# Wound Care & Hyperbaric Medicine

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Wound Care & Hyperbaric Medicine

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John S. Peters, FACHE
Publisher, *Wound Care & Hyperbaric Medicine Magazine*

# Note from the Editor

Best Publishing Company is proud to announce that owner John S. Peters, FACHE has been offered and has accepted the position as executive director of the Undersea and Hyperbaric Medical Society. Mr. Peters has been involved in healthcare for over twenty years and started his own company, Wound Care Education Partners, in 2008. His success in business and education, along with his strong leadership qualities, made him one of the top contenders in a pool of well-respected candidates. While he may be taking a step back from his responsibilities here at *WCHM* to pursue this new endeavor, we are excited to see him in this new role and wish him great success!

In *WCHM* news, Volume 5 Issue 2 represents the second issue of *WCHM* to appear online, and we are pleased to say that the debut of Issue 1 was quite a success. Our plan has been to further our reach and disseminate the content to providers in the wound care, hyperbaric, and diving arenas across the US and abroad. Thanks to the support of our sponsors, we are able to achieve this goal by making the content free. We encourage our readers to take advantage and share WCHM with colleagues and clients alike. In addition, if you have a clinic, be sure to add your details to our revamped Map of Wound Care and HBO Centers. Take advantage of this free resource and make your clinic easy to find for prospective clients and referral physicians.

Issue 2 includes submissions from our regular top-notch contributors, as well as submissions from several new authors to *WCHM*. Topping this list are UHMS physicians Steven Piper, Tracy LeGros, and Heather Murphy-Lavoie, who offer an introduction to the newest UHMS approved indication for HBOT, idiopathic sudden sensorineural hearing loss. We are also proud to feature author Philip James's introduction to his forthcoming text *Oxygen and the Brain*, a culmination of decades of research on oxygen, its physiological effects, and its importance in the treatment of injury and disease. We are grateful, as always, for all of the hard work that goes into each submission, and enthusiastically welcome you all.

We are also pleased to feature a dialogue between professionals, which has stemmed from an article published in Volume 4 of *WCHM* (please see the link on page 46 to access the original article "Wound Dressing Agents"). Author Michael Strauss clarifies the difference between wet-to-dry dressings and moist dressings, and which has an applicable use for chronic wounds. Such an exchange is what this publication seeks to elicit from its readers with every issue. We invite you to join in the discussion; please email us your thoughts, comments, and inquiries.

This issue is full of thought-provoking articles, making *WCHM*, in the words of John Peters, the MOST robust publication in the wound care, undersea, and hyperbaric medicine fields.

*Please join us in delivering the highest quality publication in the industry, focused on advancing the knowledge and practice of wound care, diving, and hyperbaric medicine, by providing your comments, case studies, industry information, press releases, and updates.*

Sincerely,

Jennifer Calabro
Managing Editor, *Wound Care and Hyperbaric Medicine* Magazine

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Wound Care & Hyperbaric Medicine

# Your Current Weather Report on ICD-10-CM

By Gretchen Dixon, MBA, CCS, CPCO, Principal Consultant at Professional Compliance Strategies, LLC

## Wooly Bear Technology

It seems over the years, the process of implementing ICD-10-CM may be considered worse than the accuracy of our daily weather reports. Why is it with the amount of technology available for providing weather reports, either nature or the Almanac seems to be the more accurate? This past fall it was predicted the Roanoke, VA area was going to have a cold and snowy winter. Now, I am not a meteorologist, but I did fall back on my grandfather's way of predicting winter weather. The alternative predictor was looking at a little wooly bear caterpillar's black and brown bands. If their brown stripe (between the black stripes) was wide that meant winter was going to be hard and we sure did have that. Twenty-two inches of snow in less than two days after our meteorologist predicted a few to six inches of snow—nothing to worry about. For future reference, if there was a larger black band than brown on the wooly bear caterpillar, winter would be mild. Much like relying on our local newscasters for weather advisements, are we using an outdated clinical documentation process?

Instead of being the naysayers in life, let's be prepared by continuing our current documentation improvement preparations during this time of reprieve to be sure we are ready. This is necessary as our healthcare infrastructure, including accountable care organizations and value-based reimbursement, requires ICD-10 which will be used for quality reporting and analytics with better data.

## What Happened?

CMS stated they were ready for ICD-10 implementation and did not expect the delay to come from Congress, per Denise Buenning, acting Deputy Director of CMS Office of E-Health Standards and Services. At present, CMS general counsel is developing an option to this delayed ICD-10 law, following protocol before reaching the Department of Health and Human Services for approval. The law does prevent DHHS from implementing ICD-10 before October 1, 2015; therefore, no specific details regarding the proposed options were provided, other than CMS is continuing on current processes. From CMS to MACs (Medicare administrative contractors) to third party payers (Blue Cross Blue Shield and United Healthcare, for example), everyone is telling providers to continue with the processes they are actively engaging.

## Currently

Look at your current state of documentation and ICD-9 code selection. What do you see? Are there a consistent number of **unspecified diagnosis codes** selected or assigned based on physician documentation? If so, this places your organization/practice in a higher-risk pool for possible future lower reimbursement based on the codes submitted to support the services provided. Diagnosis codes are necessary; however, the specificity of information has not been a focus.

So what are we supposed to do now? Focus on the source foundation for all patient services which is the provider's *clinical documentation*. The following items provide a global process:

1. Documentation Improvement: Practices have invested resources for the implementation of ICD-10-CM so don't stop your momentum. Take advantage of the delay to continue with clinical documentation improvement being the primary focus. Improved documentation today supports better ICD-9-CM codes, resulting in fewer unspecified selected codes being billed. This also supports the transition to ICD-10-CM as we now have extra months to prepare.
    a. Identify documentation gaps
        i. Ambiguity of information
        ii. Incomplete information
        iii. Copy and pasting of past information non-relevant to the reason for the encounter
        iv. Is there enough detail provided
        v. Is the information factual and accurate
        vi. Inadequately prepared templates or forms to gather detailed information
    b. Try to infrequently select unspecified diagnosis
    c. Has testing been validated for being up to date
    d. Validate status with vendor support through frequent communication during this transition to the new coding system

2. Optimize reimbursement now with selection of more specific and accurate codes/data
3. Do not procrastinate and do not ignore ICD-10 documentation requirements

## Healthcare Ambulatory Industry Weather Forecast

Weather can change before our eyes, so we have to heed the warnings and be proactive. In being proactive for the services provided, it is important to understand the change happening in outpatient ambulatory reimbursement. The ACA (Affordable Care Act) with HIPAA (Health Insurance Portability and Accountability Act of 1996) are transforming ambulatory patient care with provider risk sharing. This means there must be transparency and accountability toward value-based managed care with the focus on improving health conditions. Now the physician/provider are sharing this risk by ensuring the diagnosis codes plus the services provided are accurately supported with documentation for reimbursement.

Currently, physicians are paid for the service provided to the patient. Sometimes weather changes take time to develop as does all activities we venture into daily. At present, physician/providers focus is on being reimbursed for services rendered or provided through the reporting of CPT (current procedure terminology) codes and diagnosis codes. If there is a reasonable diagnosis, whether it was specific or unspecified, the service(s) is reimbursed. That is, you get paid for what you do!

## Partly Sunny Days: Transparency and Accountability Risk Shared

Based on informal discussions with third party payers, billers, and HIM staff, payers are now expecting more accurate or specific diagnosis with the reporting of services. There are healthcare beta-sites in many other regions of the country now reviewing the use of unspecified diagnoses. No longer does is this equation service = payment. Now both diagnosis and service have an equal weight in the equation: Diagnosis + service = payment.

The identified risk is noted in adding the "WHY" was the service provided and not just "WHAT" service was provided. Through this revised equation, there needs to be an awakening of a possible reimbursement storm if documentation habits do not change through the addition of details to the patient's encounter. The storm is coming through the use of "unspecified diagnoses" reported using unspecified diagnosis codes. How does that affect your practice? Does your documentation accurately reflect the level of medical condition acuity and intensity/complexity of care provided to the patient? Most likely not.

## Stormy Weather? Level of Acuity and Intensity/Complexity of Care

When documentation does not have the level of detail necessary to demonstrate the patient's level of acuity (disease process) and intensity/complexity of care provided, the options for reporting both in codes usually translate to unspecified diagnosis. Most wound care patients are medically complex but not well documented throughout each encounter, thus the information is translated into the unspecified realm and does not support the level of care patients are provided. Missing is the accuracy of how complicated and difficult case management of a wound care patient can be. Unspecified information equals unspecified diagnosis codes and they do affect your profiles. These are monitored by CMS and third party payers as they gather data for reimbursement changes, not only now but for the future. How will this occur? Data mining for unspecified diagnosis codes translates to mean low level of acuity, and low intensity or complexity of patient care services may result in a decrease in reimbursement.

**Concern:** If a third party payer feels procedures are overly aggressive based on vague (unspecified) diagnosis, they can remove providers from their plans.

**Concern:** Provider profiles will be affected based on best practices viewed from payers

**Example:** Diagnosis of diabetes mellitus due to underlying condition with foot ulcer and ulcer debridement service was provided (Table 1).

**Table 1. Example of What you Must Document**

| | | |
|---|---|---|
| Laterality | Left<br>Right | Without specification the code would default to unspecified extremity |
| Anatomical Location | Plantar surface midfoot<br>Heel<br>Midfoot<br>Other part of foot: toes | Without specific anatomical information the code translation would result in an unspecified code. Unspecified code defines level of acuity and intensity/complexity of services as low |
| Level of Tissue Breakdown | Skin<br>Fat layer exposed<br>Necrosis of muscle<br>Necrosis of bone<br>Unspecified severity | An unspecified code in the future likely may not support a performed procedure such as debridement since the level of documentation lacks the level of acuity and intensity and complexity of the service |

Currently, physicians are paid based on the service or procedure reported, with a code for reporting the diagnosis regardless of whether a specific or unspecified code was selected. It is recognized there are times when an unspecified diagnosis is necessary; however, these selections should be infrequently used.

The issue with this methodology is there is a lack of recognition for the work effort between the level of intensity and complexity of services provided. With the new methodology, reporting an unspecified diagnosis will only support a low level of acuity, resulting in a decrease in reimbursement for the services. If a specific, detailed code is selected, the service would expect to have a higher reimbursement based on the higher level of intensity and complexity of the procedure.

**The take away message:**
Document clinical details to ensure diagnosis codes are as specific as possible and they support the level of acuity and level of intensity and complexity of services provided.

## Sunny Skies are Returning with Accurate Levels of Acuity and Intensity and Complexity of Care

Clinical documentation is the **key source of communication** about the patient's medical condition and the provider's thought and treatment plan. Therefore, accurately report your patient's level of acuity and intensity/complexity by beginning to improve today's documentation. Make your documentation accurate, clear, concise, and detailed with specificity in descriptions using facts ensuring diagnosis and codes selected accurately supported thus decreasing claim issues and more timely reimbursements.

The risk of weather is reduced when relevancy of documentation is clearly linked to the reason for the encounter or visit. Each component of your documentation (review of systems, physical exam, assessment, and plan of care) needs to have supporting evidence corroborating the reason for the patient encounter and services rendered.

## Take an Umbrella Just in Case of Weather Change

To improve clinical documentation, our focus is on a few specific and commonly reported diagnoses in the wound care settings. It is these diagnoses which require detailed information for the future application in ICD-10-CM; however, instead of using ICD-10-CM as a prime issue, we should be focusing on the level of details in the information to accurately communicate the patient's medical condition and medical necessity for the provided services.

Table 2 illustrates how today's documentation of a few common diagnoses can result in unspecified codes and how to improve the documentation for better codes. This tool highlights the details to be documented during a patient's visit to your wound care center or department.

**Table 2. Current Documentation vs. Improved Documentation**

| Current Documentation = Unspecified Diagnosis = Low Level of Acuity with Low Intensity/Complexity of Care | Detailed Documentation = Specific Diagnosis = Accurate Level of Acuity with Appropriate Intensity/Complexity of Care |
|---|---|
| Diabetes Mellitus—Document<br>• Type as 1 or 2<br>  o Controlled versus uncontrolled<br>• Identify the body system affected by linking condition with the ulcer due to (not inclusive)<br>  o Hyperosmolarity<br>  o Neurological Disorders (polyneuropathy, mononeuropathy, etc.)<br>  o Other specified manifestations<br>  o Peripheral Circulatory Disorders (angiopathy)<br>  o Unspecified manifestations | Diabetes Mellitus – Document three items<br>• Type<br>  o Type as 1 or 2<br>  o Hyperglycemia versus hypoglycemia<br>  o Drug or chemical induced<br>  o Due to an underlying condition<br>  o Other specified DM<br>• Body system affected (not inclusive)<br>  o Circulatory complications<br>  o Hyperosmolarity<br>  o Kidney complications<br>  o Neurological complications<br>  o Other specified complications<br>  o Skin complications (ulcers)<br>• Specific complications/ manifestations affecting the identified body system |
| Anatomical Location: Ulcer of plantar surface | Anatomical location + laterality + depth of ulcer<br>• Laterality – Left or right<br>• Anatomical location e.g., toes, midfoot plantar, etc.<br>• Depth of tissue involved<br>  o Skin—limited to breakdown of skin<br>  o With exposure of fat layer<br>  o With necrosis of muscle<br>  o With necrosis of bone |

**Table 3. Example of Coding Diagnoses with ICD-9-CM and in the Future with ICD-10-CM**

| Current ICD-9-CM Code Description (Diagnosis) | Future ICD-10-CM Code Description |
|---|---|
| Diagnosis: Diabetic ulcer planter area of foot (diabetes uncontrolled) | Diagnosis: diabetic ulcer of plantar area of right foot with necrosis of tendon due to circulatory disorder (Type 2 diabetes with hyperglycemia) |
| Type 2 Diabetic Ulcer of Mid-foot-plantar surface –707.14 + | Diabetes mellitus with foot ulcer – E11.621 + |
| 2 Codes required to completely describe condition:<br>1. Diabetic ulcer due to inadequate *circulation* ( DM uncontrolled type II) –250.72 +<br>2. Identify the manifestation (peripheral angiopathy) 443.81 | Diabetes mellitus due to underlying condition with diabetic peripheral angiopathy without gangrene – E11.51 +<br><br>Diabetes mellitus with hyperglycemia – E11.65<br><br>Non-pressure chronic ulcer of right heel / midfoot (plantar surface) with necrosis of muscle (tendon/ muscle) --L97.413<br><br>[Code L97.413 is a combination code to describe type of ulcer (pressure vs non-pressure), laterality, anatomical location & tissue involvement] |

## No Umbrella Needed

Nature has finally given us the beauty she so delicately paints in a variety of colors. We take deep breaths, enjoying the freshness of the air and the newness of nature's endeavors to provide us with peacefulness. With a feeling of wellbeing as clean air is drawn into our lungs and the color of nature's beauty firmly planted in our minds, we must seize this time to revise our plans for today and the future. Plan to include in your organizational changes toward the implementation of ICD-10-CM, now set for October 1, 2015.

Confused? You are not alone in this adventure which affects all healthcare settings. Even CMS was surprised with the manner in which congress slipped a short paragraph into the Protecting Access to Medicare Act of 2014 (HR 4302) consisting of a 121 page bill to delay ICD-10 implementation for an additional year to October 1, 2015. The beauty of the delay of ICD-10 implementation will now allow those physician practices and other healthcare entities to catch up to the industry as they need. There is no specific shattering change to be made with the delay except to make the most out of the added time to learn and replace old habits related to clinical documentation. Don't get caught in the quagmire of these new codes; only correlate the current ICD-9-CM codes to the new ICD-10-CM codes used in your specialty.

Physicians, review your documentation today—take the time to identify when you select an unspecified ICD-CM code (diagnosis) and what it would take to make your documentation detailed and specific. It is not necessary to focus specifically on the new codes but to understand the level of documentation needed to accurately code a patient's encounter. Your specific area's documentation can be improved today for future data mining, reimbursement, and profiling to identify best practices.

Enjoy the challenge of the season by making an informed decision to implement or improve your process of clinical documentation improvement by providers (CDIP) as this is the foundation for your healthcare services to be reported accurately.


**Gretchen Dixon** performs audits regarding the outpatient revenue cycle healthcare compliance arena. She has conducted compliance education and audits for outpatient departments and physician services with a focus on wound care department operations for more than seven years. She holds several credentials including an MBA in healthcare management; RN with a practicing license in New York and a 23 multi-state licensure from Virginia; AHIMA approved ICD-10-CM trainer and CCS; and is a certified healthcare compliance officer. As a long-time internal healthcare auditor, she identifies issues through audits of documentation, coding, and billing practices. 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 for accurate reimbursement of billed services. Contact her at gretchendixon@cox.net for more information.

Wound Care & Hyperbaric Medicine

# Prior Authorization to Ensure Beneficiary Access and Help Reduce Improper Payments

Press Release from The Centers for Medicare and Medicaid Services

**For Immediate Release, Thursday, May 22, 2014.**

The Centers for Medicare & Medicaid Services today announced plans to expand a successful demonstration for prior authorization for power mobility devices, test prior authorization in additional services in two new demonstration programs, and propose regulation for prior authorization for certain durable medical equipment, prosthetics, orthotics, and supplies. Prior authorization supports the administration's ongoing efforts to safeguard beneficiaries' access to medically necessary items and services, while reducing improper Medicare billing and payments. The proposed rule is estimated to reduce Medicare spending by $100 to $740 million over the next ten years.

"With prior authorization, Medicare beneficiaries will have greater confidence that their medical items and services are covered before services and supplies are rendered. This will improve access to services and quality of care," said CMS Administrator Marilyn Tavenner.

The announcement builds upon lessons learned from the Medicare Prior Authorization of Power Mobility Device Demonstration. Launched in 2012, the demonstration established a prior authorization process for certain power mobility devices. Based on September 2013 claims data, monthly expenditures for certain power mobility devices decreased from $12 million in September 2012 to $4 million in August 2013 across the seven demonstration states (California, Florida, Illinois, Michigan, New York, North Carolina, and Texas) with no reduction in beneficiary access to medically necessary items.

CMS seeks to leverage this success by extending the demonstration to an additional 12 states. These states include Arizona, Georgia, Indiana, Kentucky, Louisiana, Maryland, Missouri, New Jersey, Ohio, Pennsylvania, Tennessee, and Washington. This will bring the total number of states participating in the demonstration to 19.

CMS also proposes to establish a prior authorization process for certain durable medical equipment, prosthetics, orthotics, and supplies items that are frequently subject to unnecessary utilization. Through a proposed rule, CMS will solicit public comments on this prior authorization process, as well as criteria for establishing a list of durable medical items that are frequently subject to unnecessary utilization that may be subject to the new prior authorization process. The proposed rule is currently on display at https://www.federalregister.gov/public-inspection and will be published in the Federal Register on May 28, 2014. The deadline to submit comments is July 28, 2014.

CMS will launch two payment model demonstrations to test prior authorization for certain non-emergent services under Medicare. These services include hyperbaric oxygen therapy and repetitive scheduled non-emergent ambulance transport. Information from these models will inform future policy decisions on the use of prior authorization.

Prior authorization does not create additional documentation requirements or delay medical service. It requires the same information that is currently necessary to support Medicare payment, but earlier in the process. CMS believe prior authorization is an effective way to ensure compliance with Medicare rules for some items and services.

For more information, go to: http://www.cms.gov/Research-Statistics-Data-and-Systems/Monitoring-Programs/Medicare-FFS-Compliance-Programs/Overview.html. Contactpress@cms.hhs.gov

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Wound Care & Hyperbaric Medicine

# Stresses in SCUBA and Breath-Hold Diving

## Part II: Physiological Stresses

By Michael B. Strauss MD and Stuart S. Miller MD

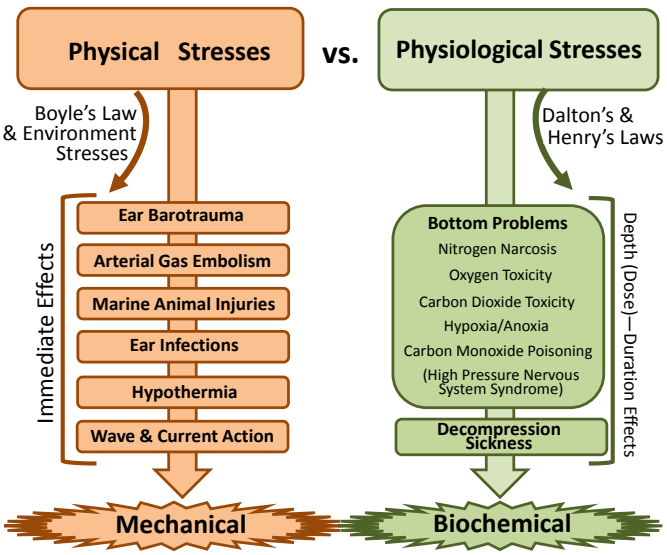
## Introduction

In the previous issue of *Wound Care and Hyperbaric Medicine* we introduced the subject of stimulus/stress—response/resolution and used this as the basis for discussing the physical stresses of diving.[1] In this edition we discussed the physiological stresses of diving using the stimulus/stress—response/resolution approach to the recognition, prevention, and management of this group of diving medical disorders. Physiological stresses of diving are a combination of factors in the diving environment and their effects on the cellular functions of the diver's body (Figure 1). In one sense, the physical stresses are mechanical while the physiological stresses are biochemical. The physiological stresses can lead to a variety of diving medical problems that range from blackouts to pressure-related problems of depth to decompression sickness. Most of the physiological stresses of diving are associated with a particular phase of the dive, such as decompression sickness occurring during the ascent/post-dive phase (Figure 2). This article describes the physiological stresses of SCUBA diving and the medical problems that can arise from them. A subsequent article will discuss the physiological stresses in breath-hold diving and their consequences.

The **physical** stresses of diving are best described by physics such as gas laws, thermal effects, and mechanical effects of the aquatic medium. The **physiological** stresses represent interactions at the biochemical and/or cellular level from breathing gases at increased ambient pressures.

The physiological stresses of diving reflect the challenges the underwater environment imposes on the normal functions of the body. The more the diver is subjected to the stress, the more likely a stress-related problem will occur; like a drug, each has a dose-duration profile. This means the physiological stresses of diving are both time and depth (i.e. pressure) related. Two gas laws, Dalton's and Henry's, provide the science for understanding the physiological stresses and their effects on the diver. As with the physical stresses, the body only has limited ways to mitigate the physiological stresses. When not resolved satisfactorily, harm comes to the diver.

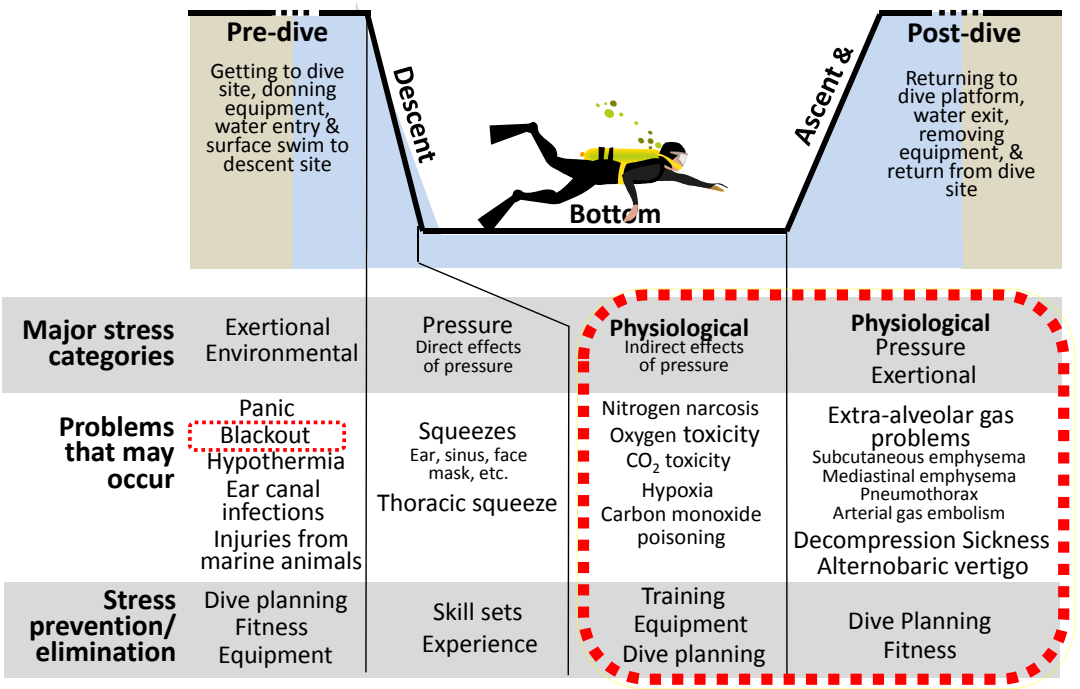
**Figure 1.**
**Physical vs. Physiological Stresses of SCUBA Diving**




**Legend:** Physical stresses of the diving environment have recognizable injury effects on the body; physiological stresses are reflected by biochemical interactions at the cellular level.

**Dalton's Law** is the gas law that explains the relationships between the components of a gas mixture and is formulated with simple arithmetic. The formula is $P_{Total} = P_1 + P_2 + \ldots$ and is verbalized as the total pressure in a confined gas mixture is equal to the sum of the partial pressures of the individual gases in the mixture. For air at sea level (only considering nitrogen and oxygen for simplification purposes), the total pressure is 1 ATA (atmosphere absolute) and equals the summation of the 0.79 partial pressure of nitrogen and the 0.21 partial pressure of oxygen. To be strictly accurate the partial pressures of carbon dioxide, water vapor, and the rare gases such as argon would also be considered even though they make up less than one percent of the total pressure.

## Figure 2.
## Phases of the Dive where Physiological Stresses Predominate




**Legend:** This all-encompassing synopsis of diving medical problems modified from our previous article[1] shows the phases of the dive where physiological stresses that can lead to medical problems occur. The blackout conditions are predominately associated with breath-hold diving and will be described in a subsequent article.

Comment: Dalton's law is particularly useful in diving science because it explains how varying the partial pressures of a gas mixture can reduce the chances of decompression sickness as well as explain the susceptibility to bottom-related problems such as nitrogen narcosis and oxygen toxicity. For example, in diving with pure oxygen as Navy divers do for clandestine military operations, there is no inert gas in the breathing gas and the total pressure in the system is entirely attributed to oxygen (Table 1). Consequently, decompression sickness will not occur, but oxygen toxicity becomes the time and depth limiting factor.

**Henry's Law** describes the effects that increasing (or decreasing) ambient pressure on a gas-liquid system has on gas moving into or out of the liquid phase. The formula is $PD_{Gas} \propto P_{Gas}$, which translates to the physically dissolved gas in the liquid phase of a gas-liquid system is proportional to the pressure of the gas in the gas phase (Figure 3). In the human body, the gas in the lungs is the gas phase of the system while the plasma (and tissue fluids) is the liquid phase. While residing at sea level, the partial pressures of the inspired gases in the lungs are in equilibrium with the partial pressures of these gases in the plasma and tissue fluids. The important "player" in this situation is the inert gas nitrogen which remains inactive, whereas the physically dissolved oxygen in the tissue fluids is utilized by the cells of the body for metabolism.

Comment: With increasing ambient pressure more gas (from the lungs) becomes physically dissolved into the plasma and tissue fluids. With decreasing ambient pressure, the opposite occurs. This has important ramifications for understanding why decompression sickness and nitrogen narcosis (as well as other bottom related problems) occur, along with the mechanisms of hyperbaric oxygen therapy.

## Table 1. Dalton's Law in Action

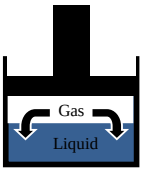
Inert gas ($N_2$ or He) fills the void between the $O_2$% in the breathing mixture & the ambient pressure… i.e. $O_2$ % + Inert gas % = 100%; *Dalton's Law*)*

| Breathing Mixture | $O_2$ | Inert Gas (to fill the void) |
|---|---|---|
| Pure $O_2$ | 100% | No void |
| Nitrox-36 | 36% | 64% |
| Air | 21% | 79% |
| 1,000 Ft dive | 1.0% | 99.0% |

Increased ↑ (Pure $O_2$ → Nitrox-36); Decreased ↓ (Pure $O_2$ → Nitrox-36); ↓ (Air → 1,000 Ft dive); ↑ (Air → 1,000 Ft dive)

* For simplification purposes, only oxygen and the predominant inert gas in the mixture are considered. To be completely accurate, water vapor, carbon dioxide, and the rare gases would need to be factored in as they are in computing diving tables. Regardless, they amount to less than 1% of partial pressure of atmospheric air.

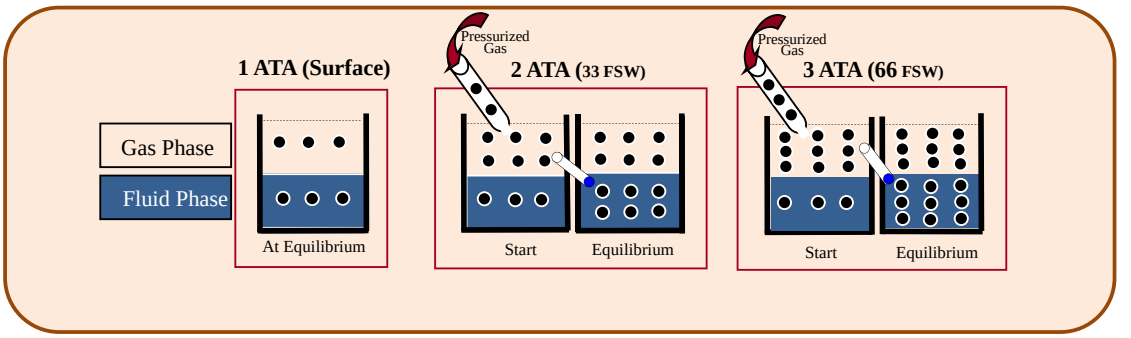
**Figure 3.**
**Henry's Law and the Dynamics of Pressure on a Confined Gas-Liquid System**



Piston Analogy

In a confined gas-liquid system the amount of gas in the liquid phase is a function of the pressure of the gas in the gas phase

The piston analogy is useful in understanding the ramifications of Henry's law




**Legend:** If the pressure in the gas phase is increased, more gas is forced into the liquid phase. This is analogous to increasing the ambient pressure of the breathed gas as the diver descends. Naturally, the opposite occurs with ascent with decreased pressure in the gas phase and corresponding movement of gas from the liquid phase to the gas phase. Equilibrium is reached when the pressures of the gases are equal in both phases.

While the piston analogy is a useful concept, solulability coefficients as well as possible chemical reactions between the gas and a liquid also occur. for understanding solubility coefficients as well as possible chemical reactions between the gas and the liquid. For example, fatty tissue on-gassing of nitrogen is five times greater than for lean tissues.

## The Stress-Response Phenomenon and the Physiological Stresses of Diving

Whereas the physical stresses in diving often become apparent before serious harm comes to the diver, there may be no or minimal warning effects before the physiological stresses manifest themselves. Consider middle ear barotrauma with descent. With this stress, and as soon as the diver feels discomfort in his/her ears he/she can mitigate its manifestation by slowing the descent. In contrast, the physiological stresses typically go unnoticed until a problem arises. For example, the diver will not be aware that an inert gas load is being added to the tissues during the descent and bottom phases of a dive, or that off-gassing during ascent and while on the surface is occurring. In a dive where decompression sickness does not occur, the diver will be oblivious to these physiological stresses. Conversely, if bubbles form, coalesce, and enlarge with ascent (Boyle's law) due to inadequate decompression, the signs and symptoms of decompression sickness arise without precursor, less-serious signs and symptoms.

## Phases of a Dive and the Physiological Stresses of Diving

When the four phases of a dive (1. Pre-dive→surface, 2. Descent, 3. Bottom and 4. Ascent→post-dive) are considered, the physiological stresses almost always occur during two specific phases of the dive. During the bottom phase of the dive the conditions of nitrogen narcosis, oxygen toxicity, carbon dioxide toxicity, hypoxia, carbon monoxide poisoning, and high-pressure nervous system syndrome occur. Decompression sickness, the other major consideration in physiological stresses, occurs only during the ascent→post-dive phase. In breath-hold diving, which is an entire subject in itself to be addressed in a subsequent article, physiological stresses leading to medical problems can occur in all phases of the dive.

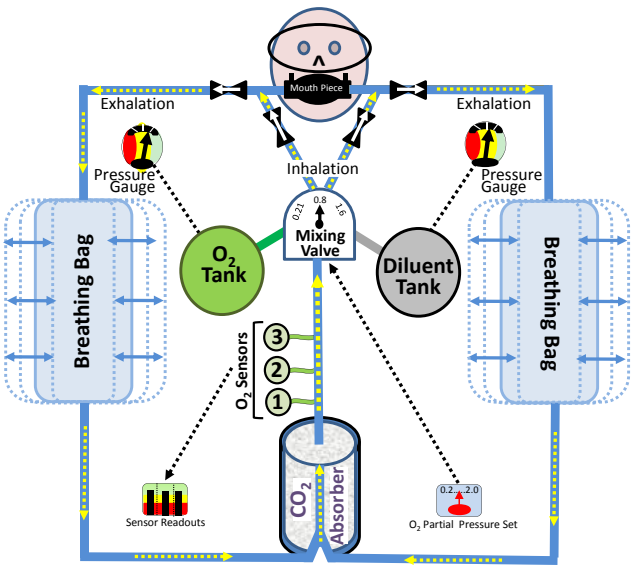
## Part II: Physiological Stresses of Diving*

*This is the second part in this article series. Part one, Physical Stresses of Diving, was published in the previous issue of *Wound Care and Hyperbaric Medicine.*

**Section A: Bottom Problems** The breathing of gases while at depth is typically associated with five diving medical disorders. They include nitrogen narcosis, oxygen toxicity, hypoxia/anoxia, carbon dioxide toxicity, and carbon monoxide poisoning, and for completeness may include a sixth, namely high-pressure nervous system syndrome. Like a drug with dose-duration effects, the longer the exposure and the deeper the depth, the more likely the bottom problems will occur. This differentiates these problems from the physical stresses of diving and reflects the interaction (or lack of in the case of hypoxia/anoxia) of the breathing gases with the body tissues. With typical SCUBA diving using open circuit air or nitrox (enriched oxygen) mixtures, the bottom problems occur infrequently. However, with closed circuit SCUBA (CCS) and the increased depths and durations of diving that can be achieved with this equipment, the diver must be ever mindful of the bottom related-diving medical disorders during the dive.

**Closed Circuit SCUBA Diving Breathing Equipment** In order to better appreciate why bottom problems are prone to occur with CCS diving gear, an understanding of how this equipment works is necessary (Figure 4). In CCS, the inert gas is recirculated as the diver breathes in and out from the gas in the breathing bags while precise amounts of oxygen are added to meet respiration demands. Carbon dioxide is filtered out by passing through a canister filled with soda lime. This is a very efficient system that maximizes the gas supply by precisely adding the amount of oxygen needed for respiration while resorbing carbon dioxide in the exhaled gas and recirculating the inert gas. In addition, the partial pressures of oxygen (i.e. number of oxygen molecules) can be varied with changing depths so optimal amounts of oxygen are available to meet respiration requirements, but not exceed limits that lead to acute oxygen toxicity (namely seizures). With this system, long duration and great depth dives can be achieved. Another advantage of the CCS is that no bubbles are exhaled, and so observing marine life is greatly enhanced. However, there are inherent concerns with using closed circuit SCUBA gear that increase the likelihood that a bottom associated medical problem will occur.

## Figure 4.
## Schematic of Closed Circuit SCUBA Equipment



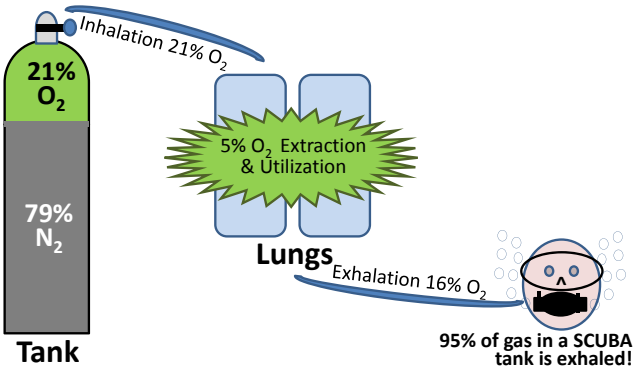

**Legend:** Closed circuit SCUBA is an efficient method to utilize gas supply, avoid oxygen toxicity, increase depth of diving, and extend diving time.

= Directional flow valve (with arrow showing the direction of gas flow).

Diving With open circuit SCUBA diving using air in the SCUBA tank, approximately 79% of the gas is nitrogen and 21% oxygen. When the diver inhales, about 5% of the oxygen is extracted by the lungs and is carried through the blood stream to body tissues for metabolic needs. Consequently, 16% (21-6%) is exhaled (Figure 5). This means that 94% of the gas in the SCUBA tank for open circuit SCUBA diving is wasted . . . such inefficiency! However, open circuit SCUBA adds a margin of safety to diving since carbon dioxide does not accumulate, being exhaled with each breathing cycle, and the oxygen percentage remains constant.

With closed circuit SCUBA, the oxygen utilization efficiency approaches 100%. With typical oxygen consumption of one liter a minute, a 12 liter oxygen tank can provide six hours of bottom time regardless of depth whereas, a 72 cubic foot tank with open circuit SCUBA diving is depth dependent and typically lasts less than an hour at a depth of 60 feet.

## Figure 5.
## Inefficiency of Gas Utilization with Open Circuit SCUBA Diving




**Legend:** Open circuit SCUBA diving is a very inefficient use of the gas in a SCUBA tank. Conversely, it is very safe because of the constant delivery of $O_2$ as well as the complete exhalation of $CO_2$.

The deeper the dive the more rapid the depletion of the gas in the SCUBA tank because the regulator delivers gas at the ambient pressure. If, for example, the pressure is doubled (33 FSW), twice the amount of gas [molecules] is breathed in with each inhalation.

It is important to understand the differences between oxygen partial pressures and oxygen percentages when respiratory physiology and combustion are concerned. For respiration purposes, the lungs are only concerned with the number of molecules of oxygen present. This is reflected as partial pressures (in accordance with Dalton's law). As long as the partial pressures of oxygen remain within a physiological range of 100- 160 mm Hg, normal lungs will function without difficulty in diffusing oxygen from the inhaled gas through the alveoli to the blood stream regardless of the ambient pressure. This, of course, assumes that the remaining gas partial pressures to bring the breathing medium to the ambient pressure are inert gases such as nitrogen or helium.

In contrast, the percentage of oxygen is a critical consideration for combustion to occur: It must be greater than 17%.

Comment This explains why a dive to 1,000 FSW (feet of seawater) requires only a 1% oxygen mixture while supplemental oxygen is almost always required for climbing the 29,000 foot summit of Mt. Everest (Table 2). Conversely, it would not be possible to kindle a flame on the 1,000 FSW dive while it is possible to ignite a camp stove at the top of the mountain because the percentage of oxygen (and not the partial pressure) determines whether or not combustion can occur. The bottom line is the lungs are a magnificent filter that are only concerned with the amount of oxygen molecules present (as reflected by the partial pressure) and not the percentage of oxygen in the mixture.

**Nitrogen Narcosis** The inert gas nitrogen in the breathing mixture begins exhibiting physiological effects when breathed at increased depths. This is often referred to as "rapture of the deep." Although the exact physiological mechanism is not known (at least four theories are used to explain it), breathing nitrogen in the gas mixture under increased pressure begins acting on the brain cells in a fashion analogous to an anesthetic agent.[2] The deeper the dive, the more likely the symptoms will occur. Symptoms range from euphoria to confusion and impaired judgment to unconsciousness. The effects, not surprisingly, are very similar to alcohol intoxication.

As in alcohol consumption, there is great variability at what depth nitrogen narcosis occurs and how its symptoms will be manifested. It appears experience, diving in warm water, clear visibility, physical conditioning, shorter duration dives, pre-dive planning, and briefing on "signals" that nitrogen narcosis might be occurring allow divers to avoid this problem even though they may exceed the three martini (150 FSW) depth.

This analogy is further expressed in the "martini law." In effect, the narcosis effects of nitrogen are paired with martini consumption such that each 50 feet of descent equates to drinking one martini (Figure 6). By 150 FSW of depth, most divers would begin to experience the effects of nitrogen narcosis similar to the inebriation from the drinking three martinis in rapid succession.

With open circuit SCUBA using air, sports divers are advised not to exceed a 130 FSW maximum depth. Using a single SCUBA tank, the gas supply would be consumed in about 15 minutes. With closed circuit SCUBA, depth and gas duration can far exceed these limitations. To mitigate the effects of nitrogen narcosis, the non-narcotic inert gas helium is used for the diluent gas in closed circuit SCUBA and deep diving activities.

As with other diving medical disorders nitrogen narcosis must be prevented by safe diving activities. Ascent, with the resultant effect of lowering the partial pressure of nitrogen, immediately ameliorates the symptoms. The danger is that while under the influence of nitrogen narcosis, the diver does something foolish (such as the typical analogy of offering his/her regulator mouthpiece to a fish to breathe with or descend following an imagined mermaid), breathe-

**Table 2. Pressure Changes with Altitude and Depth Excursions**

| Altitude/Depth Feet (Meters) | Pressure (Atmospheres) | Comments |
|---|---|---|
| 68,000 (20,726) | 0.055 | Armstrong constant; blood "boils" |
| 29,035 (8,850) | 0.25 | Mt. Everest; able to kindle a flame, but partial pressure of $O_2$ only 40 mm Hg |
| 18,000 (5,486) | 0.5 | 50% ↓ in $O_2$ partial pressure (pp) |
| 5,000 (1,760) | 0.8 | Denver—the "mile high city" |
| Iso△ p* Sea level (0) | 1.0 | $O_2$ pp = 160 mm Hg; $O_2$% = 21 |
| 16.5 fsw (5) | 1.5 | 50% ↑ in $O_2$ pp breathing air |
| 33 FSW (10) | 2 | Depth limit pure $O_2$ (pp = 2 ATA) |
| 99 FSW(30) | 4 | OC SCUBA tank gas duration ~ ¼ as on the surface |
| 1,000 FSW (305) | 33 | Breathing mixture ~1% $O_2$ |

Ascent/Altitude

Descent/Depth

Iso Δ p Indicates the descent/depth excursions needed to increase the ambient pressure by 50% with sea level pressure being the reference point. An 18,000 foot descent in the atmosphere to sea level increases the ambient pressure by 50%; in water a 16.5 foot descent from sea level results in a 50% increase in ambient pressure. These differences are a manifestation of the over 700-fold increase in density of water as compared to air as discussed in the previous edition of *WCHM*.[1]

**Figure 6.**
**The "Martini Law" and Nitrogen Narcosis**


**Legend:** The "martini law" is a graphic example of how nitrogen narcosis affects a diver with descents. The narcotic effects of nitrogen is roughly equivalent to each 50 FSW (feet of seawater) descent. By 200 FSW (4 martinis) a strong narcotic effect is likely.

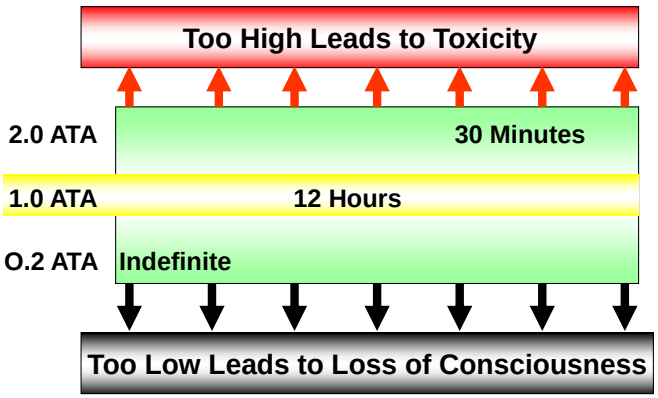
There is great variation in the susceptibility to nitrogen narcosis. Factors such as experience, diving in optimal conditions, conditioning and pre-dive planning raise the threshold. Exertion, cold water, alcohol and "downer" medications, and inexperience lower it.

in sea water, or bolt for the surface while breath-holding at the risk of sustaining an arterial gas embolism. Hopefully, an experienced dive buddy or dive guide would recognize the predicament and have the diver ascend to a depth where the nitrogen narcosis effects would disappear without incident.

**Oxygen Toxicity** Tissues of the body require a relatively narrow oxygen "window" with the most sensitive being the brain and lungs (Figure 7). The window ranges from partial pressures of about 0.2 ATA/150 mm Hg to 2.0 ATA /1,520 mm Hg. Oxygen partial pressures below the lower limits of this range lead to hypoxia/anoxia which will be discussed subsequently. Like the other physiological problems of diving, the oxygen exposures at the high end of this range which lead to oxygen toxicity are both dose/depth (i.e. partial pressure) and time (i.e. duration of exposure) related. Oxygen toxicity is manifested primarily in two different organs, the brain and the lungs.

Although guidelines to exposure exist, oxygen toxicity thresholds vary from individual to individual and appear to be lowered by activity level (possibly from carbon dioxide accumulations in the tissues), acidosis, fever, possibly steroid use, and previous history of seizures. The time honored 2 ATA (33 feet of sea water) for 30 minutes guideline has been modified by the US Navy to allow longer durations of pure oxygen breathing at shallower depths to almost unlimited times at 10 foot depths. In clinical applications of hyperbaric oxygen, exposures at 3 ATA (66 FSW) are done for decompression sickness, arterial gas embolism, carbon monoxide poisoning, and gas gangrene. After these durations either air breathing breaks or ascent to a shallower depth are made.

**Figure 7.**
**The Oxygen Window**




**Legend:** Oxygen is the respiratory gas requisite for the metabolic processes in higher organisms. The body tolerates a relatively narrow range of oxygen pressures.

Exposure durations are also a critical variable. If below the limit to sustain consciousness, unconsciousness occurs rapidly. Intermediate exposures with respect to pressure are observed after 12 hours with the target organ being the lungs and pulmonary edema the manifestation. Seizures occur with breathing high pressures of oxygen; the higher the pressure, the more quickly the seizure will occur.

In SCUBA diving the main oxygen toxicity concern is a seizure, known as the Paul Bert effect. It appears that acute, high doses of oxygen excite brain cells, causing increased discharges of electrical signals in brain neurons which manifest in the symptoms and signs of oxygen toxicity. There may or may not be prodromal symptoms before a seizure occurs. Prodromal symptoms include anxiety, tunnel vision, tremors, tinnitus, and/or nausea (ATTTN . . . attention with a triple "T"). If any of these are noted, the diver should immediately heed the warning signals, ascend to decrease the partial pressure of oxygen, and lower the set point of the oxygen mixer to that of air (i.e. 21% oxygen). When open circuit SCUBA diving with air, an oxygen seizure is very unlikely to occur since the exposure guideline would only be exceeded after a 30 minute dive to a depth of 300 feet. Problems of gas supply and nitrogen narcosis would likely occur long before the oxygen toxicity threshold is exceeded. When a seizure occurs below the predicted oxygen toxicity threshold, other factors such as hypoglycemia, a history of epilepsy (often overlooked innocently or purposefully by the diver on the pre-dive history form), and pre-dive use of "upper" drugs such as amphetamines should be sought.

To prevent seizures from oxygen toxicity, diving depth/time with Nitrox-32 (i.e. 32% oxygen) is limited to 130 feet for 20 minutes and Nitrox-36 (36% oxygen) is limited to 110 feet for 30 minutes. However because of the enriched oxygen mixtures, no-decompression stop dives are increased by 100% (10 to 20 minutes) and 50% (20 to 30 minutes) respectively versus air diving at these two depths.

## Table 3. Gas Mixtures with 0.8 ATA Oxygen "Set Point" for Closed Circuit Scuba

**Closed Circuit SCUBA**

No Bubbles

**Open Circuit SCUBA**

Bubbles "Galore"

| Depth (FSW) | ATA | $O_2$ pp | $O_2$% | $N_2$ pp |
|---|---|---|---|---|
| Surface | 1 | 0.8 | 80 | 0.2 |
| 33 | 2 | | 40 | 1.2 |
| 66 | 3 | | 26 | 2.2 |
| 99 | 4 | | 20 | 3.2 |
| 132 | 5 | | 16 | 4.2 |

0.8 = The partial pressure (in ATA) of the gas in the closed circuit SCUBA breathing circuit regardless of depth

EAD = Equivalent air depth (the depth where the gas mixture in the rebreathing circuit would be essentially equivalent to breathing air, i.e. 21% oxygen and 79 % nitrogen)

**KEY: ATA =** Atmospheres absolute, **FSW** = Feet of seawater, **pp** = Partial pressure in ATA

With closed circuit SCUBA diving, planning must give careful consideration to not exceed oxygen toxicity limits. For clandestine military 100% oxygen closed circuit SCUBA diving, depths and durations of the dive depend on the mission, but must never exceed oxygen exposure limits. For closed circuit sports diving where a diluent gas (most frequently nitrogen) is used, oxygen sensors coupled with mixing valves to add or reduce oxygen to the rebreather circuit are set to maintain a constant oxygen partial pressure regardless of the depth. Incorrect sets on the monitors or equipment failures can result in toxicity-producing exposures to oxygen. For safety purposes, typically three oxygen sensors are used to monitor the oxygen delivery. The dive should be immediately terminated if two of the sensors fail. As the diver ascends with closed circuit SCUBA, he/she may switch to higher partial pressures of oxygen (e.g. from 0.8 ATA to 1.6 ATA) to increase the gradient for nitrogen off-gassing and washout and reduce the time for decompression.

A typical oxygen partial pressure is 0.8 ATA for sport closed circuit SCUBA. At the surface the diver would breathe 80% oxygen and 20% nitrogen. As the depth increases, the 0.8 ATA oxygen partial pressure is maintained while increased partial pressures of nitrogen are needed to equilibrate with the ambient pressure as demonstrated by Dalton's law (Table 3). At a depth of 100 FSW the mixture would essentially be equivalent to breathing air with 20% oxygen and 80% oxygen.

Pulmonary oxygen toxicity, the Lorrain Smith effect, is unlikely to occur with SCUBA diving. Symptoms include dyspnea and feelings of tightness in the chest. The sustained high oxygen percentages injure the alveoli in the lungs causing them to leak fluid, which leads to pulmonary edema and impaired oxygen diffusion from the alveoli to the blood stream. When decompression sickness or arterial gas embolism is treated with deep and/or extended treatment tables, pulmonary oxygen toxicity may occur. In non-diving situations, pulmonary oxygen toxicity begins to occur after continuously breathing pure oxygen for greater than 12 hour periods. The likelihood of this occurring can be calculated using the UPTD (unit pulmonary [oxygen] toxic dose).[3]

Whereas a seizure occurring on land is a serious event, when it occurs underwater it can be life threatening. This is because of the inability to maintain or place (if the jaws are clenched from the tonic phase of the seizure) the regulator in the unconscious victim's mouth, which leads to aspiration of water, anoxia, and near drowning/drowning. In-water rescue is challenging. Attempts should be made to maintain the mouth piece in the victim's mouth and ascend with the victim's neck in the head tilt position so as not to block passive egress of air from the lungs during ascent. Usually the seizure is self-remitting so if water aspiration can be prevented, survival is possible. However, history of a seizure is a strong contraindication for SCUBA diving.

**Carbon Dioxide Toxicity** Carbon dioxide is a waste product of metabolism. Generated by metabolic processes in the cell, it diffuses into the blood stream, is carried by the blood stream to the lungs, and is exhaled by the lungs to the environment (or if underwater, the surrounding water). Carbon dioxide increases 100-fold from inspired air (0.05%) to expired gas (5.0%) from the lungs. Elevated carbon dioxide tensions in the blood have both physiological and pathological effects. As mentioned in part one of this article series, imperceptible elevations of carbon dioxide initiate breathing, a positive physiological stress, and increase the respiratory rate as associated with exercise.[1] Pathological elevations of carbon dioxide in the blood lead

## Table 4.
## Effects of Carbon Dioxide Elevation

| Symptoms/Signs | Percent | Comments |
|---|---|---|
| Anxiety, imperceptible increase in respiratory rate | >0.05 | Feeling something is wrong |
| Headache | 1 | Dilation of blood vessels |
| Dyspnea, hyperventilation | 3 | Direct effect on chemoreceptors and possibly associated hypoxia |
| Nausea, weakness, major apprehension | 5 | Extreme concerns about survival and/or extrication from predicament |
| Confusion | 5–10 | Possibly compounded by hypoxia |
| Loss of consciousness, death | >10 | Possible toxic effects on heart; possible overwhelming nervous system responses to $CO_2$ |

When confined to an enclosed space such as preparing for a lockout from a submarine hatch, carbon dioxide can accumulate as the diver exhales into the confined environment before going on SCUBA. Also, working in the head down position in a deep sea diving rig can result in carbon dioxide accumulation in the confined space of the diving helmet.

With closed circuit SCUBA, carbon dioxide toxicity must be prevented by proper pre-dive equipment preparations. Any suggestion of a possible malfunction in this portion of the breathing system (e.g. increased respiratory rate, headache, burning sensations in the mouth and airways—a sign that water has reacted with the Baralyme) should signal the diver to terminate the dive. Other management techniques include switching to the open circuit SCUBA mode or breathing gas from the safety bailout bottle.

**Hypoxia/Anoxia** Oxygen, of course, is involved with almost all of the metabolic processes of the body. The brain is the most sensitive organ to hypoxia/anoxia insults. A moment's interruption in oxygen delivery to the brain will result in loss of consciousness even though breath holding is possible for seconds or even minutes without loss of consciousness. Brain hypoxia/anoxia in open circuit SCUBA diving usually results from exhausting the gas in the tank. Only rarely is equipment failure the reason. In such situations the diver usually is immediately aware of the crisis and can surface from the dive and/or buddy breathe with the dive partner's octopus regulator. With entrapments, the victim may lose consciousness before corrective measures can be initiated. With closed circuit SCUBA, the onset of hypoxia/anoxia is usually more insidious. With closed circuit gear, hypoxia may occur leading to unconsciousness without warning signs. This is because carbon dioxide, the physiological warning mechanism, is maintained at normal levels through reabsorption in the closed circuit breathing system. Consequently, frequent checks of oxygen monitors are essential to ensure that oxygen partial pressures are adequate to maintain consciousness when diving with closed circuit SCUBA gear.

Even though one is holding his/her breath, blood is flowing to the brain and oxygen is being supplied to the brain cells. Trained individuals can hold their breaths for over 10 minutes and even longer with pre-breath-holding oxygen breathing. Conversely, with interruption of blood flow (and oxygen availability) to the brain, consciousness is lost in a second or two. This is confirmed by almost instantaneous loss of consciousness with the choke hold, a forceful Valsalva maneuver after hyperventilating (which vasoconstricts the arteries and decreases blood flow to the brain coupled with decreased heart filling with Valsalving), and third degree heart blocks. After four minutes of anoxia death of brain cells is believed to occur, but hypothermia and perhaps hyperbaric oxygen may extend the survival times or help hypoxic brain cells recover function.

**Carbon Monoxide Poisoning and High-Pressure Nervous System Syndrome** These are two additional problems which can be considered physiological stresses to the divers during the bottom phase of the dive. If the breathing gas is contaminated with carbon monoxide, symptoms similar to hypoxia/anoxia can occur. Carbon monoxide binds with hemoglobin 200 times more strongly than does oxygen. The result is deficient oxygen delivery to body tissues, which is really another type of tissue anoxia/hypoxia.

Poisoning is the preferred term describing the effects of carbon monoxide since it is not a gas associated with the normal physiology of metabolism. Conversely, toxicity is the preferred terminology for problems related to oxygen and carbon dioxide since these are substances associated with tissue metabolism.

Divers should be cognizant of tank-filling techniques and ensure the intake pipes that pressurize the SCUBA tanks are not in the vicinity of the exhaust fumes from the hydrocarbon fueled compressors. Symptoms of headache, lightheadedness, and confusion during the dive may precede the loss of consciousness and should be reason the diver surfaces and checks his/her tank for carbon monoxide as well as ascertain if any of the other divers have experienced similar symptoms. Oxygen breathing is the initial first line intervention for carbon monoxide poisoning.

High-pressure nervous system (HPNS) syndrome is mentioned for completeness as another bottom-related physiological problem of diving. It is associated with dive depths of over 600 feet. The problem is attributed to helium interacting with nervous system tissues. Symptoms include

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When breathing air at sea level, it is takes about six hours to remove 50% of the carbon monoxide attached to the hemoglobin molecule. With breathing pure oxygen the time for this to occur is one hour and with hyperbaric oxygen at 3 ATA (66 FSW), the half time for clearing carbon monoxide from the hemoglobin is 23 minutes. In addition, hyperbaric oxygen appears to prevent brain cell injury (termed latent encephalopathy) that occurs days or weeks after the victim regains consciousness and recovers from the acute effects of carbon monoxide poisoning.

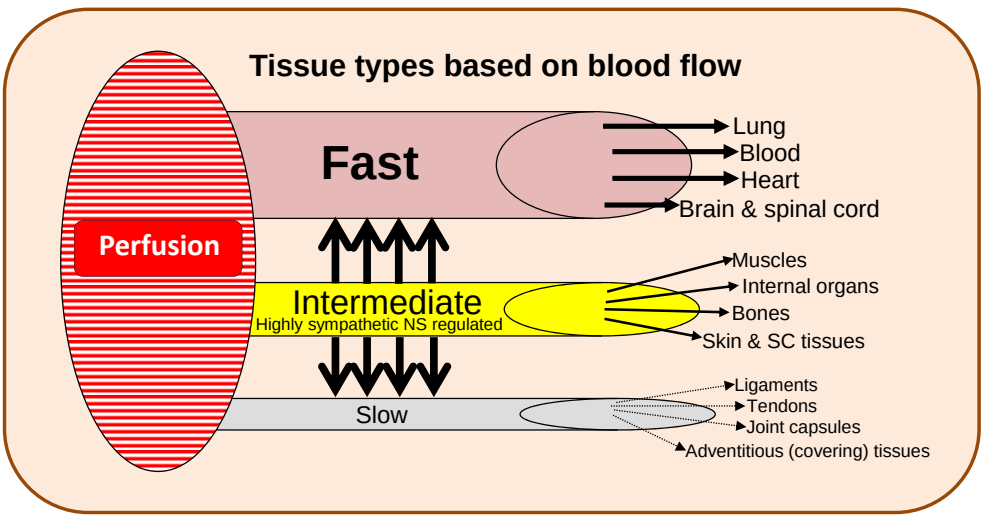
**Section B: Decompression Sickness, The Ascent Problem**

Whenever anyone discusses medical problems associated with diving, the first disorder mentioned is invariably "the bends." Although this problem occurs infrequently, about two to three incidents in 10,000 ascents, all compressed gas diving activities (with the single exception of diving with pure oxygen) are designed to prevent this disorder from occurring. Although we know much about decompression sickness (DCS), many aspects remain a mystery, such as why joint pains occur without associated physical or imaging signs, why bubbles nucleate, how much supersaturation of inert gas can occur before bubbles nucleate, why does one dive partner get bent while his/her buddy with an identical dive profile remain symptom free, why do some divers get DCS even though dive tables and/or computer profiles have not been violated whereas others flagrantly violate the tables/computers and do not get bent, when can a diver return to diving after an episode of DCS, are females more susceptible to DCS, etc.

The origin of the term "the bends" is derived from the bent over postures the turn of the 19th century Brooklyn bridge caisson workers assumed when walking after sustaining decompression sickness and having residual problems from it. Their walking posture somewhat resembled the fashionable posture ladies of luxury assumed when walking in New York City at the time and given the name, the Grecian bend. Although, the Grecian bend is a memory of the past, the bends terminology remains in common usage.

Although "the bends" is a widespread terminology, decompression sickness is the preferred term and encompasses all presentations from skin itches to joint pains to equilibrium disturbances to paralysis and even to death. Other names given to decompression sickness, most of which are related to the

**Figure 8. Variation in Blood Flow Among Different Types of Tissues**




**Legend:** The sympathetic nervous system and chemoreceptors precisely direct blood flow. The 5 liter blood volume must be distributed as needed to a vascular system that has a hundred liter potential capacity.

presenting symptoms include aeroembolism (not to be confused arterial gas embolism), aviators' bends, compressed air illness, divers' disease, divers' itch, divers' paralysis, dysbarism, silent bubbles disease, the chokes, the niggles, a vestibular "hit" (appearing as balance problems) and the staggers.

Decompression sickness is really the epitome of a physiological stress imposed on the diver, and an appreciation of the physiology of gas and fluid dynamics in the body with changes in ambient pressure is paramount in understanding this complex medical disorder of diving. At sea level the inert gas nitrogen is in equilibrium with all body fluids and tissues. With an increase in ambient pressure (such as descending and remaining on the bottom during a SCUBA dive) there is a gradient to drive the inert gas into the body fluids and tissues in order to reach equilibrium with the new ambient pressure. This is termed "on-gassing" and is explained by Henry's law. On-gassing occurs very rapidly in some tissues and very slowly in others (Figure 8).

Very rapid tissues with respect to on-gassing (and off-gassing) include the lungs, where equilibrium with the new ambient pressure occurs with the first few breaths at the new pressure, the blood stream, where the circulation time is less than 25 seconds, the heart, and the central nervous system (brain and spinal cord).

Intermediate tissues with respect to coming to equilibrium with new ambient pressures are those where blood flow is highly regulated by the sympathetic nervous system such as the muscles, the gut, the skin, and the subcutaneous tissues. For example, blood flow can increase 40-fold in muscles from the resting state to maximum exercise.

We have all heard the admonition by our mothers not to swim after eating. This makes sense when considering blood volume and blood flow distribution. With a blood volume of about 5 liters (quarts), yet a summated capacity (i.e. maximally filling every artery, vein, venous plexus, sinusoid, arterial-venous fistula, erectile tissue, spleen, and bone marrow cavity with blood) of over 100 liters, blood needs to be channeled where it is needed, be it for digesting food, exercise, wound healing, or fighting infection. From this information it is easy to understand why an abdominal cramp will occur if blood is channeled to the muscles and vice versa for a leg cramp after eating a large meal.

Slow tissues are those that have minimal perfusion and include tendons, ligaments, fascia, joint and organ capsules, articular cartilage, nerve sheaths, and adventitial tissues.

If the diver stays long enough at depth, all body fluids and tissues eventually come to equilibrium with the inert gas at the new ambient pressure and is termed *saturation*.

The establishment of inert gas equilibrium in all tissues is the principle used in saturation diving. In saturation diving, the divers remain at depth for extended periods to complete scientific studies or work missions. The divers may remain pressurized for days or even weeks while exiting their habitats for scientific observations or work projects. All tissues became saturated, i.e. they are in equilibrium with the new ambient pressure of the habitat. A single decompression/ascent is done once the mission is completed. For a diver saturated at a 50 depth habitat, ascending to the surface may take 24 hours, while a diver from a deep saturation depth (over 1,000 FSW) may take nearly two weeks to ascend.

With ascent, the opposite occurs and there is a gradient for "off-gassing" the inert gas in the tissues and blood as the ambient pressure decreases. Different tissues off-gas nitrogen (or helium if deep diving) at different rates, which are again based on perfusion. The tissue that has the most residual nitrogen is considered the "critical tissue." It varies from tissue to tissue depending on perfusion, the ascent rate, the multilevel dive profile, and the rest stop. The 64 dollar question is: how much oversaturation can a tissue tolerate before bubbles will form and lead to symptoms of DCS? Haldane postulated the ratio is 2:1, that is a 50% reduction in ambient pressure can be tolerated in saturated tissues without bubble formation and if greater than this ratio, bubbles will form. This has been observed to be appropriate for relatively short, shallow (to 80 feet) dives, but is apparently inadequate for longer, deeper dives. Consequently, Workman used "fudge" factors in modifying the 2:1 ratio with greater ratios for the shorter, shallower dives and lesser ratios for longer, deeper dives. Workman coined the term "m-values" for maximal allowable inert gas saturations for each dive profile.[5] Workman's "m-values" are used to generate the US Navy Dive Tables and provide the model for which almost all dive computer algorithms are based.

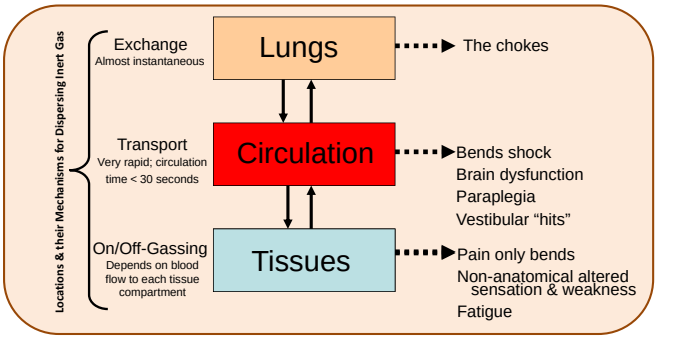
Since there are almost an infinite number of tissues and body sites that on-gas and off-gas with changes in ambient pressure, it would be impossible to measure the inert gas load in so many sites. To solve this problem, an exponential model (rather than specific tissue types) is used to calculate on-gassing, off-gassing, the critical tissue, and the safe decompression schedule.

In the exponential model, half times for on- and off-gassing are used (Table 5). Tissue half times are 5 minutes, 10 minutes, 20 minutes, to as high as over 500 minutes. In a 5-minute tissue, this translates to a tissue on/off-gassing 50% of the inert gas

over the new ambient pressure in 5 minutes and 50% again (i.e. about 75% of it) in 10 minutes. For the 10-minute tissue it would take 10 minutes to become 50% saturated—and so on as the exponential model is utilized.

It became apparent that a 5-minute tissue was inadequate to account for the on- and off-gassing of the ultra-fast tissues like the lungs and the blood. Wienke coined the term "free-gas phase" for the inert gas mechanics in the ultra-fast group of tissues.[5] As a consequence of his work, ascent rates have been decreased from one foot per second to one foot every two seconds and a three minute "rest" stop instituted at the 15 foot level of the ascent to better account for the off-gassing of these ultra- fast tissues.

## Figure 9.
## Decompression Sickness Presentations




**Legend:** The site of bubble formation determines the type of DCS presentation. Lung and perfusion presentations occur in briskly perfused tissues. Tissue presentation, e.g. limb bends, is a function of the on/off-gassing rates of the inert gas in tissues.

With this background it becomes apparent that bubbles, the basis of DCS, form when the supersaturated tissues exceed the ability of the body to off-gas the inert gas during ascent (Figure 9). If this occurs in the lungs, as seen only with explosive decompressions, it results in the chokes. Symptoms include shortness of breath, coughing, and the Behnke triad of increased respiratory rate, decreased blood pressure, and decreased pulse rate. If the bubbles form in the blood, DCS shock occurs with signs and symptoms identical to those of arterial gas embolism. When in doubt of which of the two conditions is the cause of the diver's problems, it is appropriate to label it *decompression illness*, an inclusive term that encompasses arterial gas embolism as well as symptomatic intravascular bubbles from DCS.

With the development of precordial Doppler monitoring, it became apparent that bubbles nucleate in the blood stream with almost all ascents. If not overwhelming they remain small and are carried to the lungs where they are filtered through the alveoli and exhaled to the outside environment. Because of the absence of symptoms, they are termed "silent" bubbles. This is an example of a physiological stress of diving that is resolved spontaneously, unknowingly, and without harm to the diver.

If the nucleated bubbles are not expediently delivered to the lungs and/or the gradients are over-

## Table 5. Exponential Model for Computing Tissue On- and Off-Gassing

Tissue half-time ($T_{1/2}$) and % saturation

| Time (minutes) | Tissue half-times (minutes) 5 | 10 | 20 | 40 | | 120 | |
|---|---|---|---|---|---|---|---|
| Start | -- | -- | -- | -- | -- | -- | -- |
| 5 | 50 | 25 | 12.5 | 6.3 | -- | ~0 ($1/2^{23}$) | -- |
| 10 | 75 | 50 | 18.8 | 9.4 | -- | ~0 ($1/2^{16}$) | -- |
| 20 | 94 | 75 | 50 | 25 | -- | ~0 ($1/2^{8}$) | -- |
| 40 | 99.6 | 94 | 75 | 50 | -- | 0.39 | -- |
| | -- | -- | -- | -- | -- | -- | -- |
| 120 | >99.9 | >99.9 | 99.6 | 94 | -- | 50 | -- |
| | -- | -- | -- | -- | -- | -- | -- |

= The semi-colored circles define each theoretical tissue compartment half time. That is, it is the time for the tissue to 50% off-load the inert gas partial pressure which exceeds the new ambient partial pressure. For example, for the 20 minute tissue half-time, 50% of the gas would be off-loaded in 20 minutes and another 50% (or a total of about 75%) in 40 minutes.

whelming, the bubbles coalesce, enlarge with ascent (as expressed by Boyle's law) and occlude the circulation to tissues. If this occurs in the spinal cord circulation, where a Batson plexus of veins is noteworthy for its sluggish blood flow, paraplegia results.

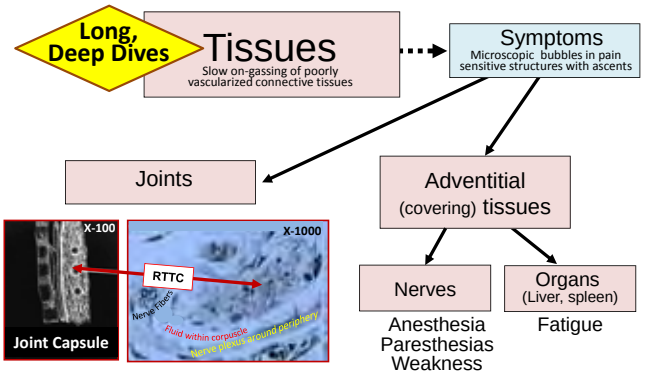
Verification of bubble formation in tissues as a cause of joint pain, minor neuropathies such as pain and paresthesias, skin rashes, and fatigue is not available. However, the phenomenon of *in situ* bubble formation, termed autochthonous bubble formation due to large gradients, is recognized as a mechanism leading to DCS and is analogous to opening a bottle of carbonated beverage, shaking it, and observing the formation of bubbles.

The Ruffini Type-2 corpuscle (RTTC) imbedded in joint capsules provides the best explanation to date why pain-only DCS occurs in joints (Figure 10). This organelle is a fluid-filled stretch receptor and helps provide the explanation why excruciating pain occurs with joint dislocations and sprains. These injuries stretch the joint capsule and presumably the RTTC, which in turn transmits the intense pain symptoms to the nervous system. It is remarkable how pain free the patient becomes after a dislocated joint is reduced and fits with the model that the RTTC is no longer stretched.

## Table 6. Types of Decompression Sickness

| Type | Symptoms | Sites Involved | Comments |
|---|---|---|---|
| 0 | Pruritus rash Fatigue | Skin, liver (postulated), muscle, gut | Usually not treated with recompression therapy, but HBO noted to relieve fatigue symptoms |
| 1 | Joint pain (limb bends), non-anatomical parethesias, weakness | Joints, peripheral tissues/ extremities | Cause not established, but Ruffini Type 2 corpuscle may be involved (Figure 10) |
| 2 | Paralysis, paresis; speech, visual, balance incoordination disturbances; dizziness, syncope, convulsion; dyspnea, shock | Spinal cord Brain Lungs Heart | Intravascular air bubbles obstructing circulation or interfering with gas exchange at lungs |
| 3 | Dizziness, vertigo, nausea, nystagmus, vomiting, tinnitus, deafness | Inner ear | Intravascular bubbles in inner balance and hearing structures |
| 4 | Joint pains, stiffness and swelling | Bone, especially juxta-articular | Intravascular air bubbles to juxta-articular bones cause bone cells to die; this leads to delayed bone collapse and arthritis |

## Figure 10. Source of Limb Bends and Other Type 1 DCS Presentations



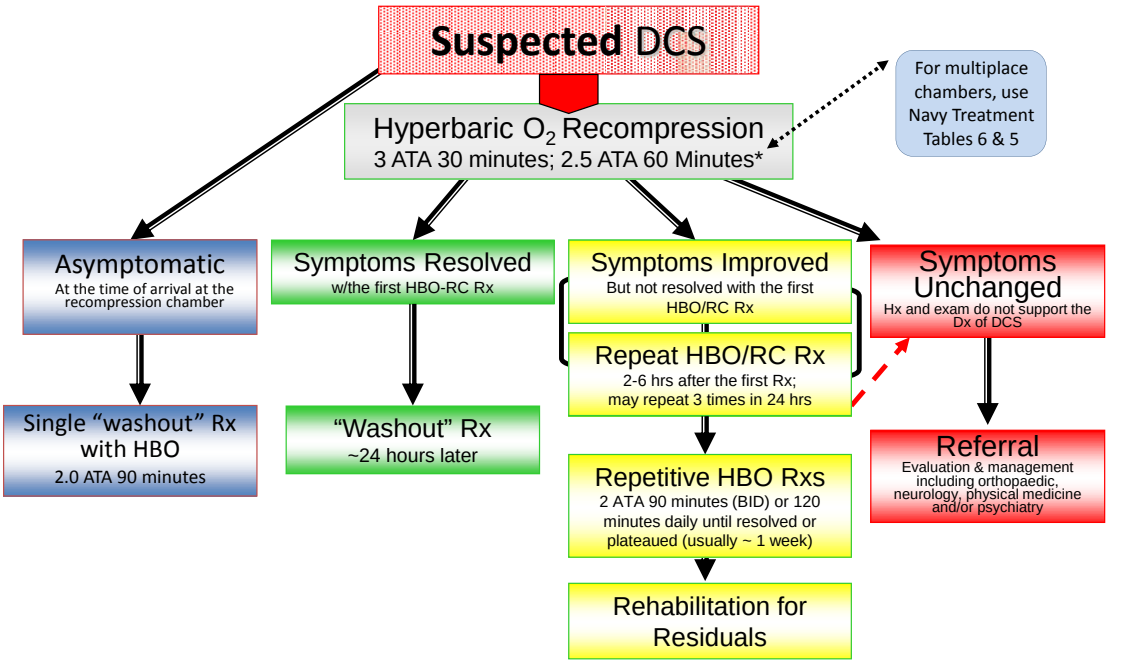

**Legend:** The Ruffini Type 2 Corpuscle (RTTC) is a microscopic organelle embedded in joint capsules and when stretched results in intense pain. The RTTC provides a good model for explaining pain symptoms in limb bends.

In pain-only bends, on-gassing is expected to occur slowly in joint capsules with the expectation that the inert gas also diffuses into the RTTC. With ascents the inert gas nucleates to form microscopic gas bubbles which coalesce and enlarge with ascent. This, in keeping with the model, stretches the RTTC from inside and generates the joint pain symptoms. Again, as in reducing a joint dislocation, the discomfort associated with pain-only DCS disappears almost immediately upon pressurization in a recompression chamber and is attributed to the reduction in the microscopic bubble size and elimination of the stretch on the RTTC. Since the bubbles are presumed to be submicroscopic in the 8-10 micron in length RTTC, they remain undetectable by our current Doppler technologies, are not visualized with imaging studies, nor have they been detected with pathology studies.

To explain the other minor decompression sickness symptoms, we postulate that microscopic bubbles form in nerve sheaths to account for non-anatomical numbness, paresthesia and muscle weakness, and in abdominal organ capsules and adventitial tissues to account for bends fatigue symptoms.

Since DCS has many presentations, they have been classified in a "lumper" system as minor (e.g. limb bends) or major (e.g. paraplegia). For classification splitters, five classes have been proposed (Table 6). Regardless of the symptoms, there are generally agreed upon treatment measures. After rescue and retrieval, breathing of 100% oxygen should be the first line of intervention. If the patient is alert enough to swallow safely, administration of clear fluids is recommended. Some advise taking an aspirin and/or a non-steroidal anti-inflammatory drug at this stage. The definitive management is hyperbaric oxygen recompression therapy

## Figure 11. Source of Limb Bends Pain and Other Type 1 DCS Presentations




**Legend:** This algorithm shows our monoplace hyperbaric chamber approach to managing decompression sickness (DCS). If at all suspected, even if the diver is asymptomatic at the time of presentation to the emergency department or chamber, hyperbaric oxygen recompression treatments are advised.

**BID**= twice per day, **Dx**=diagnosis, **Hx**=medical history, **Rx**=treatment

(HBO-RC). To discuss fully, the permutations of the HBO-RC treatment would require a description about equal to the length of this manuscript, but we include our algorithm to show that there is a logical approach to the HBO-RC management (Figure 11). In-water recompression is a controversial subject and most authorities recommend against it because of logistic considerations such as gas supplies, anchor lines, tenders, thermal challenges, and risk of seizures if pure oxygen breathing is utilized.

There is some scientific justification for fluid administration in as much as it will increase the blood volume with the expectations of speeding the washout of the inert gas as well as mitigating sludging in the microcirculation.

Aspirin and NSAIDs also have some theoretical merits. Aspirin will help counteract slugging, and perhaps more importantly, its anti-inflammatory properties may mollify the inflammatory reaction that inert gas bubbles generate in the capillary endothelium. When this is severe enough or long-standing, it may hamper the complete resolution of DCS symptoms. Information is being generated about endothelial condition for diving such as pre-dive exercise, condition through repetitive dives, and use of nitric oxide generating medications.

Steroid administration is another medication consideration with paraplegia presentations. Its benefits, if any, are probably due to its anti-inflammatory properties and moderating of the bubble-capillary endothelium inflammatory reaction.

In-water recompression involves immediately returning the diver who develops DCS symptoms at a remote site, far from a recompression chamber, to the water to re-pressurize. Two approaches exist: The Australian approach is to descend to 33 feet and breathe pure oxygen for 30 minutes. This requires an anchor line and a tender as well as an oxygen supply and equipment that can deliver pure oxygen at that depth and for that duration (which should not be a problem with rebreathing equipment). The Hawaiian approach to in-water recompression of their diver-fishermen with paraplegia or quadriplegia presentations is to place a tank on the diver and have him descend to a depth where the symptoms resolve, then gradually ascend.

The Divers Alert Network reports that delays in HBO-RC treatment for pain-only and/or other mild symptoms of DCS appear to not decrease the chances of recovery. However for paralysis symptoms, especially in remote diving areas where time delays may result in permanent residuals, in-water recompression needs to be carefully and thoughtfully considered.

After decompression sickness is treated, the first question the diver most often asks is, "When can I return to diving?" To answer this question it must be ascertained whether or not the decompression "hit" was deserved or undeserved. If deserved, and there are no residuals after the HBO-RC treatment, we recommend the diver not resume SCUBA diving for two weeks in order to allow the capillary endotheli-

um inflammatory reaction to resolve as well as counsel the diver on safe diving practices to avoid another deserved DCS incident. Other considerations in returning to diving include whether or not there are residual problems after the HBO-RC treatment and the diver's motivation to continue diving (Table 7).

The question of what constitutes a deserved versus an underserved episode of DCS is controversial. A deserved decompression "hit" is one where something can be identified that interferes with orderly off-gassing such as violating the dive computer profile, interference with circulation such as a constricting band from a neoprene suit, a syncope episode (which alters blood distribution to critical organs through loss of sympathetic nervous system tone), a cardiac arrest, etc.

A relatively recently identified disordering event is that of a patient foramen ovale. This allows the "silent" bubbles to pass from the right side of the heart to the left side of the heart and not be harmlessly filtered out and exhaled by the lungs. This results in the bubbles being carried into the arterial side of the circulation and blocking circulation to critical organs, most noticeably the brain and heart.

When an episode of DCS is not deserved, the diver should be wary of resuming diving because of the likelihood of future DCS episodes. It appears that a small segment of the population is "bends prone," which may possibly be attributed to occult hematological, perfusion, or sympathetic nervous system function disorders.

Safe diving practices include adhering to the dive computer (now almost no sport SCUBA diver uses dive tables) safe diving profiles; following the new "one foot every two seconds" ascent rates and 15 foot/three minute rest stop recommendations; maintaining good hydration and avoiding excessive alcohol consumption (which initiates diuresis and contributes to dehydration); avoiding diving when chilled or fatigued, especially at the end of the diving day; taking a day's diving "vacation" after three or four consecutive days of multiple dives; and not flying for 24 hours after SCUBA diving.

The dive computer is one of the most important developments that have occurred in sports SCUBA diving, possibly secondary only to that of the first stage dive regulator (the pressure step-down device to reduce the tank contents to a safe breathing pressure for the second stage mouthpiece). Whereas dive tables consider the maximum depth (even if just a "bounce") as the entire depth of the dive (which is appropriate for commercial and military diving projects where the divers are working at a constant depth), the dive computer takes into consideration the time spent in each depth as done in multilevel sport diving for a wide variety of tissue half times (Figure 12). This is more realistic as to what is actually happening with tissue on-gassing and off-gassing. The bottom line is that the dive computer allows more underwater time, more dives per day (Navy tables specify a limit of two dives in 12 hours), is more realistic of the amount of off-gassing that has occurred during the surface interval, and incorporates "fudge" factors for aggressiveness of the dive profiles, altitude diving, and type of gas mixture breathed. Even so, in about 50% of the cases of DCS, the diver did not violate the safe dive profiles specified in the computer readouts.

## Table 7.
## Return to Diving after a DCS Episode

1. **"Old Navy"** Guidelines: Treatment table 1=1 week→TT4=4 weeks if asymptomatic after recompression treatment.
2. **Behnke:** "Able to resume full running activity."
3. **Commercial diver workshop**: Next day if limb bends and asymptomatic after a hyperbaric oxygen recompression treatment.
4. **Strauss/Long Beach Memorial Medical Center Matrix**

| | Deserved | Undeserved |
|---|---|---|
| No Residuals | Yes (after 2 weeks, educate) | No* |
| Residuals | No (possible dive with special precautions | No |

* If the bends victim is determined to dive again then proceed with 1) cardiology consult (bubble study), 2) neurology consult (brain and spinal cord MR studies), 3) trial of recompression (60 feet for 60 minutes breathing air) and 4) instructions in conservative diving practices (nitrox, depth and time limitations, number of dives per day, etc.).

## Conclusions

The physiology-related stresses of SCUBA diving are fascinating because they represent interactions between the physics of the diving environment and the physiology of the diver (Table 8). They lead to a group of diving problems limited almost exclusively to the bottom and ascent, post-dive phases of the dive. Substantial information is known about the interactions. Dalton's and Henry's laws do much to explain why the stresses imposed by the diving environment interact with the diver's physiology. This helps to clarify why divers get medical problems related to the bottom phase of the dive and decompression sickness at the end of the dive. In addition, an understanding of the physiological stresses of diving helps provide information on how to treat the problem and when to return to diving. Even so, questions remain such as why similar stresses lead to problems in one diver and not another and in one diver on one day and not on another day with similar dive profiles. The subjects of bends-prone divers, endothelial responses to inert gas, endothelial "conditioning" to diving, the role of microparticles in evolution of bubbles, and verification of the source of pain in limb bends remain to be answered.

## Figure 12. Extended "Safe" SCUBA Dive Times Using the Dive Computer

| | 1. 90 feet 55 mins | 2. 60 feet 50 mins | 3. 80 feet 30 mins |
|---|---|---|---|
| |  |  |  |
| | **Dive 1** | **Dive 2** | **Dive 3** |
| Time at maximum depth | <1 minute | <3 minutes | 25 minutes |
| Average depth | 45 feet | 30 feet | 80 feet |
| Surface interval | ← 1 hour → | ← 3 hours → | |
| **Dive computer** | **Safe*** | **Safe*** | **Safe*** |
| Dive tables | 30 mins<br>Maximum allowable time at depth of 90 feet | 30 mins decompression required | Violations<br>Exceeds 2 dives in 12 hour rule; If tables used then hours of decompression required |

* One foot each 2 second ascent & 3 minute "rest" stop at 15 feet

**Legend:** The dive computer considers the actual on-gassing and off-gassing that occurs during multi-level SCUBA dives and the surface intervals. This allows more dives, accounts for off-gassing that occurs at shallower depths, and increases underwater time. A rectangular profile is used for Navy diving tables with the maximum depth utilized as the depth for the entire dive.

## Table 8. The Physical vs. Physiological Stresses of Diving

| Consideration | Physical Stresses Leading to Medical Problems | Physiological Stresses Leading to Medical Problems |
|---|---|---|
| Onset of Symptoms | Usually abrupt<br>e.g. ear squeezes and arterial gas embolisms | Usually gradual or a lag period |
| Precursors | Usually recognizable | Usually imperceptible |
| Applications | Physics Alone<br>Bubbles in arterial gas embolisms, pressure differentials in squeezes | Physics-physiology interactions |
| Site of Pathology Interaction(s) | Usually single system<br>e.g. middle ear squeezes | Usually multiple systems involved<br>e.g. lungs, blood, brain, heart and peripheral tissues in DCS |
| Occurrences | Usually resolved or prevented for every dive | Inherent in every SCUBA dive; invariably without symptoms |
| Gas Laws | Boyle's law | Dalton's & Henry's laws |
| Phases of the Dive | Descent and ascent, post-dive | Bottom and ascent, post-dive |
| Frequency | Ear squeezes are frequent | Bottom and ascent, post-dive problems are infrequent |

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**Michael B. Strauss,** MD, FACS, AAOS, has had a long-standing and keen interest in diving and diving medicine. His formal training started with Navy Submarine and Diving Salvage Schools. This was followed by tours on a nuclear submarine, with salvage divers in the Philippines and Vietnam and as the undersea medical officer for Underwater Demolition & SEAL Teams in San Diego. Dr. Strauss's special interests in diving include panic & blackout, disordered decompression, the source of pain in decompression sickness, diving stresses (Part two in this issue), diving in older age (published in the previous edition of *WCHM*) and mammalian adaptations to diving. As Medical Director of the Long Beach [California] Memorial Medical Center Hyperbaric Medicine Program, he continues active in diving medicine having evaluated and managed nearly 500 diving medical problems, generating over 50 papers & posters on these subjects, conducting yearly worldwide diving-diving medicine programs and authoring *Diving Science*, a well-acclaimed text that describes essential physiology and medicine for divers.


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Wound Care & Hyperbaric Medicine

# Idiopathic Sudden Sensorineural Hearing Loss: An Introduction

By Steven Piper, DO, FAAEM, UHM; Heather Murphy-Lavoie, MD, FACEP, FAAEM, FUHM; and Tracy Leigh LeGros, MD, PhD, FACEP, FAAEM, FUHM

## Introduction

The 13th edition of the *Undersea and Hyperbaric Medical Society Hyperbaric Oxygen Therapy Indications* is now available and includes the newest indication, idiopathic sudden sensorineural hearing loss (ISSHL). In June 2011, the Louisiana State University Undersea and Hyperbaric Medicine Fellowship proposed the addition of ISSHL to the list of indications, presenting the "pro" argument to the Hyperbaric Oxygen Therapy Committee at the UHMS Annual Scientific Meeting in Fort Worth, Texas. The committee voted in favor of the proposal and the addition of ISSHL to the list of indications was ratified by the UHMS Executive Board in October 2011.

## Background for $HBO_2$ Treatment for Patients with ISSHL

ISSHL is defined as hearing loss of at least 30 dB occurring within three days over at least three contiguous frequencies.[1] The most common clinical presentation involves an individual experiencing a sudden unilateral hearing loss, tinnitus, aural fullness, and vertigo.[2,3] The incidence is estimated at five to 20 cases per 100,000 people yearly (4,000 cases per annum) in the United States.[4,5] Although the etiology of ISSHL remains unclear, vascular occlusion, viral infections, labyrinthine membrane breaks, immune associated disease, abnormal cochlear stress response, trauma, abnormal tissue growth, toxins, ototoxic drugs, and cochlear membrane damage are among the potential pathophysiological mechanisms that have been described in the literature.[6]

## Rationale for $HBO_2$ Treatment for Patients with ISSHL

The rationale for the use of $HBO_2$ therapy in the treatment of ISSHL involves an appreciation for the high metabolism and vascular paucity of the cochlea.[7] The cochlea and the structures within it, particularly the stria vascularis and the organ of Corti, require a high oxygen supply and direct vascular supply is minimal.[8,9] Tissue oxygenation to these structures occurs via oxygen diffusion from cochlear capillary networks into the perilymph and the cortilymph, and perilymph oxygen tension decreases significantly with sudden sensorineural hearing loss.[9,10] This perilymph hypoxia is correctible with the extremely high arterial-perilymphatic oxygen concentration differences achieved by $HBO_2$, and additional benefits of $HBO_2$ may include anti-inflammatory effects, blunting of ischemia reperfusion injury, and edema reduction.[7, 9-11]

## Patient Selection and Clinical Management

Patients with moderate or worse ISSHL (> 40 dB) who present within 14 days of symptom onset should be considered for $HBO_2$.[7] Although the American Academy of Otolaryngology Head and Neck Foundation guidelines recommend that $HBO_2$ be considered for up to three months from the onset of symptoms, the best evidence suggests that early treatment within two weeks of symptom onset is associated with improved outcomes. Patients should undergo a complete evaluation by an otolaryngologist and audiologist, inclusive of appropriate audiological and imaging studies to confirm the diagnosis and determine disease severity. Patients meeting selection criteria for $HBO_2$ should also be treated with corticosteroid therapy and receive continued specialty follow up.[2,7] The recommended treatment profile is 100% $O_2$ at 2-2.5 ATA for 90 minutes daily for 10-20 treatments. The optimal number of treatments will vary depending on the severity of the hearing loss, the duration of the symptoms, and the response to treatment. Utilization review is recommended after 20 treatments as there is no data to suggest benefit beyond the range of 10-20 treatments.[7]

## Cost Analyses

Although there are no formal detailed cost analyses for ISSHL in the literature, the World Health Organization (WHO) has described the cost impact of hearing loss. Hearing impairment makes it difficult to obtain, perform, and retain employment. The cost of special education and lost employment imposes a heavy social and economic burden.[12] Adult onset hearing loss is the most common cause of disability globally and the third leading cause of years lost due to disability. Moreover, adult onset hearing loss is the fifteenth leading cause of burden of disease and

is projected to move up to seventh by the year 2030.[13] A review of the literature reveals more than 100 publications evaluating the use of $HBO_2$ for the treatment of ISSHL, including eight randomized controlled trials. Analyses of these studies have shown that $HBO_2$ therapy imparts a 19.3 dB gain for moderate hearing loss and 37.7 dB gain for severe cases.[14,15] This degree of improvement brings hearing deficits from the moderate/severe range into the slight/no impairment range, which has the potential to markedly improve a patient's quality of life both clinically and functionally.[12] For more details, please refer to Chapter 9 of the *Undersea and Hyperbaric Medical Society Hyperbaric Oxygen Therapy Indications*, 13th edition, and for a comprehensive review of this subject matter, please refer to Murphy-Lavoie et al. 2012.[7]

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# Hyperbaric Oxygen Therapy Indications
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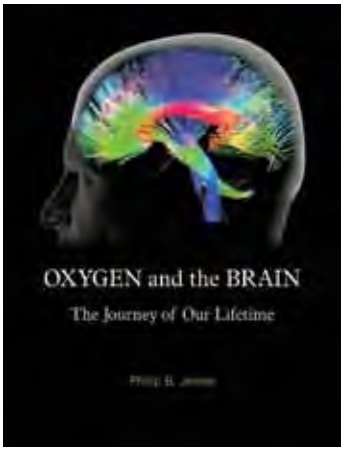
Wound Care & Hyperbaric Medicine

# Oxygen and the Brain, The Journey of Our Lifetime: An Introduction

By Philip B. James, Emeritus Professor of Medicine

No one can dispute that the brain uses oxygen or that the first indication of a reduced oxygen supply is loss of consciousness. Nevertheless, in a common illness like stroke where a reduction of blood supply leads to cell death from oxygen deprivation, giving more oxygen is not advocated in mainstream medicine. Yet, no organ exemplifies J.S. Haldane's seminal observation, "Lack of oxygen not only stops the machine, it wrecks what we take to be machinery" more than the brain. Despite it being obvious that normal blood oxygen levels cannot guarantee that tissue oxygen levels are adequate, haemoglobin saturation has achieved the status of a clinical constant. Equally around the world there is no undergraduate teaching of the importance of barometric pressure to oxygen delivery, or that oxygen is central to recovery– whether it is drug based or following surgery. This means that students qualify without knowing the basics of oxygen transport and most react with denial when confronted with hyperbaric oxygen treatment. A black hole has been created in medicine.




Equipment is obviously needed to increase the plasma oxygen level significantly, that is a pressure enclosure, and physicians nor, indeed, surgeons can claim to be unfamiliar with the technology: They probably fly more than any other section of society. Commercial aircraft use cabin pressure differentials similar to clinical treatment chambers and they are also equipped with oxygen equipment. This book, Oxygen and the Brain: The Journey of our Lifetime, traces the development of the use of oxygen and pressurisation in aviation through to space exploration – few know that there is a hyperbaric chamber on the International Space Station. The vast majority of doctors do not know how to use an increase in oxygen pressure to correct tissue hypoxia—from patients with problem wounds to those with head injuries—simply because it is not taught at medical schools. However, many doctors involved in using hyperbaric medicine, at least in the West, have entered the field from treating the bubble-related illnesses in diving and it is unfortunate that the fears engendered by the underwater world permeate hyperbaric oxygen treatment.

Lay people researching the literature discovering that a relative has died of a treatable condition like carbon monoxide poisoning quickly realise that there may be a failure of a duty of care. As long ago as 1977, faced with the insurmountable evidence of benefit from hyperbaric oxygen treatment in carbon monoxide poisoning, a judge awarded $5,888,000 to a young man left untreated with severe brain-injury. However, memories are short and, as with any professional group, ranks are closed and life goes on; little changes and carbon monoxide poisoned patients continue to languish in intensive care units. Nevertheless change is in the air: Medical libraries may still be inaccessible to the public but the Internet is allowing the facts to be found. It is to be hoped that with the direction this book provides, the genie will finally be out of the bottle.

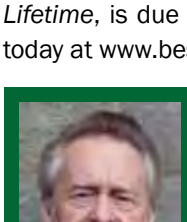
For most, the term "wound" evokes images of skin damage, but closed wounds result from internal injuries as, for example, in head trauma with damage to the soft tissues of the brain. Fortunately today, teaching is based on principles of disease and not rote learning. The critical and neglected principle in tissue damage is that not only are cells involved, the blood vessels within the tissue are inevitably compromised. This reduces both blood flow and oxygen delivery by two mechanisms: First, increased vessel permeability causes oedema. With the poor solubility of oxygen in water, the collection of fluid forms a barrier to oxygen transport. Secondly, disruption of blood vessel integrity causes haemorrhage, simply known as bruising. Both trigger the inflammatory response and oxygen controls inflammation.

Mention must be made of the dreadful impact of evidence-based medicine on thinking in the field of hyperbaric medi-

cine. There is no substitute for oxygen in either normal metabolism or healing, just as there are no substitutes for water or glucose. Oxygen is not a placebo and even small changes at the cellular level can have profound effects. Those who deny using more oxygen in treatment are, in effect, alleging that we cannot improve on the dosage in air and this, of course, changes with the weather and is reduced at altitude. Sadly, again failing to understand pressure, doctors are quick to marginalise those using chambers by implying "hyperbaric" oxygen is somehow different to ordinary oxygen - perhaps $O_4$? This is compounded by the incorrect grammar used by many in hyperbaric medicine: "Hyperbaric oxygen" is wrong; it should be "hyperbaric oxygenation."

Few could have anticipated the astonishing developments over the last decade about the role of oxygen in the control of our genes. The very same mechanisms that form capillaries in the developing embryo are responsible for the new capillaries that form throughout life in wounds. Changes in oxygen levels regulate over 8,000 of our most critical genes and much-maligned oxygen free radicals are essential to our very existence. Oxygen underpins every aspect of medicine from the release of stem cells, to the control of infection and the reduction of oedema in the swelling of head injury. *Oxygen and the Brain* is a celebration of the importance of oxygen in maintaining health and its pivotal role in the treatment of injury and disease.

Dr. James's book, *Oxygen and the Brain, the Journey of Our Lifetime*, is due to be released this June. Order your copy today at www.bestpub.com.



*Philip B. James, MB, ChB, DIH, PhD, FFOM qualified in medicine from Liverpool Medical School in 1966 and after a fellowship in surgical research studied industrial medicine in Dundee, Scotland. After Royal Navy training in 1973, he specialized in diving medicine, combining an academic post in the University of Dundee with consultancies to many international diving contractors. In 1983, he received the Craig Hoffman Award from the Undersea and Hyperbaric Medical Society for diver paramedic training and contributions to diving safety, including a minimum oxygen content in helium, high oxygen partial pressures in diver's emergency supplies, and the use of helium/oxygen mixtures in recompression treatment. In 1982, he published evidence for sub-acute fat embolism as a cause of multiple sclerosis in the Lancet comparing the pathology to decompression sickness and endorsing the use of hyperbaric oxygen treatment. Five multiple sclerosis patients treated by Dr. James in 1981 founded a community hyperbaric facility in Dundee, and there are now 65 charity centres operating in the UK and the Republic of Ireland providing low-cost hyperbaric oxygen treatment for neurological conditions. The centres were deregulated by Act of Parliament in 2008. Dr. James retired in the same year, but continues as honorary adviser to the charity, as a consultant to the offshore oil and gas industry, and a passionate advocate for using oxygen in treating disorders of the brain.*

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# Press Releases

## John Peters, CHT, MBA, FACHE Selected as Incoming UHMS Executive Director

Durham, NC, April 22, 2014. The Board of Directors of the UHMS is very pleased to announce that Mr. John Peters, CHT, MBA, FACHE has been selected and has agreed to assume the position of Executive Director of the Society effective at the time of Dr. Bennett's retirement this summer. We anticipate that this transition will occur between 1 June and 1 July 2014 depending on some final details that need to be concluded.

The search was very adeptly chaired by Dr. James Holm, our President-Elect. Other members of the committee include Dr. Neil Hampson, Dr. Bret Stolp, Dr. Laurie Gesell, Dr. Brett Hart, Dr. Michael Bennett, Mr. James Joiner and myself (Dr. John Feldmeier). After initial screening we narrowed the field to three finalists who were interviewed in person at the Durham office. The three finalists were all highly qualified, and I am convinced that any of the three would carry out the responsibilities with energy and skill. The Search Committee made its recommendation to the Board of Directors last week. The Board voted to approve this selection. Mr. Peters has agreed to accept the position and we have begun the final stages of contract negotiation with no issues expected to hamper the conclusion of an agreement.

Join me, the Board of Directors, the Search Committee and our staff in welcoming Mr. Peters to the Executive Director's position. John is a young man but well known for his accomplishments and expertise in business and hyperbaric medicine. He has committed to a 150% effort to the position. I believe that we will have his services as Executive Director for years to come and with this stability and the skill sets he brings, we will best be able to face the challenges to hyperbaric medicine anticipated in the next several years to come.

John J. Feldmeier, D.O., FACRO, FUHM
President, UHMS
Email: jfeldmeier@aol.com

###

## Congratulations to Gwen Gatlin on Receiving the Associate's Recognition Scholarship

The Associates Recognition Scholarship has been set up by the UHMS Associate's Council in order to recognize individuals who have shown initiative and promising careers in the field of hyperbaric medicine, but have not been able to attend or present at a UHMS conference previously. Candidates may be new to the field of hyperbaric medicine, or perhaps have worked in the field for a while, but have never participated in a UHMS conference because they did not have the funding to do so. The scholarship award is for $2,000 and is meant to encourage broader participation by the up and coming associate members.

The recipient of this year's Associates Recognition Scholarship is Gwen Gatlin. Gwen submitted a very compelling abstract that examines the increased number of insured patients, as a result of the Affordable Care Act, and the role Advanced Practice nurses can play in ensuring adequate providers are available to meet the increased demands.

Gwen began working in the field of hyperbaric medicine in October 2010. She currently serves as the Nurse Manager for three monoplace hyperbaric programs within the Sentara Health System. One of these programs serves as the only 24/7 hyperbaric program in the Hampton Roads area. Prior to that, she served 26 years in many roles in the U.S. Navy.

Gwen is currently enrolled in the Doctorate of Nursing Practice at Old Dominion University in Norfolk, Virginia, where she obtained her Master of Science in Nursing Administration in 2011, and her Bachelor of Science in Nursing, Summa Cum Laude in 1997.

The Associate's Council wishes to congratulate Ms. Gatlin for winning the scholarship. Additionally, we would like to encourage other Associate members who might qualify for this scholarship to begin thinking about a possible submission for next year.

###

UNDERSEA & HYPERBARIC MEDICAL SOCIETY

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Wound Care & Hyperbaric Medicine

# Wound Care Specialists in Home Health Care: The Light at the End of the Tunnel

By Michael O. Fleming MD, FAAFP; Monica Timko-Progar BSN, RN, CWS; Bobbie Stallings BSN, RN, CWON; Jerri Drain BSN, RN, CWON; Naomi Ward RN, BSN, CWS; Kimberly Smith BS, RN, CWON; Kimberly Bare BSN, RN, CWON

The Patient Protection and Affordable Care Act (PPACA) was signed into law by President Obama in 2010. It is the most significant change to the US health care system since the passage of Medicare and Medicaid in 1965. While the aim of PPACA is to reform the health insurance market, it also intends to reduce the overall costs of health care to make the Medicare and Medicaid programs more sustainable and at the same time improve the overall quality of care received.[1] As a result of changes in reimbursement and the need for hospitals, rehabilitation centers, and skilled nursing facilities to reduce length of stays, reduce readmissions, and control associated costs, the shift of care has moved from one of inpatient stays back to a home/community setting. Despite this shift, according to the Congressional Budget Office, the PPACA is predicted to reduce Medicare spending on home health care (HHC) by over $4 billion for federal fiscal years 2010-2014 and approximately $40 billion through 2019.[2] The result is the HHC industry must implement strategies to care for a more medically complex population while operating under reduced reimbursement. Providing a robust level of care to an increasing senior population with multiple co-morbidities, while maintaining high quality outcomes in a cost-effective manner and preventing re-hospitalizations, seems an impossible challenge when coupled with a reduction in home health care reimbursement.

In a recent article published in the *Journal of Wound, Ostomy and Continence Nursing* (*JWOCN*), out of 300,000 home health care episodes of care reviewed, 34% involved a surgical wound, pressure ulcer, or lower extremity venous ulcer and 60% of the episodes identified patients with bowel or urinary incontinence.[3] These statistics are significant as wound care costs remain a large part of home health expenditures. In addition, complications that arise from wounds such as infection contribute to the overall cost of health care and impact the acute care hospitalization readmission rates. In this dark time of outcome-driven reimbursement and rising health care costs with decreasing reimbursement, we are left wondering how to begin to move forward. Fortunately, there is light at the end of the tunnel to manage the rising cost of wound care and it comes in the form of a wound care specialist (WCS).

Research has shown patients who were cared for by or had involvement of a WCS had improved outcomes in terms of healing rates and cost to care for the patient as compared to those who did not.[4] In one study, HHC patients who had involvement of a WCS resulted in 78.5% of wounds healing compared to 36.3% when cared for by general staff nurses.[5] Despite what the research shows us, there is much confusion in regard to who exactly is considered a wound care specialist and what exactly that person does, which can lead to under use and under value of the role. In our complex health care system and particularly in HHC, both providers and patients require assistance in determining who is best qualified to deliver specialized wound services and management. How do we get the right care to the right patient at the right time when it comes to wounds in HHC?

Wounds and wound care in general present a challenge to HHC. Typically, a wound that does not respond to normal medical care within 30 days is considered a chronic wound. Chronic or non-healing wounds do not show significant improvement and are usually associated with other co-morbidities that impact healing such as diabetes, impaired circulation, immobility, incontinence, trauma, and surgery. Other factors such as nutritional status, medications, and lifestyle also impact healing. Non-healing wounds affect the quality of life of the patients we care for. Often times these patients are referred to a wound center for evaluation and subsequent care provision at home by an HHC agency. While the use of a WCS in both acute and long term care settings has caught on, only approximately 13% of WCSs practice in the HHC arena.8 Lack of knowledge about the specialty area of practice and confusion about what a WCS exactly is, who is really considered to be a WCS, and how a WCS can positively impact HHC impede home health care agencies from understanding the value in placing a WCS on their payroll.

So how do you know, when it comes to nurses practicing wound care in HHC, which are really considered WCSs and what is the most effective utilization their services? There are many programs and educational venues promoting wound care specialization and certification. These pro-

grams range from completion of a one to five day learning session available to anyone in the health care industry, to those with specific entry level requirements and ongoing testing to continue the credentialing. The later involve a much more complex degree of classroom and hands on learning in preparation of the certification exam and are nationally and internationally recognized as an advanced practice specialty. The wound, ostomy, continence nurse (WOCN) and the certified wound specialist (CWS) require a minimum of a bachelor's degree to be considered for the specialty practice and passage of a board certification exam at regular intervals to maintain the prestigious certifications. These certifications identify the clinician as a subject matter expert in a specialized area of practice, committed to cost-effective, evidence-based, outcome-driven wound care.

To further clarify the credentialing, the Wound, Ostomy, and Continence Nurses Society (WOCN), recently released their position statement defining the role and scope of practice for the advanced practice WOCN, the WOCN specialty nurse, and non-WOCN care providers (or what they refer to as wound treatment associates).[6] The American Board of Wound Management clarifies their certified wound specialist (CWS) credentialing to recognize a master level of knowledge and specialty practice in wound management.[7] These advanced certifications serve a variety of roles including direct patient care provider, administrator/wound care program manager, educator, and consultant. The WCS can provide ongoing support and expertise through holistic assessment, planning, implementation, and oversight of patient wound care. This includes providing quality patient management and continuity of care, enhancing dialogue between health care professionals and physicians, and acting as a liaison between the patient, family members/caregivers, and health care professionals to promote consistent outcomes, cost-effective wound care, and appropriate use of HHC services and resources. In addition, the WCS's familiarity with advanced wound care products and adjunct treatment modalities, along with early problem identification and prevention programs, are valuable services that can enhance outcomes and control costs. Taking advantage of current available technology options, including photography programs for wounds, can help to streamline consultative services and allow for an increased number of consults to be performed without the WCS leaving her desk. Highly certified WCSs possess specialized knowledge in wound care, anatomy and physiology, and general nursing concepts for provision of patient care. They demonstrate skills as assertive leaders with superior critical thinking skills along with effective organizational skills. As leaders and consultants within the health care industry, WCSs make a difference in the quality of their patients' lives by minimizing the potentially severe consequences of wounds. Helping wound patients heal and return to prior function is a top priority.

Innovative HHC agencies find ways to utilize the WCS to their advantage to get a bigger impact. For example, our large HHC agency relies on the expertise of our WCS to achieve positive outcomes with a focus on quality provision while providing cost-efficient wound care. Our wound care department consists of a director and five regional wound care program managers (RWCPM). Each RWCPM is responsible for responding to consultative and educational needs for their designated region. In order to support their respective regions, the responsibilities and functions of their role are ones of quality support, training, coaching, and field oversight. Their goal is to establish an ongoing relationship with clinical provider field staff and regional leadership in order to promote consistent, evidence-based, quality, and cost-effective wound care delivery. Our RWCPMs access reports, analyze data, and assist in formulating plans in conjunction with regional leadership. Based on identified need, they travel to care centers to assist with education and skills. This structure affords us the ability to assess and monitor outcomes while looking at product use and appropriateness of the plan of care. The consultative services help us establish partnerships with our care centers to work towards our goal of clinical superiority and providing a high level of quality care to our patients. Collaborative efforts with multiple departments within our agency have helped us to drive down the cost of wound care while maintaining positive outcomes.

## Conclusion

Regardless of the specifics of the WCS role and the environment in which they practice, several points remain constant:

- Their ongoing education and knowledge of current wound management guidelines, industry best practices, and evidence-based practice guidelines ensure patients receive the most up-to-date and highest level of quality care.
- They are able to identify and effectively manage the root causes for acute care hospitalization due to wound complications.
- Their correct identification of wound etiologies (pressure, diabetes, vascular disease—both arterial and venous, neuropathic, surgical, and trauma) ensures a plan of care that addresses not only topical wound care but adjunct treatments and therapies to promote healing as well.
- They are able to identify and effectively manage comorbidities, medications, nutrition, influencing factors and impacting lifestyles such as smoking, obesity, home environment, and support systems.
- They understand how utilizing current available technology options, including digital photography programs and telemedicine programs for wounds, streamline consultative services and allow for an increased number of consults to be provided.

- They provide effective pain management (both topical and systemic) to promote patient comfort.
- They can educate family/caregivers in proper, up-to-date, evidence-based practice.
- They encourage active participation of the patient and family/caregivers in setting goals and in implementing the plan of care.
- The education of provider field clinicians empowers them to drive quality wound care provision.
- The utilization of effective resources including wound management professionals, wound care teams, and wound care centers makes for success.

## Outcome Analysis

HHC agencies who remain open to innovative care provision models and consider the addition of a WCS to their team may find them to be the light at the end of the tunnel.

Meet our wound care team as we share some of our "PEARLS of Practice." These tips make a difference in wound healing times and outcomes.

**Monica Timko-Progar BSN, RN, WOCN, CWS, FAACWS**

The use of barrier skin wipes or skin protectants to the peri-wound skin is imperative in preventing peri-wound skin complications which may delay wound healing and therefore increase wound care costs. Including a barrier wipe or use of a petrolatum or zinc-based skin protectant is considered a best practice and should be included in any orders for wound care. In HHC it is particularly important as patients and care givers may be performing their own wound care a majority of the time with a nurse coming in once a week to assess progress towards healing. Using a protective skin barrier wipe or ointment provides relative assurance the peri-wound skin will remain intact and healthy and not become a deterrent to wound healing.

**Bobbie Stallings, BSN, RN, CWOCN**

A wound can be treated with the most expensive product available on the market, but without proper nutrition our efforts and goals of healing will fail. Early in my WOCN education at Emory University in Atlanta, Georgia, my instructor, Dorothy Doughty, compared healing a wound to the building of a house. In order to build a house you have to have bricks and mortar. If the supplies you need are not delivered you can't build a house. The same applies to the healing of a wound; you have to have adequate nutrition to fill in the defect. Throughout my many years of practice I still recall this pearl. Many times I have seen patients struggle to heal, and once we were successful with improving their nutrition the wound also healed. Good nutrition is essential for wound healing.

**Kimberly Smith BS, RN, CWON**

When treating a patient with a wound, remember to perform a comprehensive assessment. A comprehensive skin assessment should be performed with every home health care visit whether the patient is at risk for skin breakdown, already has skin breakdown, or has no breakdown at all. A comprehensive assessment should also take into consideration factors such as nutrition, pressure management, and comorbidities, as these can also affect wound healing. Focusing solely on the wound and not the entire patient will delay or even prevent wound healing. Remember; treat the whole patient not just the "hole" in the patient!

**Naomi Ward RN, BSN,CWS**

The use of compression in combination with topical advanced wound products is an important therapy needed to heal venous stasis ulcers. Compression applied from the base of the toes to base of the knee aids in edema control and contraction of the wound in patients with venous insufficiency. Use caution: Assess the patient's arterial status with use of Doppler studies and ankle brachial index (ABI) and assess color, temperature, and capillary refill of lower extremities prior to application of compression wraps. When using compression, include treatment of the surrounding skin. Stasis dermatitis is common and can be resolved with barrier cream or moisturizers in conjunction with compression wraps. Application tip: when applying layers of compression, always have the patient keep the foot in dorsiflexion, "toes to the nose," to prevent creasing of the layers at the upper foot and ankle area and preventing irritation or pressure. Once wounds are healed, patients with venous insufficiency must be treated with compression for life to prevent the re-occurrence of stasis ulcers. Compression is the gold standard.

**Kimberly Bare, RN, BSN, CWON, MBA**

The "Crusting technique" is often used when treating peristomal skin irritation but is also useful in managing peri-wound skin issues as well. This technique uses stoma powder and no-sting skin barrier wipes or sprays. The application process is easy to do and includes cleaning of the skin, allowing it to dry, sprinkling the powder on the irritated skin, and blotting with no-sting skin barrier wipe to seal the powder. May repeat up to three times if the skin is weeping.

Investigate the cause of the irritated skin such as wound/stoma in a crease or fold, allergic reaction, or excessive perspiration to establish appropriate plan of care to address and correct.

**Jerri Drain BSN, RN, CWOCN**

The use of abdominal binders for surgical wounds that have dehisced or are at risk of dehiscence is a simple intervention that can make a tremendous impact on wound healing. The use of a binder to reduce tension on the incision line is very helpful in preventing dehiscence, and using

abdominal binders for wounds that have already dehisced can be beneficial as well. An abdominal binder will help to reduce tension on the newly granulating tissue and approximate the wound edges together to reduce the amount of scar tissue formation necessary to fill the wound bed, leading to faster healing.

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Wound Care & Hyperbaric Medicine

# Four Easy Tips to Improve Your Clinic's Community Education Initiatives

From the Wound Care Education Partners blog, *Rx Pad*

By providing community education to potential patients you are, in the words of John Peters, incoming Executive Director of UHMS and Managing Partner of Wound Care Education Partners, "building awareness, and with that awareness we're able to impact healing outcomes for patients in our community."

By educating the medical community and patients about where to go to have their problem wounds treated, you will be saving your entire local healthcare network time and money in not having to triage and refer the patient through the system. At the same time you will be saving patients from days or weeks of frustration in having to advocate for themselves while trying to navigate a healthcare network that they don't understand.

## #1: Bring Awareness to the Local Medical Community of the Services You Offer

Get out from behind the desk and make personal visits to potential referral sources in your community. Getting out into the medical market, accessing physicians and their office staff, home healthcare organizations, DME's stores, pharmacists - even those at the major chain stores, i.e. CVS, Walgreens, etc,. and the like will elevate the awareness of the specialty services provided at your institution. More than 75% of all new referrals coming into wound care programs are directed from the formal medical community while the remainder of patients are self-referred. Evaluating how patients accessed your program is critical in your understanding and monitoring the health of your business/practice.

## #2: Hold a Free Community Education Series

Organize regularly scheduled free lectures for your community on topics of popular interest (e.g., diabetes, carbon monoxide poisoning). Ask your physicians and/or clinic practitioners to provide 45 minute educational lectures followed by a 15 minute question and answer session. Remind the lecturing faculty that the audience is comprised of current and potential patients, not fellow practitioners, and to be mindful of the terminology and phraseology used


in delivering the message. Hold the lectures at least once a month. In the beginning, you may need to hold the lectures at different times and on different days to see what results in the highest attendance (e.g., lunchtime on Wednesday versus at 5:30 p.m., or Saturday morning 9 a.m.). A key to remember is to be consistent with the series; don't just schedule a talk once and be done. You will be more effective in building awareness through consistent, regular messaging.

## #3: Create a Short Video or Commercial

In today's multi-media driven culture, it is critical to have visually appealing content that clearly explains the services you provide to a target audience in a minute or less. The first step in creating video content is to sit down with your marketing team (or strategic planning team) and thoughtfully identify your target audience (e.g., patients at high risk for necrotizing fasciitis), what services you want to promote to them (e.g., hyperbaric medicine), and what action you want them to take (e.g., calling your clinic for an evaluation/consultation).

As for promoting and distributing the video or commercial, you may want to consider posting it on your hospital or clinic webpage, promoting it via your social medial channels (such as Facebook or LinkedIn), or uploading it to YouTube. If you have resources in your budget, you may also want to consider contacting the local television stations to run your commercial.

## #4: Write an Article for a Local Newspaper, Magazine, or Blog

Newspapers, magazines, blogs and journals are always looking for good, high-quality, engaging content. In today's

marketplace, producing educational content (content marketing) is proving to be a very effective marketing approach. To further explain, are you familiar with the term "advertorial?" The term is a blend of the words "advertisement" and "editorial." Advertorials are widely used to effectively communicate a marketing message through storytelling. Advertorials promote a product or service by using a story that your target audience can relate to. For example, using patient testimonials (with proper consent from the patient) could effectively explain the benefits of hyperbaric medicine from a patient's perspective, in a way that potential patients can understand. If a reader feels like they are better informed after having read your article, you have taken the first step in building their trust. At the end of your article/story be sure to include a "call to action"; direct the reader to take the next step and contact your clinic for more information (i.e., for a consultation). Consult with your hospital or facility's outreach, education, or marketing department about creating educational content that is also entertaining and engaging.

### Take Action Now!

Ready to get started improving your facility's community education initiatives but don't have a marketing department? Wound Care Education Partners has teamed up with industry partner Best Publishing Company to offer multimedia and production services. We specialize in the diving, wound care, and hyperbaric medicine industries. Need a video or commercial created, flyers or marketing materials designed? Contact us today and let's get started!

*Do you have questions for the wound care experts, or want to continue your education and improve the health of your clinical practice? Follow the Wound Care Education Partners' Blog, Rx Pad today at: http://www.woundeducationpartners.com/rx-pad.html*

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Wound Care & Hyperbaric Medicine

# Letter to the Editor Regarding Wet-to-Dry Dressings

I read with interest the article that Dr. Strauss wrote in your journal (Wound Dressing Agents. *WCHM*. 2013; 4(1):41-71) about wound care. He made an interesting statement about wet-to-dry dressings being a forgotten modality and how it was appropriate for certain wounds.

The CMS Physician Quality Reporting System (PQRS) is a reporting program that uses a combination of incentive payments and payment adjustments to promote reporting of quality information by eligible professionals (EPs). The program provides an incentive payment to practices with EPs (identified on claims by their individual National Provider Identifier [NPI] and Tax Identification Number [TIN]). EPs satisfactorily report data on quality measures for covered Physician Fee Schedule (PFS) services furnished to Medicare Part B Fee-for-Service (FFS) beneficiaries. Beginning in 2015, the program also applies a payment adjustment to EPs who do not satisfactorily report data on quality measures for covered professional services.

PQRS item 246 - Chronic Wound Care: Use of Wet to Dry Dressings in patients with Chronic skin ulcer (Overuse measure) states:

> *A moist wound environment is essential to accelerate wound healing. Nevertheless, wet to dry gauze dressings are the most widely used primary dressing material in the United States and evidence suggests that they are used inappropriately. In a recent study examining wound care practices, the use of wet to dry dressings to maintain a moist wound environment ranged from 47.1% to 58.5% for diabetic and venous ulcers respectively. Wet to dry dressings* ***should not be utilized in the care of patients with chronic wounds as they may actually impede healing and are associated with an increased risk of infection, prolonged inflammation, and increased patient discomfort.***

CMS will start financially penalizing physicians who use saline wet to dry dressings as part of their PQRS Program beginning in 2015.

Physicians should be aware of all the PQRS measures that pertain to chronic wound care.

Sincerely,

Thomas M. Bozzuto, DO, FACEP, UHM/ABEM, FFACHM

Medical Director, Phoebe Wound Care and Hyperbaric Center

To read the original WCHM article, "Wound Dressing Agents," CLICK HERE.

# Author Response Regarding Wet-to-Dry Dressings

I appreciated Dr. Bozzuto's comments and feel that the readership of *WCHM* should be aware of this regulatory information. I am pleased to have the opportunity to respond to his comments with the following five points:

1. I deplore the term "wet-to-dry" dressing which is ambiguous in itself. First does this mean the wound base dries out between dressing changes? Certainly this would be counterproductive in all wounds except those with a thin crust or eschar (such as may occur over a "skinned" elbow or similar type wound where epithelialization is occurring under the cover, i.e. nature's BAND-AID®). Second, does "wet-to-dry" mean the contact layer with the wound is moist and then sufficient dressings are applied so the outer layer is dry? Regardless, maintaining a moist wound base is paramount to the contemporary management of wounds. Nowhere in the article do we recommend "wet-to-dry" dressings; the terminology we use is moist dressing. As we state in the article a healthy, healing wound base will remain moist between moist dressing changes and, if not so observed, switching to a gel or other wound dressing agent is indicated.

   **The Bottom Line:** The wound base remains moist with the moist dressing in almost all situations.

2. The definition of chronic wound needs elaboration. At one extreme the chronic wound is one that is not improving and requires something else be done such as addressing the major confounders to healing such as deep infection, deformity, and/or wound hypoxia/ischemia. At the other is a wound that is not improving after a 30-day period of "optimal" care. We subscribe to the former definition and are quick to initiate other interventions when improvement such as reduction in exudate, resolution of cellulitis, decrease in wound size, and development of granulation tissue are not observed. Invariably, one or more of the three confounders are the reason for this.

   **The Bottom Line:** When wounds do not improve, seek other causes. The reason is usually not the selection of the wound dressing agent, but rather a confounder to wound healing.

3. We typically initiate our wound dressing agent selection with moist (usually acetic acid or Dakin's solution) gauze dressings, especially if exudate, systemic signs of infection, initial post-operative bleeding, cavitation/tunneling, large size, and/or cellulitis are present. As the wound improves, we switch to other agents that require less intensive nursing care as is illustrated in Figure 8 (The Changing Roles of Wound Base Management and Wound Dressing Selection as the Wound Improves) of the referenced article.

   **The Bottom Line**: As the wound improves, the wound dressing selection should be changed to reflect economies, comfort, nursing time, and ease of application. If there is only one choice (e.g. with severe limitation of resources), the moist dressing (or if funding available, negative pressure wound therapy) use will suffice from initial presentation to almost complete healing and is totally independent of wound size.

4. This brings us to the question of economies. No dressing in terms of materials' cost alone is less expensive and more versatile than the moist dressing (see Table 9: Criteria for Evaluating Wound Dressing Agents with Specific Reference to Moisten Gauze of our article). With increasing reductions in resources by insurance companies and the Centers for Medicare/Medicaid Services (CMS), cost-effectiveness and cost-benefit considerations are essential. When the family member is taught the moist dressing change technique, there is no more cost-conscious use of resources. We have observed dramatic healing in long-standing (even weeks to months to heal) wounds with this family directed care with moist dressings—especially where other options because of costs were not available.

   **The Bottom Line:** With shrinking of funding and resources, increasing use of family members to provide wound care is a reality and the moist dressing fulfills this role well until the wound reaches a healthy enough state that a gel or ointment becomes appropriate for wound care.

5. In the "infinite" wisdom of the CMS, I take strong exceptions to many of their decisions regarding directives on diabetic foot ulcers (see article "Wagner Wound Grading System: What is it? What Are its Ramifications for Hyperbaric Medicine?" *WCHM.* 2012; 3(4):38-44) and feel their decision that moist dressings are an impediment to wound healing with increased risk of infection, prolonged inflammation, and increased patient discomfort are not supported by the reality of the situations.

   **The Bottom Line:** The moist dressing has a role, especially with the family applying the dressings, in chronic wound care and may become the only reasonable alternative with severely constrained resources and lack of availability of professional nursing care.

In summary, as in the case of Blount's classic article "Don't Throw Away the Cane" (*J Bone Jt Surg.* 1956; 38(3):695-708), the same admonition is applicable to the moist dressing in wound care. Nonetheless, Dr. Bozzuto's contribution to our article is appreciated and should there be a rewrite of the article, at the least, his information will be incorporated into the text.

Finally, the subject of Dr. Bozzuto's letter and my responses would be a thought provoking topic for a point, counterpoint presentation at a national hyperbaric medicine or wound care conference.

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## Postscript From Dr. Bozzuto

I agree with Dr. Strauss concerning the effectiveness of moist dressings (as a matter of fact, I use Dakins on a regular basis). It is important for physicians to know the difference between moist dressings and wet-to-dry dressings, which will trigger an audit with CMS. Dr. Strauss does an excellent job of pointing out the difference in his reply to my initial letter.

*Do you have a question or comment on an article published in WCHM?*
*Submit your questions to info@bestpub.com.*

# Test your Knowledge of Wound Care

By Dr. Jayesh Shah

1. Ultrasound therapy for wounds appears to . . .

A. modulate fibroblast activity
B. modulate inflammatory response
C. modulate cytokine production
D. all of the above

2. Ultrasound therapy helps with wound healing by all of the above except:

A. Formation of stronger granulation tissue
B. Correction of hypoxia
C. Promoting vasodilation and angiogenesis
D. Alteration in bacterial load during treatment of chronic wounds

3. Following statements about ultrasound therapy are true except:

A. Low frequency ultrasound has the potential of delivering energy with less attenuation than high frequency ultrasound.
B. Low frequency ultrasound treatment may be useful in the management of venous leg ulcers.
C. RCTs of low frequency, noncontact ultrasound treatment suggest an effect on neuropathic, superficial diabetic foot ulcers.
D. High frequency ultrasound therapy has much better evidence for wound healing than low frequency ultrasound therapy.

4. Following statements about ultrasound debriders are true except:

A. Ultrasound debriders can be useful when non-painful, selective debridement is required particularly in a nonsurgical setting.
B. The clinical debridement of necrotic and infected tissue with nonsurgical low-frequency, contact ultrasound devices is selective and superior to debridement with a scalpel.
C. Debridement with contact, low frequency devices allows a good wound bed preparation.
D. Noncontact ultrasound can be useful as an adjunct to sharp debridement to improve the characteristics of the granulation tissue and allow a more rapid wound closure or delayed primary closure.

5. TIME principle of wound bed preparation includes:

A. Tissue debridement
B. Infection
C. Moisture
D. Edge effect
E. All of the above

See the next page for answers.

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# ANSWERS to Test your Knowledge of Wound Care

By Dr. Jayesh Shah

## Answers:

**1.d, 2.b, 3.d, 4.b, 5.e**

## Question 1. Answer (d)

Ultrasound appears to modulate fibroblast activity,[1,2] the inflammatory response,[1,2] and cytokine production[3]

## Question 2. Answer (b)

Ultrasound may enhance the formation of a stronger granulation tissue.[4] Low frequency ultrasound treatment may be helpful in the clinical management of ischemic wounds by promoting vasodilation and angiogenesis.[5,6] Ultrasound may be useful in the alteration of the bacterial load during the treatment of chronic wounds.[7]

## Question 3. Answer (d)

Ultrasound is able to exert a biologic effect by low or high frequency since absorption, which is frequency dependent, is the major cause of intensity attenuation. By increasing the frequency, the wave length shortens and absorption increases. Low frequency ultrasound has the potential of delivering energy with less attenuation than high- frequency ultrasound. Low frequency ultrasound shows a deeper tissue penetration that high frequency Ultrasound. In recent years low frequency ultrasound devices have been increasing used for wound healing, serving as debriders or as healing promoters.[8]

## Question 4. Answer (b)

Ultrasound debriders can be useful when non-painful, selective debridement is required, particularly in a nonsurgical setting. Noncontact ultrasound can be a useful adjunct to sharp debridement to improve the characteristics of the granulation tissue and allow a more rapid wound closure or delayed primary closure. The clinical debridement of necrotic and infected tissue with nonsurgical low-frequency, contact ultrasound devices is selective and as effective as debridement with a scalpel.[9,10]

## Question 5. Answer (e)

TIME Principle of wound bed preparation includes: Tissue debridement, infection management, moisture control, and edge effect.[11]

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# Press Release

## Systagenix Launches Superabsorbent Dressing in Germany and the UK

**For Immediate Release**

**GATWICK,** 7 May, 2014 – Systagenix announced today the launch of BIOSORB™, a hydro-active dressing containing superabsorbent fluid-retaining polymers. BIOSORB™ actively manages wound exudate in chronic wounds, thus reducing the risk of maceration.[1,2]

Superabsorbent dressings are one of the fastest growing categories in the advanced wound care market providing a solution for fluid management in moderate to highly exuding wounds.

Wound exudate is produced as a normal part of the healing process and at varying levels throughout. An optimal wound healing environment is moist but not wet. In acute wounds the volume of exudate produced is normally manageable, whereas in chronic wounds the exudate levels may be less predictable.[3]

"Designed to efficiently manage high volumes of exudate in a variety of wound types, BIOSORB™ absorbs and retains large quantities of wound exudate which can be detrimental to the healing process if left unmanaged," said Marc Blamire, Systagenix Global Category Director. "Thus, BIOSORB™ may allow for an extended wear time, leading to the potential of both direct and indirect cost savings for clinicians."

BIOSORB™ is currently available in Germany and the UK.

**About Systagenix**

Systagenix is a global advanced wound care company headquartered in the UK. Systagenix recently joined KCI and LifeCell to form one globally diversified wound care, biologics and regenerative medicine company committed to providing healthcare professionals with transformational healing solutions that improve patient lives. For more information about combined company or Systagenix advanced wound dressings please visit www.systagenix.com.

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BONUS CONTENT

Photography © Jay Easterbrook/Divetech Inner Space

# The RF3 Proceedings are Now Available!

The Rebreather Forum 3 Proceedings have been published in both print and electronic format following the safety symposium held from 18–20 May 2012 at the Caribe Royale Hotel in Orlando, Florida. Two years in the planning and two years in the writing, the 324-page rebreather publication showcases state-of-the-art and science of rebreather diving through the experience and knowledge of some of the world's leading specialists in education, operations, physiology, medicine and safety. This meeting followed the Rebreather Forum 2 conference which was held in 1996.

Photography © Rick Melvin

The goal of Rebreather Forum 3 was to positively impact the safety of recreational, professional, scientific, media and military rebreather divers by sharing latest developments and best practices. Educational sessions took place in seminars that discussed history and evolution, medicine and physiology, business and operations, incidents and their investigation, design and testing, and training and operations. Safety was the key theme and participants were informed about the most common causes of rebreather incidents and fatalities in hopes of reducing their future occurrence. To round out the meeting, participants were able to gain hands-on experience through various pool sessions.

The 11-member, Rebreather Forum organizing committee comprised Dr. Nicholas Bird (DAN), Mark Caney (PADI), Dr. Petar Denoble (DAN), Michael Lang (American Academy of Underwater Sciences), Rosemary E. Lunn (The Underwater Marketing Company), Christian McDonald (American Academy of Underwater Sciences), Dan Orr (DAN), Dr. Neal W. Pollock (DAN), Dr. Drew Richardson (PADI), Karl Shreeves (PADI), and Dr. Richard Vann (DAN).

"We planned this Forum to introduce divers without rebreather experience to the equipment as well as to assist experienced rebreather divers in refining their skills. This was the ideal format to share ideas and listen to some of the world's most respected experts on rebreather diving," stated Dr. Richard Vann, former Vice President of Research, Divers Alert Network.

Publishing the Rebreather Forum 3 proceedings is a significant accomplishment by the AAUS, DAN and PADI. All three partners are very pleased to share this invaluable resource with the diving community, in order to help make diving safer. It is hoped that the knowledge gained will reduce incidents and fatalities among rebreather divers.

PDF copies of the complete RF3 Proceedings can be downloaded immediately at no-cost from the DAN website. Print copies will be available in the near-future through a print-on-demand service. More detailed information can be found at the Rebreather Forum 3 website; www.rf30.org.

Many of the RF3 talks were recorded and put online. These are an excellent free resource tool for divers, instructors, or anyone involved with rebreathers. You can access these lectures by going to the RF3 website; www.rf30.org. Then click "presentations" before choosing the specific session you wish hear.

**The Editors of the RF3 Proceedings (from l to r); Dr Neal W. Pollock, DAN Research Director | Dr Richard D Vann, Professor Emeritus Duke Anesthesiology | Dr Petar J Denoble, Vice President DAN Research. Photography © Rick Melvin.**

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