dollars to cure it. (i.e. "An ounce of prevention is worth a dollar of cure.") Each strategy will be separately discussed in our succeeding *Wound Care & Hyperbaric Medicine* articles.

articles on risks factors, indirect and direct causes of diabetic foot wounds and the four-pronged approach to preventing diabetic foot ulcers.^{33,34} This paper expands on these topics and lays the foundation for separate articles on each of the preventions strategies.

Periodic examinations with care providers is necessary in integrating the prevention strategies. Whereas annual physical examinations rely largely on laboratory data to make management recommendations, the prevention of new and recurrent foot wounds is dependent on examination of the patient's feet. As a further contrast, much of the preventive care offered to the patient from laboratory data is achieved through medications. For prevention of foot problems, the care is largely hands-on, such as skin and toenail care, selection of protective footwear and proactive surgeries. There are mutual concerns, of course, such as smoking history and obesity that are fundamental components of an examination and have ramifications for the entire body as well as the feet. Consequently, there is no better portal for prevention of new and recurrent foot wounds than examination of the feet. Awareness of the five major risk factors (deformity, previous wound, previous amputation, peripheral artery disease and neuropathy) for developing foot wounds is the examination starting point for any patient to prevent new and recurrent foot wounds.

In the introduction, the magnitude of foot wound problems was mentioned. Examination of cost considerations give added justification for being proactive in preventing new and recurrent foot wounds. If a foot wound evolves to a necrotizing soft-tissue infection with underlying deep infection and/or osteomyelitis, hospitalization is required to save life and limb. The hospitalization for a serious diabetic foot wound typically exceeds \$30,000. One or two surgeries at an estimated \$10,000 each and 10 days of hospital care at \$2,000 a day is an example of how quickly hospital care charges amass. Conversely, a lower-limb amputation is not necessarily a cost-beneficial approach to the foot wound problem. More than 15 years ago the estimated cost of a below-knee amputation with 18 months of follow-up including prosthesis costs and rehabilitation was more than \$50,000.^{25,26} Today the total expenditures may be double this amount. For example, the charges for a below-knee prosthesis is about \$15,000, and the computerized knee component alone for an above-knee prosthesis is \$50,000.

The costs of hospital care and the prostheses do not represent the complete long-term costs and emotional toil a lower-limb amputation accrues. These "costs" can arise from inability to return to gainful employment and/or continue in an independent living status. Each has its own detriment to

a patient's emotional status and self-worth. Assisted living or skilled nursing costs \$3,000 to \$5,000 or more a month. The maintenance of independence in a marginal ambulator with their limbs intact, but managing to continue in an independent living status is far superior to the above options.

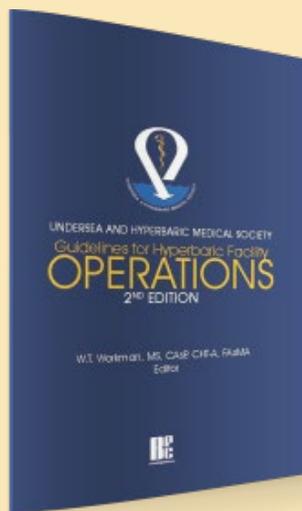
The donning and removing of a prosthesis and walking with the additional weight of the prosthesis plus loss of lower-limb proprioception may confine the patient to being able to use only the prosthesis to do transfers. Such scenarios can be emotionally devastating for the patient and extremely challenging for the patient's family. Hence, the prevention of new and recurrent foot wounds extends beyond cost considerations alone.

A final consideration: Is it fair to discriminate levels of care between diabetics and nondiabetics (as mentioned in the fourth misconception discussed previously) with actual or impending foot wounds? Presently, there are major discrepancies between what care options are available through Medicare/CMS provisions and other third-party insurance providers for diabetics and nondiabetics. Two obvious examples include the provision of protective footwear for diabetics in accordance with the Diabetic Footwear Bill and the use of hyperbaric oxygen for specific diabetic foot wound problems (e.g., diabetic foot ulcers not improved with surgery and antibiotics for 30 days or more that involve deep abscess and/or osteomyelitis).

As noted before, Wagner expanded his diabetic foot grading system two years after his original publication to include nondiabetic foot wounds.¹⁴ Wagner differentiated only between the diabetic and nondiabetic using ankle-brachial indexes greater than 0.45 in the diabetic versus 0.35 in the nondiabetic for making decisions about limb salvage. Other than that, his algorithms for management were identical. Thus, we advocate equal diligence in preventing new or recurrent foot wounds regardless of whether or not the patient is diabetic.

Conclusions

The prevention of new and recurrent foot wounds is more than a cost-benefit consideration. Of all the preventive measures (not withstanding immunizations) used in medicine and surgery, the prevention of new and recurrent foot wounds is one of the most predicable and cost beneficial. Many misconceptions exist about foot wounds and their prevention. This article presents seven examples and provides facts and answers to dispel the misconceptions. Additional articles to follow in this series will elaborate on the specific strategies to prevent new and recurrent foot wounds and will include the subjects of patient education,



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skin and toenail care, protective footwear and proactive surgery. Although much attention has been given to this subject in the diabetic, we feel that all foot wounds — especially in the nondiabetic with peripheral artery disease — deserve the same attention as for the diabetic. ■

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About the Authors

MICHAEL STRAUSS, M.D., an orthopaedic surgeon, is the retired medical director of the Hyperbaric Medicine Program at Long Beach Memorial Medical Center in Long Beach, California. He continues to be clinically active in the program and focuses his orthopaedic surgical practice on evaluation, management and prevention of challenging wounds. Dr. Strauss is a clinical professor of orthopaedic surgery at the University of California, Irvine, and the orthopaedic consultant for the Prevention-Amputation Veterans Everywhere (PAVE) Problem Wound Clinic at the VA Medical Center, Long Beach, California. He is well known to the readers of *WCHM* from his multiple articles related to wounds and diving medicine published in previous editions of the journal. In addition, he has authored two highly acclaimed texts, *Diving Science* and *MasterMinding Wounds*.



LIENTRA LU is a research assistant at the VA Medical Center in Long Beach, California, under the guidance of Dr. Ian Gordon, a vascular surgeon, and Dr. Michael Strauss, the first author of this paper. She received a bachelor of science degree in chemical biology at the University of California, Berkeley, in 2015 and subsequently has taken medically related courses at the University of California, Los Angeles. Miss Lu is helping with diabetic foot and venous leg ulcer studies in addition to research on abdominal aortic aneurysms at the VA Medical Center while also serving as an assistant in patient care at the PAVE Clinic there. Her other interest is disaster preparedness, where she works with the American Red Cross.



ANNA M. TAN, DPM, is a second-year podiatric medicine and surgery resident at Long Beach Memorial Medical Center. She graduated cum laude from the University of Southern California in 2006 and received the Dean's Award for her undergraduate research on netrin-1, a protein involved in axonal guidance. Subsequently, she attended the California School of Podiatric Medicine at Samuel Merritt University in Oakland, California, receiving her doctor of podiatric medicine degree in 2014. Dr. Tan has special interests in surgical management of problem wounds, limb salvage and unifying the classification of mental disorders and personalities. In her spare time, she enjoys Bikram yoga, cooking and traveling.



Atypical Wounds Q&A

By Jayesh B. Shah, MD, CWSP, UHM

QUESTIONS

1. A patient has a diabetic wound that probes to the bone. ESR -103, CRP - 6. An X-ray suggests changes of periosteal elevation and sclerosis suggestive of osteomyelitis. Which of the following would you do next?

- A. radionuclide bone scan
- B. MRI of right foot
- C. start six weeks of IV antibiotics
- D. obtain a bone biopsy for culture prior to antibiotics
- E. culture the drainage prior to antibiotics

2. A 32-year-old male who works as a field worker in the U.S. and resides in Mexico developed small sinus lesions on the left foot three years ago and now has developed multiple recurrent sinuses on that foot. The patient took multiple courses of antibiotics from Mexico.



He presents to the wound clinic with a completely deformed left foot, swollen with multiple sinus lesions with purulent drainage as seen in the photo. Gram stain shows Gram-positive nonspore-forming anaerobic bacilli with sulphur granules. What is the diagnosis?

- A. actinomycosis
 - B. skin abscess because of drug use
 - C. Charcot foot arthropathy
 - D. sporotrichosis
3. A 32-year-old woman is evaluated for a five-day history of nodules over her lower extremities, which happened after she visited a local spa that used whirlpool foot baths. She reports shaving her legs with a razor before her visit. Tissue culture grows a mycobacterial species

within five days. Which of the following is the most likely cause of the infection?

- A. *Mycobacterium marinum*
- B. *Mycobacterium ulcerans*
- C. *Mycobacterium fortuitum*
- D. *Mycobacterium avium* complex

4. A 70-year-old female with a history of Type 2 DM, ESRD, on hemodialysis, presents with an extremely painful wound on her leg, which started as small darkened area that progressively increased in size with worsening pain. What is the diagnosis?

- A. spider bite
- B. pyoderma gangrenosum
- C. calciphylaxis
- D. calcinosis cutis

5. A 65-year-old HIV-positive patient with recurrent hidradenitis suppurativa lesions on both buttocks now presents with fungating growth as seen on the picture. What is the diagnosis?



- A. basal cell cancer
- B. squamous cell cancer
- C. Kaposi sarcoma
- D. fungal infection

ANSWERS

1. D — The patient with osteomyelitis should get a bone biopsy and culture before being subjected to six weeks of IV antibiotics. There was a huge discrepancy in the patient with open wounds with osteo between swab culture and bone culture.² Also probe to bone sign is more specific for diagnosis of osteomyelitis than any other radiological study.¹ In patients with clinical sign of probe to bone and X-ray positive for osteo, the next best test is to do a bone biopsy.
2. A — This patient has actinomycosis. *Actinomyces israelii* is Gram-positive, nonspore-forming anaerobic bacilli.³ The treatment is surgical excision and antibiotics for six months. Ampicillin is the drug of choice.
3. C — *Mycobacterium fortuitum* furunculosis is a well-described skin infection in patients who obtain pedicures at nail salons that use contaminated whirlpool footbaths. *M. fortuitum*, *M. chelonae* or *M. abscessus* are rapid-growing mycobacteria, and culture grows in less than seven days. All other mycobacteria listed in question are slow-growing organisms.
4. C — The patient with long-standing ESRD who is on hemodialysis develops dysfunctional calcium and phosphorus balance leading to calciphylaxis. A patient with calciphylaxis has a high risk of mortality, with 60 to 80 percent of patients dying within six months of diagnosis. The patient usually develops sudden superficial skin necrosis followed by painful, pruritic, violaceous skin discoloration in livedo reticularis pattern with black eschar. Biopsy shows metastatic calcification within the lumen of arterial vessels.⁵
5. B — This patient with hidradenitis suppurativa now has fungating growth, patient's ulcers has transformed into malignancy, and most common malignant transformation in chronic ulcer is squamous cell cancer.⁶ ■

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About the Author

JAYESH B. SHAH, MD, CWSP, UHM, is president of South Texas Wound Associates, PA, and of TIMEO2 Healing Concepts, LLC, both in San Antonio, Texas. His degrees include an MBBS (bachelor of medicine and surgery) from Maharaja Sayajirao University in Baroda, India, and an MD in internal medicine from St. Luke's Roosevelt Hospital, Columbia University, New York. He is board certified in internal medicine and in undersea and hyperbaric medicine and certified in wound management and in hyperbaric medicine.



Shah has more than 18 years' experience in wound care and hyperbaric medicine practice and more than 12 years' experience as program director for continuing medical education courses. He currently serves as the medical director for the Northeast Baptist Wound Healing Center. An adjunct professor in the Department of Family and Community Medicine at the University of Texas Health Science Center, Shah is coeditor of the *Wound Care Certification Study Guide, First Edition* (published by Best Publishing Company). He created the WoundDoctor app for smartphones and authored 19 chapters on various wound topics in four books in addition to more than 30 scientific articles on wound care and hyperbaric medicine.

Continuing Education in Wound Care: It's A Big Deal

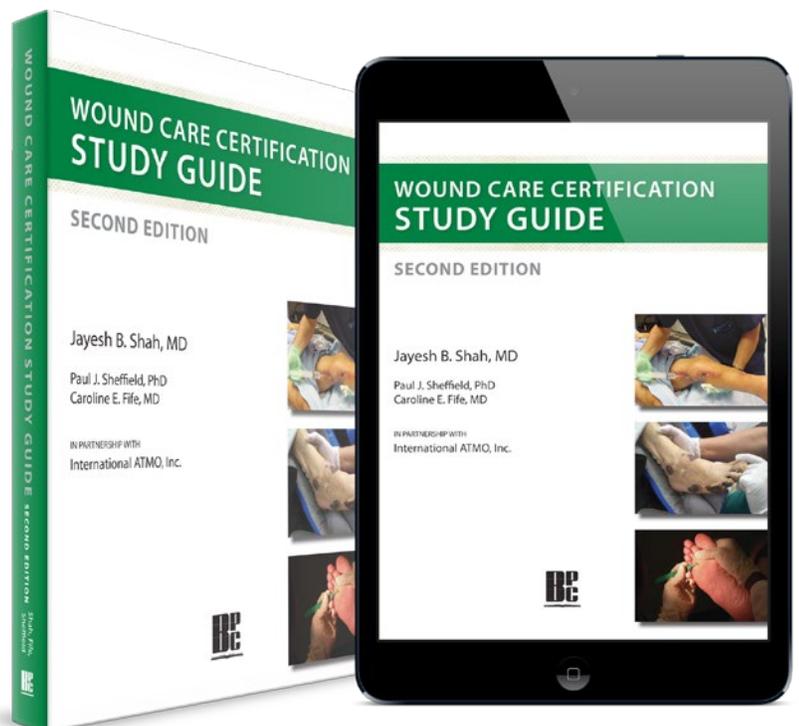
By Terry Treadwell, MD, FACS, Medical Director,
Institute for Advanced Wound Care, Montgomery, Alabama

Almost everyone who provides health care has some basic “smarts” or they would not have made it through school to become a practitioner. I would be the first to agree that professional schools and postgraduate training programs are difficult, requiring significant dedication to finish. Once one walks out of that room or auditorium with the final certification and license to practice, there is a great feeling of finally being “done.” Unfortunately, that time in our lives only marks the beginning.

Despite all we have learned, it has been shown by many people through the years that at least half of what we learn in school and postgraduate programs will be useless or proved wrong by the time we finish our careers.¹ I can truthfully say that fully half of the technologies and products we use daily in our wound treatment center did not exist 10 years ago. For these reasons, continuing to look for the best approach to treating wounds is critical.

A recently published book, *Wound Care Certification Study Guide, 2nd Edition*, edited by Drs. JB Shah, PJ Sheffield and CE Fife is a resource I would recommend to help with your search for information.² As one can tell by the title, it has been developed to assist anyone planning to take any of the wound certification examinations, but the amount and scope of the information in the book can serve as a textbook for anyone who wants an updated source of wound care information.

The book contains 33 chapters covering all the basic and advanced topics likely to be faced by someone treating patients with any type of wound. Since the first edition was published in 2011, all of the chapters have been updated, and a chapter introducing the use of hyperbaric oxygen has been added. Each chapter has numerous references to the topics if further reading is desired. There are illustrative



questions included at the end of each chapter to help the reader evaluate his/her comprehension of the subject before moving to the next.

Each chapter contains numerous photos that detail the issues being discussed. The underlying pathophysiology of each condition is included, leading the reader to a better understanding of the problem and why certain treatments are selected. Chapter 7: Patient Preparation and Education presents a unique perspective for helping the patient and family understand what to expect from having a chronic wound and the proposed treatments, including pain management, dressing changes and possible treatments outside of the wound center.

Because of the importance of bacteria in chronic wounds, the editors have included three chapters dealing with the

topic. The first is an overview of microbiology to refresh one's memory about the basics of the subject. This is followed by a chapter on infection control and subsequently, an overview of wound infections. If a biopsy culture is to be obtained and anesthesia is required, it is important to remember many topical anesthetics have antimicrobial actions of their own, which should preclude their use when taking specimens for culture. Berg and associates reported that any topical anesthetic, especially Emla cream, has a rapid and powerful antibacterial effect within 30 minutes of applying it to the wound surface.³ Use of this anesthetic can lead to unreliable tissue culture results. For this reason, the only anesthetic recommended for tissue biopsies done for culture is 1 percent preservative-free lidocaine.³

The chapter on lymphedema reminds the reader of the importance of this condition. Lymphedema is often overlooked when one deals with chronic wounds, but it should be remembered that the periwound lymphatics are always damaged, resulting in much of the periwound swelling being due to high-protein lymphedema fluid and not just low-protein edema fluid. This should remind all of the importance of compression bandaging and other forms of compression therapy in the treatment of any patient with swelling of the periwound tissues. It should also be realized that compression therapy can be successfully and safely used in the treatment of patients with an ankle/brachial index (ABI) of less than 0.8. Many feel this should not be done, but the safety and efficacy of careful compression in this group of patients has been well documented.^{4,5,6} If these patients are denied compression therapy, they will not improve or get well.

The chapter on burns presents a brief overview of the types of burns that may be encountered. Most severe burns are the purview of burn surgeons treating these patients in burn units. The topical treatment of second-degree burns is mentioned, but care should be taken when using any silver dressing in the treatment of these wounds. The toxicity of silver-containing products is well-recognized and is known to delay the healing of these burns. It must be noted that silver-containing topicals and dressings can be especially hazardous in infants and children.⁷⁻¹²

The chapter on dermatological review and unusual wounds should be of importance to all.

Treating patients in a wound-center setting does not rule out the possibility of being asked to see any patient with a skin defect or problem. Many times differentiating one skin defect from another can be difficult and must be done accurately and timely to avoid treatment that can be inappropriate, wasting both resources and time. The

number of patients seen with "unusual wounds" may depend on where one lives and one's diagnostic abilities. For example, in the South, brown recluse spider bites are not uncommon, whereas in the Northeast they are rarely seen. Many people say they never see pyoderma gangrenosum in their wound centers. I did not either until I was able to make the diagnosis. Do malignancies occur in or masquerade

About the Author

DR. TERRY TREADWELL received his medical education at the University of Texas Southwestern Medical School in Dallas, Texas. He served his general and vascular surgical residencies at Scott and White Medical Center in Temple, Texas, and practiced vascular and general surgery in Montgomery, Alabama. In October 1998, Treadwell founded the Institute for Advanced Wound Care at Jackson Hospital in Montgomery. He served as medical director of the center and treated wound patients on a full-time basis, providing the best possible care to this seemingly forgotten group of patients. In February 2006, the Institute for Advanced Wound Care moved to Baptist Medical Center in Montgomery. Treadwell serves as the medical director of the institute. He has been involved with numerous educational and research initiatives and directs wound-care educational programs at his wound center to help educate physicians and other medical personnel in the current therapy of acute and chronic wounds.



Treadwell has shared his experience in the treatment of chronic wounds through lectures, presentations and publications. Wound-care practitioners from around the world have attended preceptorships at the Institute for Advanced Wound Care. He has assisted in the establishment of wound treatment centers in Ghana, Africa, and Port-au-Prince, Haiti. He is the clinical editor of *Wounds* magazine and is a member of the World Association of Medical Editors. He is a member of the Wound Healing Society and the Association for the Advancement of Wound Care. He has served two terms as the Physician Member of the Association for the Advancement of Wound Care board of directors, and has served as the president and past-president of the AAWC.

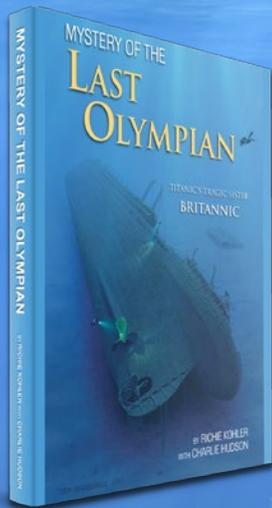
He is serving on the World Health Organization Committee, the World Alliance for Wound and Lymphedema Care, to develop wound education and treatment guidelines for treatment of acute and chronic wounds in underdeveloped countries of the world. He is the current vice president of the World Alliance for Wound and Lymphedema Care board of directors and the president-elect. He was recently named Associate Professor of Wound Care at the University of Medicine and Pharmacy, Port-au-Prince, Haiti.

as ulcers? It depends on how hard one looks for them. I encourage wound biopsy if you are unsure of the diagnosis or if the wound has failed to respond to what you consider good therapy after four weeks of treatment.¹³⁻¹⁴

All in all, this is a good book for learning about and updating one's knowledge of wounds, whether in preparation for a certifying exam or for continuing learning. Many consider continuing education a waste of time or something for others. If we do not continue to learn throughout our professional careers, we will soon find ourselves behind everyone else. We certainly want to provide the best care possible for our patients. By increasing and updating our knowledge, we can provide better care for our patients. ■

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**MYSTERY OF THE LAST OLYMPIAN: *Titanic's Tragic Sister Britannic***

by Richie Kohler with Charlie Hudson

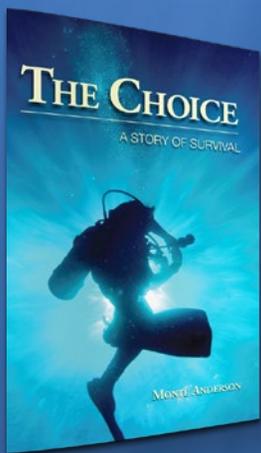
For 100 years the mystery surrounding the sinking of *Titanic's* tragic sister *Britannic* was a riddle waiting to be solved. This book gives you a firsthand account as Richie Kohler takes readers on the intriguing journey from the rise of the magnificent Olympians to the ship's fateful sinking in 1916. He then moves forward in time through multiple expeditions, beginning with the great Jacques Cousteau, who located the wreck of the ocean liner in 1975. Each successive team of divers who risked their lives uncovered new clues, but it was not until 2009 that Kohler and his dive partner definitively pinpointed the secret that had eluded everyone before then.

Join Kohler, host of the History Channel's *Deep Sea Detectives* and featured in the bestselling book *Shadow Divers*, as he solves the *Mystery of the Last Olympian*.



"In Richie Kohler's new book, the same drive for adventure that captivated my father comes alive as Kohler rediscovers the mysteries surrounding the ship's fateful demise. Their journey spans across past and present, honoring the legacy of an unsinkable ship and the determination of those who risked, or even lost, their lives in the search to uncover its secrets."

~ Jean-Michel Cousteau, explorer, environmentalist, educator, and producer

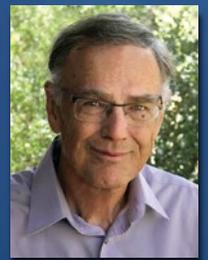
**THE CHOICE: *A Story of Survival***

by Monte Anderson

As three friends drove across the Navajo Reservation in northern Arizona after backcountry skiing in Colorado, they talked about their lives. Then one said, "I really shouldn't be alive today."

David Scalia's astounding story occurred in 1982, when a scuba equipment failure caused a devastating accident, but he had a scrapbook documenting everything that happened. He suffered incalculable damage to his body for more than 12 grueling hours. Days later, he was given a profound choice — to live or to die. Almost unbelievable, this is his true story — and it involves some friends and colleagues you may know, including Dr. Gregory Adkisson, Dr. Tom Neuman, and Dr. Paul Phillips.

About the Author: Monte Anderson completed a medical residency at Creighton University and continued his studies with subspecialty training in gastroenterology and hepatology as an army officer at Fort Sam Houston in San Antonio, Texas. After his discharge from the military, most of his career was happily devoted to the Mayo Clinic in Arizona. Feeling that true tales tend to be more compelling than fiction, he has always preferred reading nonfiction, especially since something is always learned in the process. *The Choice: A Story of Survival*, his first effort outside of scientific writing, is nonfiction.



"Dr. Monte Anderson makes his debut in nonmedical writing with *The Choice: A Story of Survival* and does so with a splash. The nonfiction book relates the fascinating story of his friend's 1982 diving accident near a remote island in Mexico. Dr. Anderson's recounting of the details reflects his tremendous investigative ability, as well as the diver's will to survive."

~ Neil B. Hampson, MD, author of *Cherry Red*

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