Wound Care & Hyperbaric Medicine magazine

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Spring 2015

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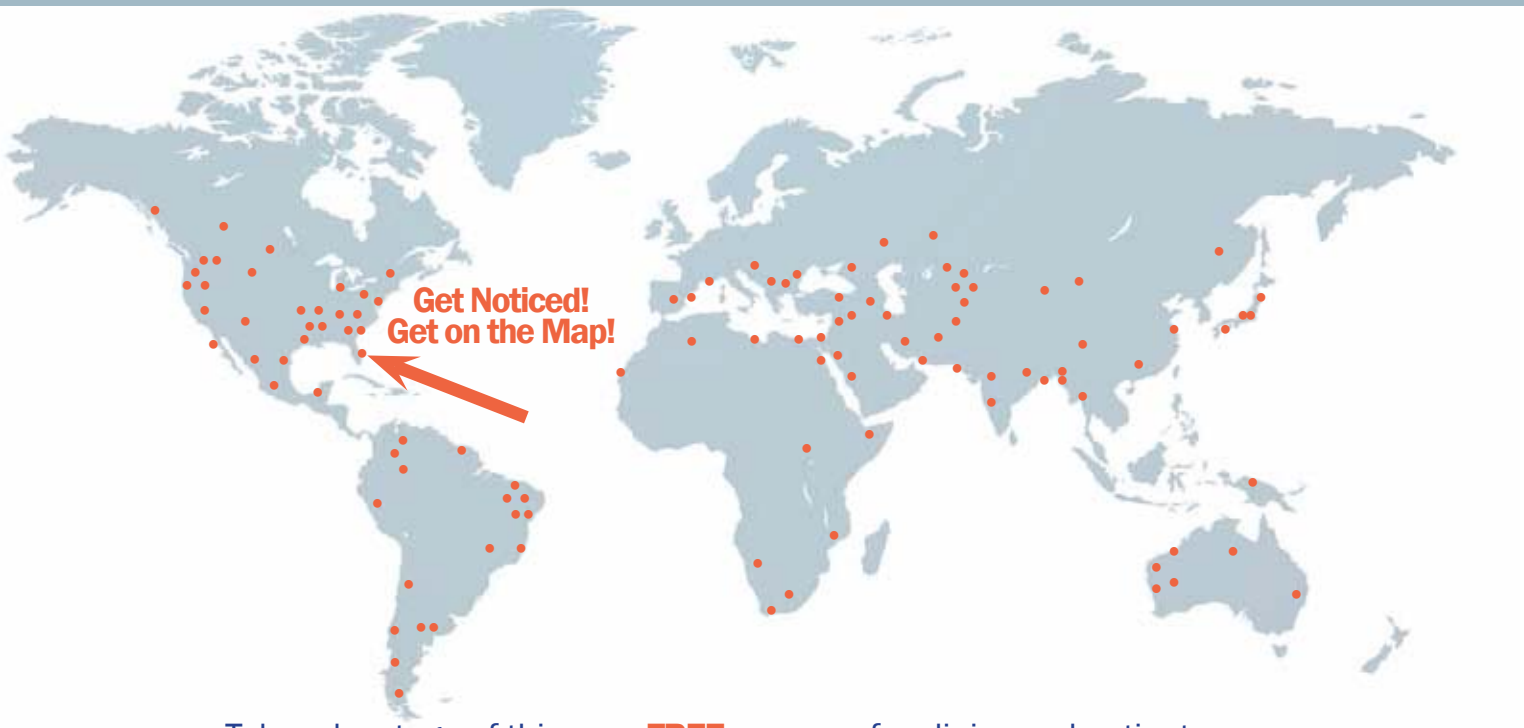

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Note from the Editor

In this issue of WCHM, which marks our sixth year, we continue to search out practical applied topics and wound care, diving, and hyperbaric medicine. First, Gretchen Dixon continues coverage of the changing requirements for billing and coding. CMS will implement a prior authorization model for non-emergent hyperbaric oxygen therapy in Illinois, Michigan, and New Jersey to ensure the services are in compliance with Medicare rules. For more information, start reading on page 7.

Our prolific authors Drs. Strauss, Le, and Miller continue their exposition on diving stresses with near-drowning and drowning events on page 17. Drs. Strauss and Miller and also contribute an article on the considerations of oxygen in wound healing on page 47.

We welcome Drs. Nasole, Paoli, Bosco, and Camporesi, and their article on sinus and ear disorders as effects of hyperbaric oxygen therapy. See their article on page 35.

Darren Mazza continues his coverage on safety issues for CHTs with his article on the importance of pre-treatment patient education on page 45.

Also included is this issue's Clinic In Focus, which features the Memorial Hospital Wound Clinic/Hyperbaric Medicine Department in Colorado Springs, Colorado. If you are a part of an exceptional hyperbaric or wound care center, contact us today to be our next featured clinic!

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 Map of Wound Care and HBO Centers.

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, articles, industry information, press releases, and updates.

Sincerely,

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

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Wound Care & Hyperbaric Medicine magazine

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Your Double-Headed Dragon Requires Attention

Wrangling Compliance and Documentation

Gretchen Dixon, MBA, CCS, CPCO

Working with compliance and documentation—your double-headed dragon—can call to mind the expectation of a challenge; however, getting both of these heads to work together is vital to each provider's business survival. Often discovered through revenue cycle audit results is a valley of missing information necessary to bridge these components and ensure our patient care services are accurately reported. This valley affects the accuracy of your patient's acuity level and intensity of services with complexity of care provided. This article will discuss the activities you need to embrace and the specific documentation details required for accurate ICD-10 code assignments related to wounds.

ICD-10-CM implementation is less than 9 months away—what have you done during last year's reprieve in preparation? Many provider practices as well as small- to mid-size hospitals continue to squander this time. The compliance section of this article is to be used as an evaluation tool to determine your current status and readiness towards implementing ICD-10-CM. An additional section will provide key clinical documentation verbiage to assist in providing the specific details necessary to ensure selected ICD-9-CM or ICD-10-CM codes accurately reflect the patient's level of acuity and intensity of service with complexity of care provided.

Topics in this article affecting wound care services include

- Compliance and documentation
- Changes with Modifier 59 when reporting evaluation and management services
- CMS Model for pre-authorization of non-emergent hyperbaric oxygen therapy
- New HBO HCPCS Level II code

Begin by Understanding Compliance

To simplify the definition of COMPLIANCE™, this mnemonic device uses each letter to identify the clinical documentation improvement activities necessary to meet today's and tomorrow's regulatory guidelines, which will involve providers and payers sharing service accountability through the Affordable Care Act.

Evaluate your clinical documentation activities. How does COMPLIANCE™ fit into your everyday patient care documentation activities?

C = CHANGE begins by recognizing old documentation habits and building new habits through understanding the requirements of detailing information to support the level of acuity and intensity of service with complexity of care.

O = OPTIMIZE your clinical documentation details to provide an accurate, concise, and complete story of the patient's encounter.

M = MENTOR your peers by encouraging their clinical documentation improvement (CDI).

P = PRACTICE daily adding details to your clinical documentation.

L = LEARN with enthusiasm how to incrementally add details in a patient's medical record. Start with the most common diagnosis in your office and learn the specific details ICD-10-CM expects. This endeavor should improve your current ICD-9-CM code selection and prepare you for the code change on October 1, 2015.

I = INSTILL a positive outlook towards changes along with the long-term benefits to your practice. Long-term benefits could be as simple as knowing you complied with federal, state, and local regulations; reducing the number of held claims, which require timely re-work; or experiencing a decrease in AR days as well as an increase in timely reimbursement.

A = ACTION includes addressing weak areas in your clinical documentation. Enlist the assistance of your coders or internal auditors who have reviewed your documentation. Listen to their suggestions and then help your peers to understand the value of clinical documentation change.

N = NEVER ASSUME health care professionals involved with your patients' medical records will be able to interpret your documentation for patient services if the information is not accurate, clear, concise, and complete.

C = COMPLY with clinical documentation improvement to better represent the acuity of patient care and intensity of services with complexity of care. Without detailed clinical information available, often the acuity of care and intensity of services with complexity of care are not supported. This data can affect your patient care profile monitored by third-party payers and external agencies as well as patients.

E = EDUCATE yourself and peers by seeking opportunities to learn new clinical documentation elements. It can be as simple as how you select verbiage for a sentence. Changing verbiage or re-aligning thoughts often help others to read and accurately interpret your information.

Compliance Summary

Evaluating your current habits, staff skills, and level of ICD-10-CM understanding; preparing with education, both formal and informal (on-the job); and interacting with payers and systems for their ICD-10-CM readiness will all be vital to a successful transition. Mitigating discovered weaknesses in your processes now will help to reduce the last minute scurry and re-work load as we approach October 1, 2015. Will there continually be opportunities before and after October 1, 2015? Absolutely. However, by working through affected staff education, answering questions objectively, ensuring processes are current and technology is up to date for the implementation of ICD-10-CM, you will be in a better position for the change, thus managing your two-headed dragon. Your bridge of preparedness will be stronger for this dynamic change.

Proactive Documentation Direction

Our focus is to provide specific information as it relates to describing the specific wound characteristics that need to be documented in detail in the medical record. It is the details that will direct either the provider or coder to select specific ICD-10-CM codes to accurately support services provided.

NOW: Currently, providers are paid for services using CPT codes with a limited focus on the diagnosis, which most likely is represented by an unspecified diagnostic ICD-9-CM code. Payers reimburse by service provided as long as the diagnostic code is reasonable or within the diagnosis coding category. Your data is being messaged now by third party payers.

THEN: Starting October 1, 2015 under the Affordable Care Act (ACA) there is a transition that needs to be understood and engaged. Providers and payers will be held equally accountable for accurately providing, reporting, and reimbursing healthcare services based on clinical documentation. The ACA with HIPAA continues reforming ambulatory patient care to include provider risk sharing. **This involves a transparency and accountability movement toward value-based purchasing (providers managing the patient care) with a focus on improving health conditions**. How you treat your patient's medical condition(s) and results/outcomes will be more closely monitored by your payers.

NOTE: Payers can remove you from their contracts if the clinical information reported with ICD-10-CM codes and CPT codes do not align to support the level of acuity and intensity of services with complexity of care based on your data or the data of your peers for the same specialty.

Transition

Part 1: After the implementation of ICD-10, providers will no longer be paid just for the service they provided; there must be a clear reason (diagnosis) stating why the service was provided. Presently, part one of the equation looks like this: **S=P**

Service = **P**ayment

(as long as there is a diagnosis code in the broad category to support why the service was performed)

Part 2: There must be a level of certainty with each diagnosis, which means specificity in the details of the clinical documentation. The specific diagnosis provides a more accurate selection of ICD-9-CM/ICD-10-CM codes, thus avoiding the selection of unspecified codes. We have always been paid for unspecified ICD-9-CM codes, so why is it necessary to change? On October 1, 2015 the equation will become: **D + S = P**

Diagnosis code(s) [Why] + **S**ervice [What was provided] = **P**ayment

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Data Collection after October 1, 2015

Tracking of Unspecified Code Usage

Why is it suddenly necessary to reduce the use of unspecified diagnosis codes that have commonly been used in the past? Starting on the ICD-10-CM go-live date, the Centers for Medicare & Medicaid Services (CMS) will be gathering data on the use of unspecified diagnosis codes. It is anticipated the data collection will be conducted for up to two years and may result in changes to Relative Value Unit calculations, which may affect your reimbursement of services.

Also, it is anticipated there will be a reduction in payments for low level of acuity and low intensity of services with low level of complexity of care related to the diagnosis and services provided. **While there are times when an unspecified diagnosis code may be appropriate to report conditions of uncertainty, using unspecified diagnosis codes will also be viewed as a reflection of low acuity and low intensity of services with low complexity of care.** The above chevron provides a visual effect.

Third party payers have already begun to monitor providers' use of unspecified codes. As they build their data banks, each provider's frequency in reporting unspecified codes may cause additional claim and documentation scrutiny. Identifying unspecified code usage could have a negative outcome for providers—this includes audits of clinical documentation to determine if there is specific medical necessity information for the services provided.

Profiling will escalate with like peers comparing this data of unspecified codes routinely reported on claims. Continued routine use of unspecified codes could result in the provider being removed from certain health plans with a possible negative financial outcome on a provider's business.

When to Use an Unspecified Code[1]

The use of unspecified codes is tracked in most data collection systems and identifies these codes by specialty, resulting in a list of the top ten reported unspecified codes. However, it is a known fact there are times when an unspecified code is acceptable or even necessary to report signs or symptoms and is the best choice to accurately reflect what is known about the patient's medical condition at the time of the visit. Coding guidelines instruct coding to the level of certainty known for the visit; if a definitive diagnosis has not been established by the time the patient visit has concluded, it is then appropriate to select an unspecified code. Every code selected by a provider or coder must be supported by clinical documentation, as it would be inappropriate to select codes that are not supported by medical record documentation or that are medically unnecessary, such as for diagnostic testing.

Clinical Documentation Improvement

The following example uses the documentation of a common wound care patient diagnosis coded in ICD-9-CM and then in ICD-10-CM. The reader should note the complexity of the information documented in the patient's medical record; this is absolutely necessary to select the most accurate diagnosis code that reflects the level of acuity and intensity of services and complexity of care.

Original documentation: This is an established patient with a chronic ulceration of the calf (which calf). Ulcer measurements noted. Necrotic or devitalized tissue is estimated to be present in 75% of the ulcer bed with 25% beefy granulation in the wound bed. Debridement performed.

As you can see, the diagnosis codes have different descriptions based on the limited information in the provider's documentation. ICD-9-CM coding definitions are less descriptive than the ICD-10-CM codes, and the detail within the new code descriptions will more accurately reflect the level of acuity thus supporting the level of intensity for services and complexity of care.

ICD-9-CM	ICD-10-CM
707.12 – Ulcer of calf	**L97.209** – Non-pressure chronic ulcer of **unspecified** calf with **unspecified severity**

Since ulcers involve the integumentary system the level of documentation needs to include laterality (right, left or bilateral), ulcer characteristics, and level of severity. Ulcers are divided into two categories in ICD-10-CM: non-pressure chronic ulcer and pressure ulcer. Codes for non-pressure chronic ulcers of the lower limb include those documented as:

- Chronic ulcer of skin
- Non-healing ulcer of skin
- Non-healing infected sinus of skin
- Trophic ulcer

The following illustration provides reference information easily located in any vendor's ICD-10-CM mappings manual. These manuals create a crosswalk between ICD-9-CM diagnostic codes and all of the relative ICD-10-CM codes, which provides options for a diagnosis code to be selected based on the details within the provider's documented information. Using this type of manual will allow you to review those codes frequently used by your specialty and begin to add the level of details in the documentation to avoid an unspecified code selection. This example of code selection in ICD-10-CM demonstrates the need to document certain elements as related to an ulcer of the calf to avoid an unspecified ulcer code selection.

- State status: acute versus chronic (in this instance chronic will be used)
- Type of ulcer: pressure versus non-pressure (in this instance non-pressure will be used)
- Anatomical location: calf
- Laterality: right, left, bilateral
- Severity of ulcer must be documented
    - Limited to breakdown of skin or
    - With fat layer exposed or
    - With necrosis of muscle or
    - With necrosis of bone or
    - With unspecified severity (this should never be selected for a diagnosis in a wound care department)

Improved documentation: This is an established patient with a chronic ulceration of the left lateral calf. This ulcer was caused by an injury during a vacation 3 months ago and would not heal while under treatment from the primary physician. The patient has been in treatment by this clinic for 3 weeks and the ulcer measurements have made only minimal improvement for healing from last visit. Agree with clinical wound assessment measurements. Necrotic devitalized tissue of the subcutaneous tissue remains and is estimated to be present in 75% of the ulcer bed with 25% pinkish granulation in the wound bed. Debridement performed.

The following table lays out the amount of information required to code the diagnosis of ulcer of calf in ICD-10-CM accurately portraying the patient's medical condition which supports the level of acuity with intensity of services and complexity of care. The color coding refers to where the Improved Documentation scenario supports the appropriate ICD-10-CM code.

ICD-9-CM	ICD-10-CM
707.12 – Ulcer of calf	L97.201 Non-pressure chronic ulcer of unspecified calf limited to breakdown of skin
	L97.202 Non-pressure chronic ulcer of unspecified calf with fat layer exposed
	L97.203 Non-pressure chronic ulcer of unspecified calf with necrosis of muscle
	L97.204 Non-pressure chronic ulcer of unspecified calf with necrosis of bone
	L97.209 Non-pressure chronic ulcer of unspecified calf with unspecified severity (***Should not be selected as a diagnosis in a wound care department***)
	L97.211 Non-pressure chronic ulcer of Right calf limited to breakdown of skin
	L97.212 Non-pressure chronic ulcer of right calf with fat layer exposed
	L97.213 Non-pressure chronic ulcer of right calf with necrosis of muscle
	L97.214 Non-pressure chronic ulcer of right calf with necrosis of bone
	L97.219 Non-pressure chronic ulcer of right calf with unspecified severity (***Should not be selected as a diagnosis in a wound care department***)
	L97.221 Non-pressure chronic ulcer of left calf with limited breakdown of skin
	L97.222 Non-pressure chronic ulcer of left calf with fat layer exposed
	L97.223 Non-pressure chronic ulcer of left calf with necrosis of muscle
	L97.224 Non-pressure chronic ulcer of left calf with necrosis of bone
	L97.229 Non-pressure chronic ulcer of left calf with unspecified severity (***Should not be selected as a diagnosis in a wound care department***)

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Based on the clinical documentation in this example, ICD-10-CM code **L97.222 Non-pressure chronic ulcer of left calf with fat layer exposed** should be reported to accurately reflect the level of patient acuity with intensity of services and complexity of care.

Documenting Underlying Conditions

Any associated underlying medical condition such as atherosclerosis, gangrene, chronic venous hypertension, varicose ulcer, and post-phlebitic or post-thrombotic syndromes affecting the lower extremities requires additional details. In ICD-10-CM many code descriptions may now combine medical conditions. The following example shows the specific information needed to accurately identify an ICD-10-CM diagnosis code for atherosclerosis. This example of code selection in ICD-10-CM demonstrates the need to document additional specific elements as related to the underlying condition.

- Anatomical location of ulcer—include the terms lateral, medial, inferior, superior as needed
  - Thigh
  - Calf
  - Ankle
  - Heel and mid-foot includes the plantar surface of the foot
  - Other part of foot includes toes
  - Other part of lower leg
  - Unspecified site **(should not be selected as a diagnosis)**
- Laterality: right, left, bilateral or unspecified (should not be selected)
- Atherosclerosis of native arteries of lower extremity or
  - Atherosclerosis of autologous vein bypass graft(s) of extremity or
  - Atherosclerosis of non-autologous biological bypass graft(s) or
  - Atherosclerosis of non-biological bypass graft(s) or
  - Atherosclerosis of other type of bypass graft(s)
  - Atherosclerosis of other type of bypass graft(s) of extremities with gangrene
- An additional ICD-10-CM code is required to describe the severity of the ulcer tissue per coding guidelines which must be supported with documentation to include one of the below:
  - Limited to breakdown of skin or
  - With fat layer exposed or
  - With necrosis of muscle or
  - With necrosis of bone or
  - With unspecified severity **(should not be selected as a diagnosis)**

Improved documentation with underlying condition: This is an established patient with a chronic ulceration of the left lateral calf due to atherosclerosis of autologous vein bypass (lower extremities) for over 1 year and would not heal while under treatment from several provider specialists, thus supporting the referral to the wound care department. The patient has been in treatment by this clinic for 3 weeks and the ulcer measurements have only made minimal improvement for healing from last visit. Agree with clinical wound assessment measurements. Necrotic devitalized tissue of the subcutaneous tissue remains and is estimated to be present in 85% of the ulcer bed with 15% some light pinkish granulation in the wound bed. Debridement performed.

ICD-9-CM	ICD-10-CM*
707.12 – Ulcer of calf	I70.232 Atherosclerosis of native arteries of right leg with ulceration of calf
	I70.242 Atherosclerosis of native arteries of left leg with ulceration of calf
	I70.332 Atherosclerosis of unspecified type of bypass graft(s)native arteries of right leg with ulceration of calf
	I70.342 Atherosclerosis of unspecified type of bypass graft(s)native arteries of left leg with ulceration of calf
	I70.432 Atherosclerosis of autologous vein bypass graft(s) type of right leg with ulceration of calf
	I70.442 Atherosclerosis of autologous vein bypass graft(s) type of left leg with ulceration of calf
	I70.532 Atherosclerosis of biological non-autologous vein bypass graft(s) type of right leg with ulceration of calf
	I70.542 Atherosclerosis of biological non-autologous vein bypass graft(s) type of left leg with ulceration of calf
	I70.632 Atherosclerosis of non- biological bypass graft(s) type of right leg with ulceration of calf
	I70.642 Atherosclerosis of non- biological bypass graft(s) type of left leg with ulceration of calf
	I70.732 Atherosclerosis of other type of bypass graft(s) of right leg with ulceration of calf
	I70.742 Atherosclerosis of other type of bypass graft(s) of left leg with ulceration of calf

*Each of these codes requires an additional code to identify the severity of the ulcer)

These codes require an additional ICD-10-CM code to complete an accurate picture of the ulcer's level of severity and intensity of service from the L97–L97.49 category of ICD-10-CM diagnostic codes. Without the L97 codes, the diagnosis code would not be complete, causing a possible claim denial or hold for further information. The necessary supplemental codes are noted below.

ICD-10-CM codes identifying ulcer severity
L97.201 Non-pressure chronic ulcer of unspecified calf limited to skin breakdown
L97.202 Non-pressure chronic ulcer of unspecified calf with fat layer exposed
L97.203 Non-pressure chronic ulcer of unspecified calf with necrosis of muscle
L97.204 Non-pressure chronic ulcer of unspecified calf with necrosis of bone
L97.209 Non-pressure chronic ulcer of unspecified calf with unspecified severity*
Laterality + Severity Additional Detail for Right calf ulcer
L97.211 Non-pressure chronic ulcer of right calf limited to skin breakdown
L97.212 Non-pressure chronic ulcer of right calf with fat layer exposed
L97.213 Non-pressure chronic ulcer of right calf with necrosis of muscle
L97.214 Non-pressure chronic ulcer of right calf with necrosis of bone
L97.219 Non-pressure chronic ulcer of right calf with unspecified severity*
Laterality + Severity Additional Detail for Left calf ulcer
L97.221 Non-pressure chronic ulcer of left calf limited to skin breakdown
L97.222 Non-pressure chronic ulcer of left calf with fat layer exposed
L97.223 Non-pressure chronic ulcer of left calf with necrosis of muscle
L97.224 Non-pressure chronic ulcer of left calf with necrosis of bone
L97.229 Non-pressure chronic ulcer of left calf with unspecified severity*

*Unspecified codes should not be selected in a wound care clinic

Based on the clinical documentation in this example, ICD-10-CM codes **I70.442 Atherosclerosis of autologous vein bypass graft(s) type of left leg with ulceration of calf** and **L97.222 Non-pressure chronic ulcer of left calf with fat layer exposed** should be reported to accurately reflect the level of patient acuity with intensity of services and complexity of care.

Modifier 59 Morphing Into Subsets

Before closing, HCPCS modifier 59 has been used to tell the payer a service is separate and distinct from the reason for the encounter. As of August 15, 2014, CMS Transmittal 1422 has redefined the application of modifier 59 into four subsets to further define the meaning. CMS felt modifier 59 has been overused, abused, and incorrectly applied, therefore necessitating the change. Part of the issue with modifier 59 is the broadness of its definition to cover many different scenarios. CMS hopes that by redefining modifier 59 with these four subsets more precise coding options will be available to define specific encounter activity and thus reduce errors associated with modifier 59 overpayments, as well as provide a better understanding of when and why providers use this modifier.

How will this change affect your practice? Your two-headed dragon, documentation and compliance, continues to be affected by this change in the healthcare business. CMS will continue to have the OIG (Office of Inspector General) review the use of modifier 59 over the next several years. Therefore, we need to stay abreast of it, as daunting as it sometimes seems. Within the Transmittal, directions were provided for Medicare Administrative Contracts to review data regarding the application of these modifiers on claims. The following is taken from Transmittal 1422 and defines the new HCPCS modifiers.

XE—Separate encounter, a service that is distinct because it occurred during a separate encounter

XS—Separate structure, a service that is distinct because it was performed on a separate organ or structure

XP—Separate practitioner, a service that is distinct because it was performed by a different practitioner

XU—Unusual non-overlapping service, the use of a service that is distinct because it does not overlap usual components of the main service.

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Prior Authorization of Non-Emergent Hyperbaric Oxygen (HBO) Therapy

Note: the following information involves two components: physician documentation requirements and facility prior authorization of **Medicare beneficiaries** and specific billing processes.

On May 5, 2014, CMS issued a press release titled "Fact Sheets: Prior Authorization of Non-Emergent Hyperbaric Oxygen (HBO) Therapy." This document appeared low on the radar but set the stage for the implementation of demonstration programs regarding prior authorization for non-emergent HBO in the states of Illinois, Michigan, and New Jersey. This is a test to help ensure HBO services are provided in compliance with Medicare coverage, documentation, coding, and payment rules prior to the anticipated services to be performed, specifically HBO treatments.

History For this Decision

CMS is concerned about patients receiving non-medically necessary non-emergent HBO therapy in the identified three states due to their high utilization of HBO therapy and improper payment rates for the services. This falls under the Social Security Act, Section 1115A, allowing that the Secretary has the authority to test innovative payment and service delivery processes to reduce program expenditures while preserving quality of care to beneficiaries/patients.

No new clinical documentation criteria will be required. **The objective is to ensure the clinical documentation is sufficiently detailed to support the decision for the HBO treatment and the relevant coverage and coding requirements are met prior to performing HBO treatment and submitting claim for reimbursement.**

Prior Authorization Process

This process of prior authorization will allow relevant clinical documentation to be submitted for review prior to providing HBO services. CMS or its contractors (your Medicare Administrative Contractor) will review the request and provide a yes or no decision. Payment will be made as long as there is an affirmative prior authorization decision and all other requirements have been met. *Without the affirmative prior authorization decision the submitted claim will be denied.*

It is recommended the reader obtain a copy of all related articles noted in the reference section of this article.

Planning ahead for beginning a patient's HBO therapy will require understanding the decision timeline, which is divided based on whether the request is an initial or subsequent.

- Initial request: this decision is to be postmarked within 10 business days (2 weeks)
- Subsequent requests: this type of request will be processed within 20 business days (4 weeks)
- Provisional affirmative: decision may affirm up to 40 HBO treatments in a year

Six Common Conditions Covered for HBO

The following six conditions are most commonly diagnosed in wound care settings and are arranged in order of frequency.

1. Diabetic wounds of the lower extremities in patients who meet the following three criteria
   a. Patient has type I or Type II diabetes with a lower extremity wound that is due to diabetes
      i. Coding: The physician must document there is a casual relationship between the wound and diabetes by using the term "due to"[2]
      i. Without the casual relationship noted, the two conditions are coded separately[3]
   b. Patient has a wound classified as a Wagner grade III or higher
   c. Patient has failed an adequate course of wound therapy as defined in the NCD (reference noted below).
2. Chronic refractory osteomyelitis, unresponsive to conventional and surgical management
3. Osteoradionecrosis as an adjunct to conventional treatment
4. Soft tissue radionecrosis as an adjunct to conventional treatment
5. Preparation and preservation of compromised skin grafts (not for primary management of wounds)
6. Actinomycrosis, only as an adjunct to conventional therapy when the disease process if refractory to antibiotics and surgical treatment

"...in the December 10 presentation there was a change in the HBO HCPCS code to G0277 for per 30-minute intervals of treatment."

What is the Criteria for Conventional Treatment?

For HBO therapy to be covered as an adjunctive therapy, certain minimum criteria must be clear and detailed in the clinical documentation as follows:

- Failure to respond to standard wound care occurs when there are no measurable signs of healing for at least 30 consecutive days or
- There are no measurable signs of healing for at least 30 days of treatment with standard wound therapy
- HBO must be used in addition to standard wound care
- HBO therapy is covered as an adjunctive therapy only after there are no measurable signs of health
- Wounds must be evaluated at least every 30 days during the HBO therapy program
- Continuing HBO therapy is not covered if there is a lack of measurable signs of healing noted within any 30 day period of treatment.

It would be most prudent to review your understanding of what constitutes conventional treatment and the specific documentation required to meet the criteria.

Beginning Timeline Involves Two Dates

March 1, 2015 is when your Medicare Administrative Contractor for the three listed states will begin accepting prior authorization requests for one of the 6 conditions for HBO treatment to begin on or after April 13, 2015

- All HBO treatments for one of the six conditions with a date of service on or after April 13, 2015 must have completed the prior authorization process or the claims will be stopped for prepayment review
- Model data gathering will be for 3 years with annual updates of findings.
- Anticipate further expansions involving other states with high volume of HBO therapy and high costs.

New HBO HCPCS Code Starting January 1, 2015

On November 4 and December 10, 2014, CMS conducted an Open Door Forum to inform the wound care industry of the change for prior authorization of HBO in the three states. CMS documented in the November 4 presentation that the HCPCS code C1300 would continue. **However, in the December 10 presentation there was a change in the HBO HCPCS code to G0277 for per 30-minute intervals of treatment.** This code is listed in the most current HCPCS Level II manual for facility reporting of services. Validate your department Charge-master data and update any forms, paper or electronic, which may have the old C1300 HCPCS code.

Physician services are still reported with CPT code 99183. However, if the facility does not have prior authorization or has a non-affirmed prior authorization, the associated physician claims with the 99183 code will be subject to medical review related at this time to the three states listed.

Summary

With all the types of changes in front of healthcare providers for 2015, take a deep breath and exhale slowly, but with gusto, to calm down your two-headed dragon. Approach these changes pro-actively and incrementally: Gather your team, prioritize the issues that need to be addressed and resolved, designate and hold accountable those selected for specific tasks, and entrust your staff to use their knowledge and skill to complete each component in a timely and realistic manner. Working on the issues in small bits with a thorough understanding of the goal will ensure you will be ready to manage the two-headed dragon as you complete your readiness for the implementation of ICD-10-CM on October 1, 2015.

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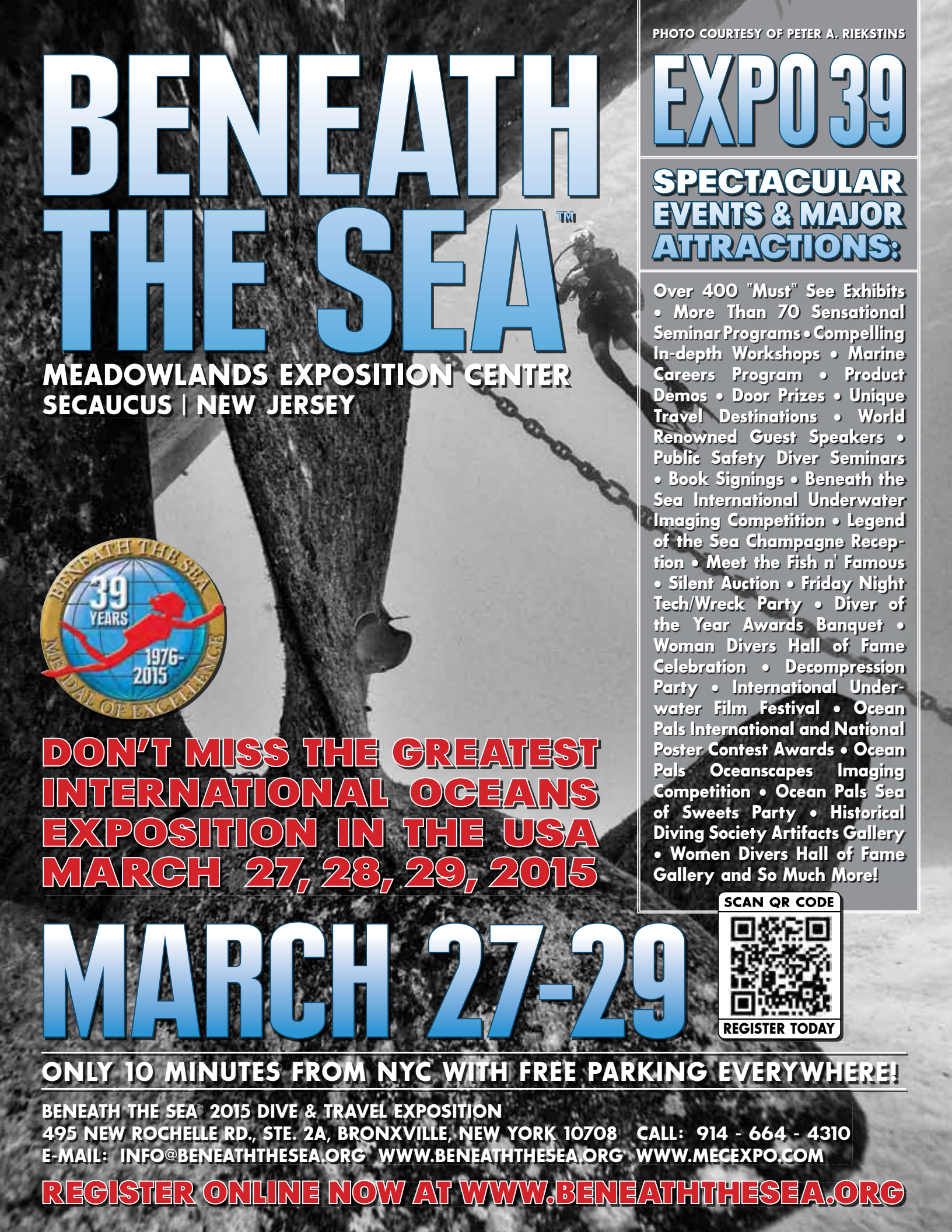

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Stresses in SCUBA and Breath-Hold Diving

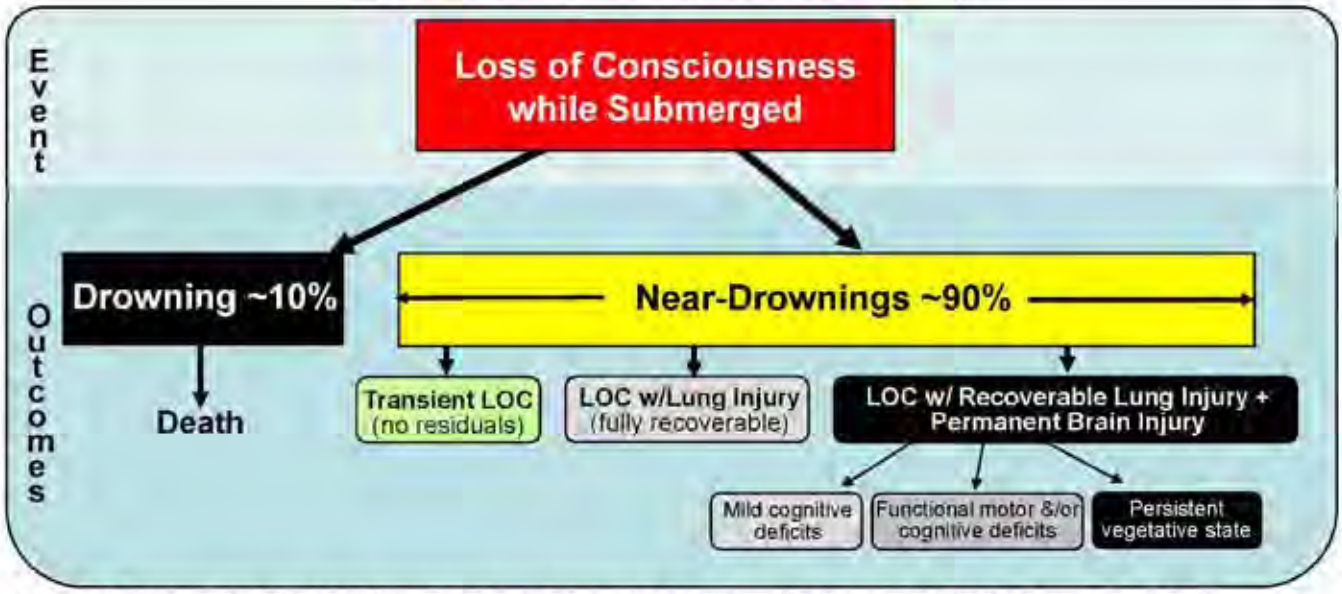
Part V: Near-drowning and Drowning

Michael B. Strauss, MD; Phi-Nga Jeannie Le, MD; and Stuart S. Miller, MD

Introduction

In the four previous issues 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, physiological, psychological, and no-panic syndrome stresses of diving.[1-4] In this article we will discuss the ultimate and most dreaded stress of all water related activities: suffocation in the water. It is associated with oxygen deprivation to the brain with loss of conscious as well as various degrees of insult to the lungs. Unfortunately, the body's reactions/responses to these devastating stresses are very limited in the absence of restoration of ventilation. The responses that somewhat mitigate these stresses are observed in the diving reflex and hypothermia, both of which will be discussed. Although 33 different definitions have been ascribed to drowning incidents, we will refer to them as near-drowning and drowning.[5] The near-drownings are further subdivided into events with no residual neurological problems and events with neurological residuals (Figure 1). While this article considers the subjects of near-drownings and drownings in general, as much of the information as possible will relate to SCUBA and breath-hold diving activities.

Figure 1: Drowning and the Spectrum of Near-Drownings



Legend: Drownings and near-drownings must always be considered in the context of the event, i.e., loss of consciousness while submerged, and the outcomes. The outcomes range from no residuals to coma. It is suspected that transient LOCs almost always go unreported.
Key: **LOC** = Loss of consciousness

Extensive literature exists on near-drownings and drownings and is excellently summarized in articles by Golden et al. (1997) and Szpilman et al. (2012).[6,7] Another authoritative source of information on near-drownings and drownings comes from the 2002 and 2011 World Congresses on Drowning (WCOD), with the resulting information adopted by the World Health Organization (WHO). The WCOD uses the Utstein template and defines drowning as "...experiencing respiratory impairment from submersion/immersion in liquid."[8] It supplements drowning information with outcomes that include death, morbidity, or no morbidity. Other terms such as wet, dry, near, passive, delayed/secondary, and the time interval between the event and pronouncing the victim dead are used to describe drownings, but the WCOD consensus is that these terms should no longer be used.

Whereas drowning deaths throughout the world generate large numbers, it is fortunate that only a small proportion occur in SCUBA divers. Statistics on SCUBA diving related drowning deaths from the Divers Alert Network (DAN) show that approximately 100 deaths (with about a 10 percent variance from year to year) occur in SCUBA divers in the USA, and approximately half are designated as drownings without any established etiologies for the loss of consciousness while submerged.[9] The data from drowning deaths in snorkelers and breath-hold divers is less well documented since these incidents tend to be lumped into all deaths where a mortal submersion occurs, and the recording of such deaths, especially for surface swimmers and snorkelers, is even less rigorous. The collection of data on breath-hold diving related drownings is being initiated by DAN, but numbers are not known to us at this time. Furthermore, there is no incentive to report near-drownings, especially those with no residual lung or brain injury. In the USA, near-drownings are estimated to be 500-600 times more common than deaths from drowning.[10] Regardless, any loss of consciousness in a water-related activity is a serious concern and especially tragic when the activity is voluntary and done for recreational, fitness, and/or sports-related purposes.

The actual number of drowning deaths throughout the world is unknown, with estimates as high as over 500,000 in 2001 and 372,000 in 2012, according to the WHO. In the USA 40 percent of drownings occur in children younger than four years old.[6] Drowning is a leading cause of death worldwide in children five to fourteen years of age. In the USA it is the second leading cause of non-disease, injury-related deaths (secondary to motor vehicle accidents) in children one to four years of age.[7] In terms of exposure adjusted person-time estimates, Szpilman et al. note that the chances of drowning is 200 times higher than such estimates from motor vehicle accidents.[7]

This article describes the precursors/risk factors associated with near-drowning and drowning, the pathophysiological events that occur with submersion injury, factors that influence favorable outcomes, and patient management from first response interventions to definitive management for victims that lose consciousness in the aquatic environment. Special consideration is given to relating these subjects to SCUBA and breath-hold divers. The question of whether or not to use the term "drowned" has some pertinence and is discussed next.

Never Say Drowned

Although death occurring while immersed in water is tantamount to drowning, and is the terminology we advocate, the admonition of "never say drowned" should always be remembered (Figure 2) and is well founded for two reasons. First, never say "drowned" because recoveries, some seemingly miraculous, have occurred after unconscious victims of immersion have been rescued and revived. Some recoveries have occurred after immersions of up to 60 minutes.[7,11] Age, absence of panic, the oxygen conserving/diving reflex, and cold water are factors associated with recoveries from prolonged (up to 30 minute) immersions and, in part, reflect the body's limited responses to the anoxic stress of submersion. A corollary to this admonition of "never say drowned" is the hypothermic victim with a profound bradycardia or asystole immersed for less than 30 minutes. Only after rewarming with no evidence of recovery should the victim be labeled

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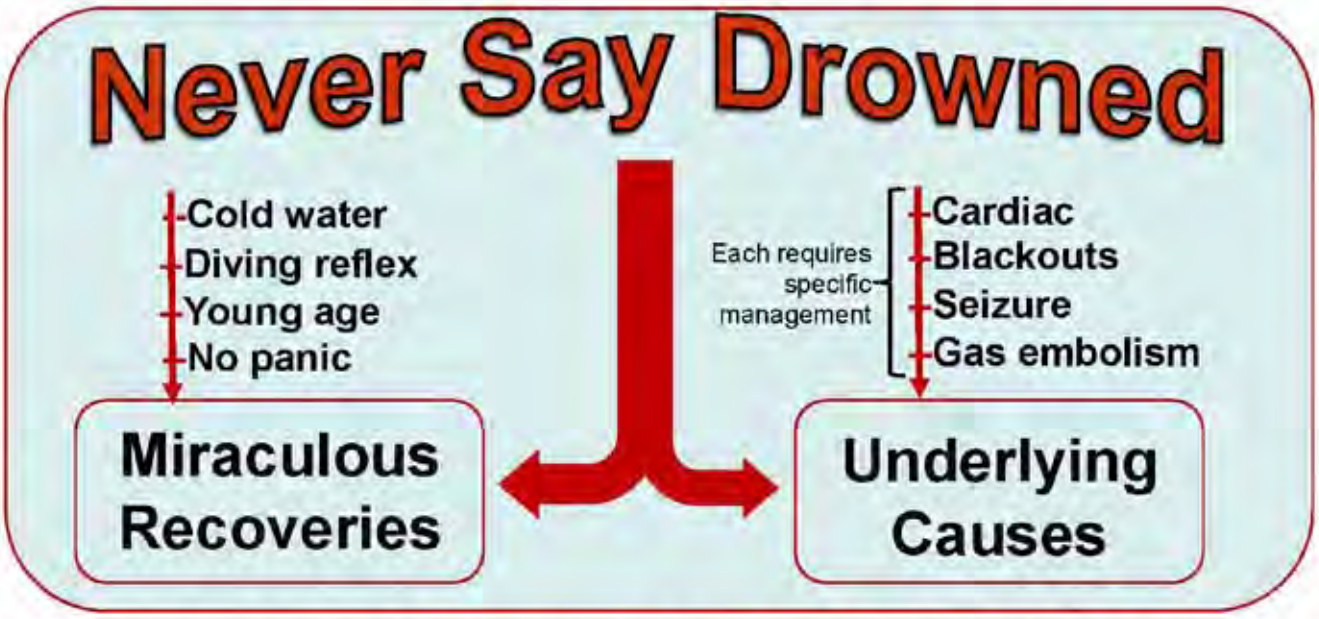
Figure 2: Why "Never Say Drowned"



Legend: When a victim is found unconscious in the water, they should not automatically be labeled as dead. About 90% of the victims recover. Furthermore, the caregiver should ascertain the cause of the loss of consciousness so etiology-specific appropriate care is provided.

as dead and nonresuscitatable. How these factors associated with the diving reflex affect recovery for the unconscious victim of water immersion will be discussed later in this article.

A purported (but not verified) record for recovery after prolonged immersion is that of a newborn being cast into a toilet bowl when the mother did not want to keep the baby. As the information goes, after two hours she reconsidered her decision and retrieved the immersed neonate. Miraculous spontaneous breathing and recovery occurred.

Comment: The new born status, the cold water, and the oxygen-conserving reflex are factors that would have contributed to a miraculous recovery of this sort. The oxygen-conserving reflex is strongly exhibited in the fetus and the newborn with its most characteristic sign being that of bradycardia, an objective sign of the fetal distress syndrome.[12]

The second reason for "never say drowned" is that in many victims of water immersion a preceding event leads to the loss of consciousness in the water. If the problem that led to the loss of consciousness is not recognized and appropriate interventions are not initiated, recovery will be hampered. For example, loss of consciousness from a cardiac event while immersed requires markedly different treatment than that from water aspiration associated with a blackout (see reference 4) or from unconsciousness due to an arterial gas embolism. This caveat of ascertaining the reason for the loss of consciousness while immersed is especially true for the near-drowning victim, where appropriate immediate early management is so crucial to achieve good outcomes. Often when a drowning occurs it makes the headlines of the local newspaper; however, follow-up information as to the cause of the loss of consciousness is almost never reported.

Causes and Risks Factors for Near-drowning and Drowning

There are multiple reasons why near-drowning and drowning occur. Probably the least frequent is that of forceful immersion as a consequence of homicide, attempted homicide, or torture. Conversely, the most frequent cause for near-drowning and drowning is that of risk-taking, especially with re-

Table 1: Risk-taking that Leads to Near-drowning and Drowning in Divers

Problem	Comments/Examples
General Risk Factors (see reference 1)	
1. Exceeding one's capabilities	Diving too deeply (nitrogen narcosis), swimming to/returning from dive sites (exhaustion)
2. Lack of awareness of diving conditions	Open water dives (disorientation), cave diving, hull penetrations (panic), diving in currents, rip tides, traversing surf zones (exhaustion and panic)
3. Alcohol and illicit drug use	Impairs judgment (panic, disregard for risks, increased susceptibility to nitrogen narcosis) (see reference 2)
Special Risks Associated with SCUBA Diving (see reference 2)	
1. Equipment related	Lack of familiarity (buoyancy control), inoperable or in need of servicing (equipment failures), loss of monitors —flooding, dead battery (disorientation, uncontrolled ascents, decompression obligations)
2. Entanglements	Especially with kelp and hull penetrations (panic, exhaustion of air supply)
3. Exposure and exhaustion	Hypothermia, surface swimming against currents (exhaustion)
4. Sensor failures, wrong gas mixtures	Insufficient oxygen partial pressures (hypoxia) with closed circuit rebreathers
Special Risks Associated with Breath-hold Diving (see reference 4)	
1. Profound hyperventilation	Blackout from hypoxia before CO_2 elevation signals the diver to breathe
2. Deep dives with hypoxia on ascent	Diffusional blackout (see reference 4)
Miscellaneous	
1. Diving with medical problems	Impaired heart function, uncontrolled diabetes, seizure disorder, stroke residuals
2. Diving in dangerous environments	Overhead boats (propeller/head injuries), polluted waters (toxic chemicals), sharks
3. Envenomation from marine animals	Usually from carelessness (stonefish) or handling (blue-ringed octopus, sea snakes)

spect to diving (Table 1). Several subcategories of risk-taking exist: first there is risk-taking associated with exceeding one's diving capabilities. Second there is risk-taking due to unawareness of the diving conditions or challenges. Third there is risk-taking with equipment-related situations. Fourth there is risk-taking associated with alcohol and/or illicit drug use in association with water-related activities. Alcohol has been reported in about 50 percent of drowning deaths, although the majority of these are in non-diving related water associated activities.[15] Other risk factors are those of SCUBA diving without adequate supervision/pre-dive briefings and disregarding the buddy system.

A 1994 event that made news headlines was that of a woman who drove her car into a pond with two of her children inside in order to kill them.[13] This is the epitome of a forced immersion.

Another news headliner is that of waterboarding as an interrogation technique. Of all forceful interrogation techniques, including drugs, sensory deprivation, absence of sleep, bodily harm, etc., waterboarding presumably is the most effective and the quickest from which to obtain responses.[14]

The technique of waterboarding is relatively simple. The victim is securely bound, placed on a slight downward incline, and the face covered with a cloth. Water is then used to block the nostrils until the victim is on the verge of suffocation. The urge to breathe apparently makes this the most effective interrogation technique without inflicting bodily harm, although deaths, presumably from aspiration of vomitus and/or cardiac causes, have been mentioned.

Comment: The bottom line is that oxygen deprivation before loss of consciousness (in the absence of no-panic syndromes) can be such a profound stimulus to breathe it can generate confessions even in the most hardened suspects.

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In addition, several factors are associated particularly with breath-hold diving. Profound hyperventilation before submersion is a significant risk factor for loss of consciousness during underwater swimming and breath-holding diving activities.[4] Another risk factor in this category is the breath-hold dive with resulting diffusional blackout.[4] Finally, there are serious risks for those who attempt to set world unlimited and free dive breath-hold depth records (now greater than 500 feet).

In SCUBA diving excess risks are associated with inadequate training, lack of familiarity with equipment, and/or poor fitness.[1,16] As mentioned earlier, drowning deaths in SCUBA divers are rare with reported deaths in the USA consistently remaining around 100 (±10%) per year.[9] Especially significant risks to SCUBA divers include diving too deeply with air, resulting in nitrogen narcosis; panic, which is frequently caused by entanglement; and depletion of air supply. Drowning deaths from decompression sickness and arterial gas embolism are exceedingly rare because the victims are usually on the surface when symptoms manifest themselves and a buddy diver is usually in attendance. Other SCUBA diving causes/risk factors associated with loss of consciousness in the water include hypothermia and exhaustion. With closed circuit rebreather diving, drowning deaths most often occur due to hypoxia from insufficient oxygen partial pressures caused by human error or equipment malfunctions.[3,17] There are also medical conditions that can cause loss of consciousness in water such as myocardial infarction, heart arrhythmias, stroke, seizure and hypoglycemia.[4] Trauma from boating accidents and shark bites (with acute blood loss) can be another cause of loss of consciousness in the water. Finally, there is the potential (possibly non-existent) for loss of consciousness in divers from venomous marine animal bites and stings, such as from the sea snake and the blue-ringed octopus.

Related Near-drowning and Drowning Terminology

A number of other terms associated with drowning are eschewed by the WCOD in favor of the simple outcome terminology of morbidity, no morbidity, or mortality after water immersion, as previously mentioned. The problem with this simplified terminology is that additional descriptions are required in order to define and/or explain the morbidity. Nonetheless, it is important to be aware of other terminology associated with near-drowning and drowning. **Sudden** (instantaneous) **drowning** was described by Keatinge in 1977.[18] He postulated that the immediate loss of consciousness and drowning deaths in aviators whose planes were shot down over the cold North Sea waters was due to uncontrollable gasps in the near freezing water. If the head were submerged, water would be aspirated and consciousness almost immediately lost due to brain hypoxia. **Wet and dry drowning** refers to whether or not enough water is aspirated to cause electrolyte imbalances in the body. Further discussion of this will occur later in this article. **Secondary drowning** refers to the delayed onset of pulmonary edema after a near-drowning episode[19] and is most frequently reported in near-drownings of children. It is believed to be due to a hypoxic insult to the alveolar capillaries, which gradually lose their integrity so that diffusion of serum into the alveoli occurs and causes the victim to become progressively hypoxic with dyspnea, tachypnea, confusion, agitation, and eventually lose consciousness. Treatment requires all measures necessary to manage pulmonary edema including breathing enriched oxygen mixtures, diuretics, intubation, and positive end-expiratory pressure ventilation.

Dive Scenario: A healthy, fit, well-trained SCUBA diver loses consciousness on the bottom in about 30 feet of water with no apparent cause. Fortunately, the dive buddy recognizes the situation and brings the unconscious diver to the surface. Immediate cardiopulmonary resuscitation is initiated and breathing and heart activity resume. The victim, while still unconscious, is brought to a nearby hyperbaric chamber where he is recompressed on a Navy Treatment Table 6-A (maximum depth of 165 feet) because of suspected arterial gas embolism. Near the end of the six-plus hour treatment the semiconscious diver becomes agitated. Immediately on completion of the treatment table the patient is transferred to an emergency department where he requires intubation for hypoxemia. Subsequent x-rays demonstrate extensive pulmonary edema.

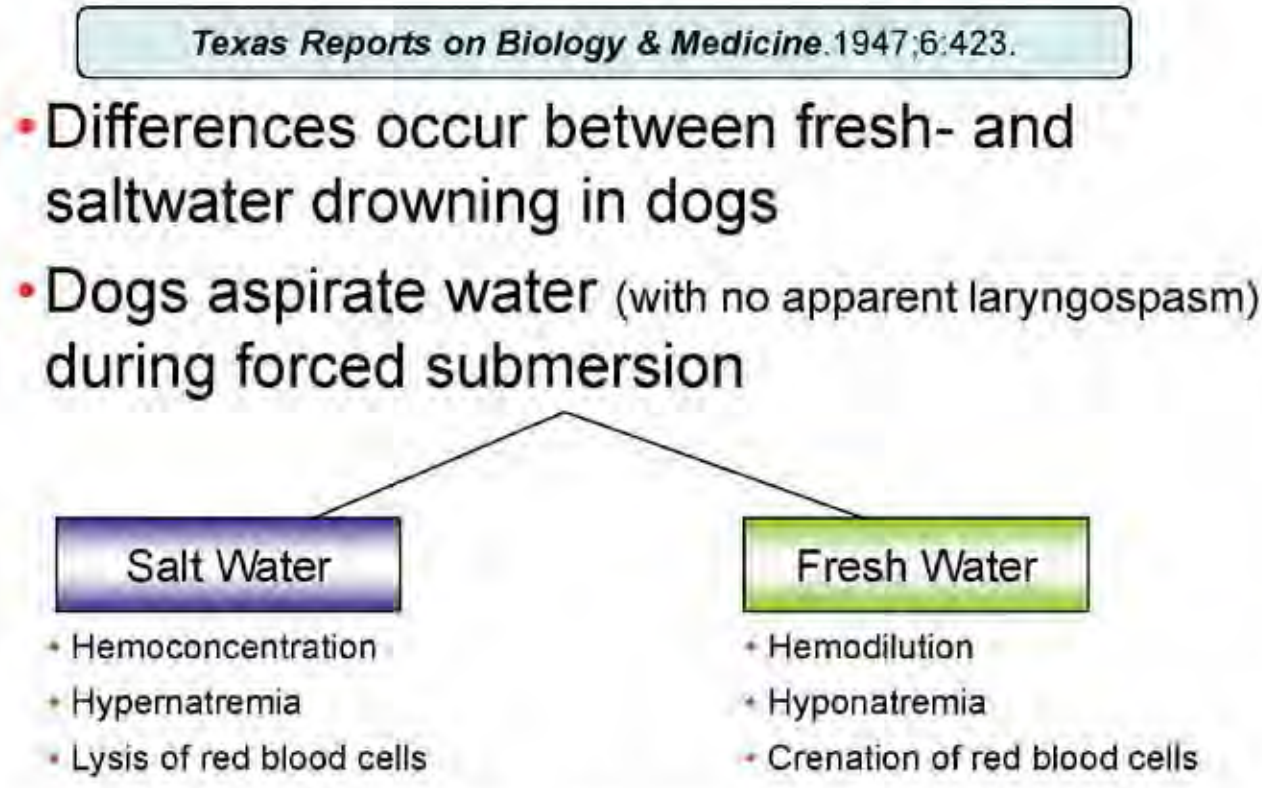
Comment: Many questions arise with this scenario, including why loss of consciousness occurred almost immediately on reaching the shallow bottom depth. An arrhythmia is probably the best explanation for this since no equipment or gas mixture problems were found during the investigation. Second, did the victim experience an arterial gas embolism since consciousness was lost on the bottom and not upon surfacing? Did the treatment table with high pressure oxygen breathing contribute to the pulmonary edema? Regardless, the onset of pulmonary edema (as manifested by agitation and confusion secondary to hypoxemia and "white out" of the lungs later obtained on a chest x-ray) was presumably due to the alveolar insult with delayed manifestation from water aspiration. These findings occurred nearly six hours after retrieval from the water and are postulated to be an example of "delayed" drowning.

Pulmonary edema, apparently in the absence of near-drowning, has been reported in SCUBA divers without loss of consciousness while diving.[20] The cases described occurred in cold water, but mention of water aspiration was not noted. The pathophysiology and management appear to be the same as described for secondary drowning.

Comment: It is known that small amounts of water aspiration into the lungs precipitates changes in lung function (described later in this article). Whether occult aspiration of water during the dive or moisture in the breathing gear was a contributing factor is not known.

Another variant of "delayed" drowning was associated in a breath-holding thoracic squeeze episode.[21] Three hours following an apparent full recovery after loss of consciousness and retrieval

Figure 3: Swann's Dog Studies from the 1940s



Legend: During the first author's time in medical school, this was the prevailing information on what happened (in humans) in near-drownings and drownings and from which test questions were derived.

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during ascent of a breath-hold dive (i.e., diffusional blackout—see reference 4), the victim became progressively hypoxic and failed to respond to treatment measures. Autopsy demonstrated serum and blood in the alveoli. The three-hour latency period represented the time it took for serum and blood to accumulate in the alveoli and reflected the characteristics of a delayed drowning.

Evolution of the Understanding of the Pathophysiology and Management of Near-drowning

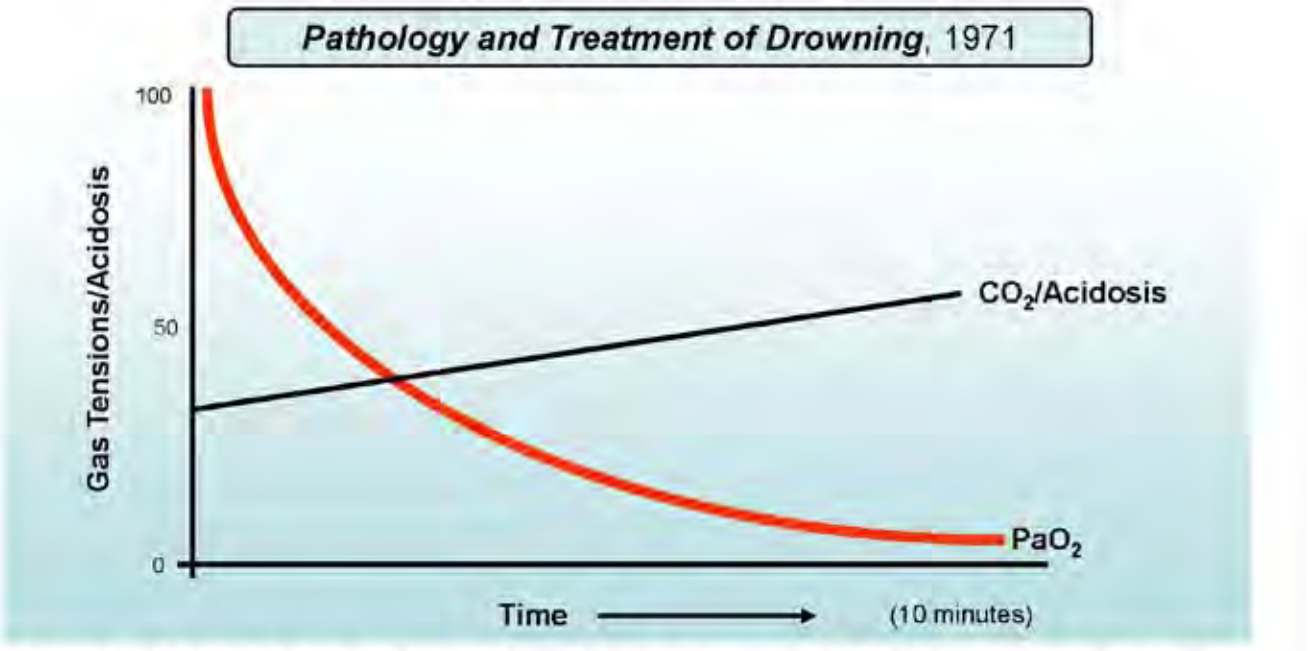
The understanding of what happens in near-drowning has evolved from total misinformation to a sound physiological basis today. Based on dog studies by Swann in the late 1940s, distinction was made between what occurs in fresh- and saltwater drownings (Figure 3).[22] Therapy was consequently directed at maintaining electrolyte balance because of hemodilution with freshwater drownings and hypernatremia with saltwater drownings.

Swann's study involved immersing anesthetized canines in fresh water and salt water. Dogs, apparently as a natural response to immersion, aspirate large quantity of waters (in contrast to humans); enough to cause severe disturbances in electrolytes and red blood cells (RBCs). In fresh water hemodilution, hyponatremia, and RBC hemolysis were observed in the canine model. In salt water the opposite occurred with hemoconcentration, hyponatremia, and crenation of the RBCs.

Comment: Observations in human near-drowning and drowning events indicate in almost all occurrences that insufficient water is aspirated to alter electrolytes or RBCs. More than 1.5 quarts of water need to be aspirated before significant changes in intravascular fluids and electrolytes occur. However, in almost all near-drowning and drowning events in humans, water is aspirated and differentiates the drowning as a "wet" type, as will be discussed shortly.

Modell in the late 1970s found that fluid and electrolyte imbalances were not the reason morbidity was associated with near-drownings.[23] Rather, it was due to hypoxia. With the newly acquired availability of arterial blood gas measurements, Modell showed that blood oxygen tensions fell precipitously with asphyxia in water, approaching nearly zero within 10 minutes (Figure 4). He advocated

Figure 4: Modell's Blood Gas Studies in Drownings



Legend: Modell demonstrated that hypoxia was the pathophysiological event that initially occurred in drownings.

Table 2: Modell's Method for Pulmonary Management of Drownings

Category	I	II	III
PaO_2	>50	<50	<50
Rate	<35	>35	>35
Insp Force	WNL	>25 cm H_2O	<25 cm H_2O
Vital Cap	WNL	>500 cc	<500 cc
Management	Monitor IPPB Chest PT	CPAP	Intubate PEEP Swan Ganz

Key: CAP=capacity, **cc**=cubic centimeters, **cmH_20**=centimeters of water, **CPAP**=continuous positive airway pressure, **Insp**=inspiratory, **IPPB**=intermediate positive pressure breathing, **PaO_2**=partial pressure of arterial oxygen (mmHg), **PEEP**=positive end-expiratory pressure, **PT**=physical therapy, **WNL**=within normal limits

using advanced pulmonary life support measures to achieve adequate blood oxygenation to protect the brain, and his approach utilized three categories of severity using blood gasses and respiratory parameters (Table 2). For the more severe presentations continuous positive airway pressure (CPAP) was used, and in the most severe situations positive end-expiratory pressure (PEEP) was initiated. Modell observed that if the near-drowning victim arrived in the emergency department alert, 100 percent recovery was observed. If the sensorium was blunted, 90 percent recovery occurred. However, if the victim was comatose at the time of arrival full recovery with his techniques only occurred in 50 percent of the near-drowning victims.

Subsequently, Conn amended Modell's recommendation to stress cerebral resuscitation in comatose patients after near-drowning.[24] He advocated "HYPER" therapy, which was an acronym for interventions to modify hydration, ventilation, body temperature, excitability, and rigidity (Table 3). With use of his "HYPER" therapy in 18 patients who arrived comatose, he observed that 61 percent had full recovery and only 5.5 percent had residual brain damage. He contrasted this in 21 patients using Modell's approach where 28 percent had full recovery and 38 percent had residual brain damage.

Current management for near-drownings is based on improved understanding of the pathophysiology of water immersion and optimization of management. In 85-90 percent of human drownings, water is aspirated and consequently the event could be considered a "wet" near-drowning, drowning event. This is confirmed at autopsy by the findings of diatoms from the aspirated water in the alveoli. The other small percentages of drownings are "dry" types where water does not enter the alveoli secondary to laryngospasm.

Drowning victims are too busy struggling (unless blackout has occurred) and too "air hungry" to yell for help. Once water enters the alveoli, four pathophysiological events occur (Figure 5). These include 1) decreased lung compliance, 2) ventilation-perfusion mismatching, 3) intrapulmonary shunting, and 4) surfactant washout. The common final denominators are hypoxemia and acidosis that lead to secondary problems of encephalopathy, acute respiratory distress syndrome, cardiac problems (ischemia, infarction, and/or arrhythmias), and renal shutdown.

All therapy is directed at maintaining adequate arterial blood saturations (above 90 percent) and acid-base balance including CPAP, PEEP, vasopressors, fluids, diuretics, acid buffers, intubation with barbiturates for sedation, etc. If aspiration of contaminated water is suspected, antibiotics are given. Finally, the use of steroids for reducing cerebral edema is controversial and apparently neither Mod-

Table 3: The Five Components of Conn's "HYPER" Therapy

- **H**ydration (dehydrate to reduce cerebral edema)
- **V**entilation (hyperventilate for vasoconstriction)
- **P**yrexia (cool to reduce cerebral metabolism)
- **E**xcitability (barbiturate coma to rest brain)
- **R**igidity (paralyze & intubate to optimize ventilation)

Note: With Conn's "HYPER" therapy, 61% of his 18 patients who arrived comatose demonstrated full recovery and only 5.5% had residual brain damage. He contrasted this with experiences where 28% of patients had full recovery and 38% had residual brain damage.

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ell nor Conn used them in their resuscitation protocols. An excellent algorithm for the evaluation and management of drowning victims has been generated by Szpilman et al.[7] They grade the victim from "Dead" to "Rescue" with six intermediate grades (1 to 6) based on the duration of immersion and the physical examination findings at the time of presentation to the emergency department. Management is specified for each grade with accompanying survival rates. Hyperbaric oxygen would seem a logical adjunct for mitigating the brain pathophysiology of hypoxia and cerebral edema (see text box below). Unfortunately, we are not aware of any reports of using HBO for such.

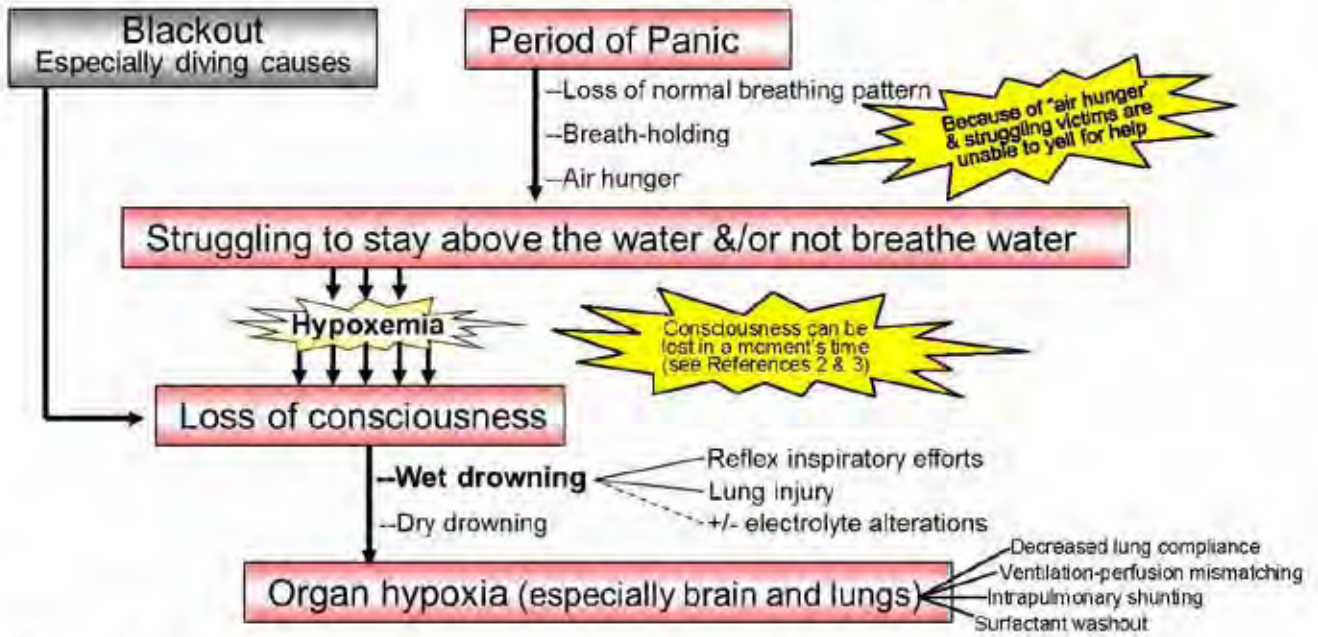
Hypothermia and hyperbaric oxygen (HBO) are two other therapeutic interventions that may have roles in improving outcomes for brain insult consequences of near-drowning, as well as other brain injuries. While Conn mentions hypothermia to slow brain metabolism it tends not to be used in near-drownings, though there is increasing awareness of its use for acute traumatic brain injuries.

The use of HBO is even more controversial. Since the acute brain insult in near-drowning is a combination of hypoxia and edema, possibly coupled with a reperfusion injury element, the acute use of HBO has justification (Figure 6).[25,26] Laboratory studies show significant improvements in outcomes when HBO is used in their models and the treatment has been coupled with hypothermia for a possible additive benefit.[27-29]

The Diving Reflex

Near-drownings, and the seemingly miraculous recoveries that have been observed after rescues, require a discussion of the diving reflex. The diving reflex is a series of innate responses that are associated with immersion in water (Figure 7). The reflex is highly developed in diving mammals and other aquatic animals, allowing them to remain submerged from six minutes (porpoises) to two hours (blue whales).[30] This series of physiological responses conserve oxygen and direct blood flow exclusively to the two most vital organs needed to safely continue the breath-hold dive; namely, the heart and the brain. The diving reflex has three components: 1) bradycardia, 2) vasoconstriction with shunting of blood to all body systems (except the heart and brain), and 3) anaerobic metabolism. Absence of panic, minimizing moments of the extremities, immersion in cold water, and young age (especially the fetus) facilitate the effectiveness of the diving reflex.

Figure 5: Sequence of Events in Diving-related Near-drownings and Drownings



Legend: Modell demonstrated that hypoxia was the pathophysiological event that initially occurred in drownings.

Figure 6: The Role of the Acute Use of Hyperbaric Oxygen for Near Drownings

Legend: The mechanisms of hyperbaric oxygen (HBO) have applications for the pathophysiology of near drowning, especially with respect to the brain injury.

The diving reflex is initiated by water coming in contact with the nasal branch of the trigeminal nerve, and the effect can be very profound. For example, heart rates in the seal slow by 90 percent to 10 beats per minute during the diving reflex.[30,31] Selective vasoconstriction is intense with almost total cessation of blood flow to all areas of the body except the heart and brain. This allows the oxygen content of the blood to meet the oxygen needs of the brain and thereby maintain consciousness during the dive. Anaerobic metabolism allows muscles for propulsion and feeding purposes to function in the absence of the usual oxygen requirements for aerobic metabolism. The "cost" of this is an oxygen deficit in the tissues that is met after surfacing by breathing air, which resolves bradycardia and vasoconstriction.

As previously mentioned, elements of the diving reflex exist in humans. Heart rates have been observed to decrease 40 percent in experienced breath-hold divers with immersion.[30] Other components of the diving reflex, such as vasoconstriction and anaerobic metabolism, also occur in humans. Vasoconstriction offers some protection from hypothermia by decreasing perfusion to the extremities. This helps maintain core temperature while reducing heat loss from extremities through the radiator effect of the relatively large surface area to mass of the limbs. Conditioning directly leads to improved tolerance of elevated levels of carbon dioxide and decreased levels of oxygen as well as the consequences of anaerobic metabolism.

First-response Interventions for Near-drowning Victims

The first step in any near-drowning event is retrieval of the victim from the water. If on the surface, the Red Cross water safety adage of "throw, tow, row and only then go" is sound advice. Certainly, the rescuer should not be put in jeopardy. What is worse than a drowning is a double drowning with the rescuer as the second victim. The next steps in the first response interventions are 1) getting the victim to a stable platform like a boat if in open water or the shore if nearby and 2) activating the Emergency Response System (best initiated by dialing 911 if in the USA). After this, basic life support (BLS) measures, which continue to evolve, should be initiated (Table 4). While performing BLS, the hypothermic victim should not be rewarmed. The reason for this is the hypothermic victim may be in asystole, and during the rewarming the initial car-

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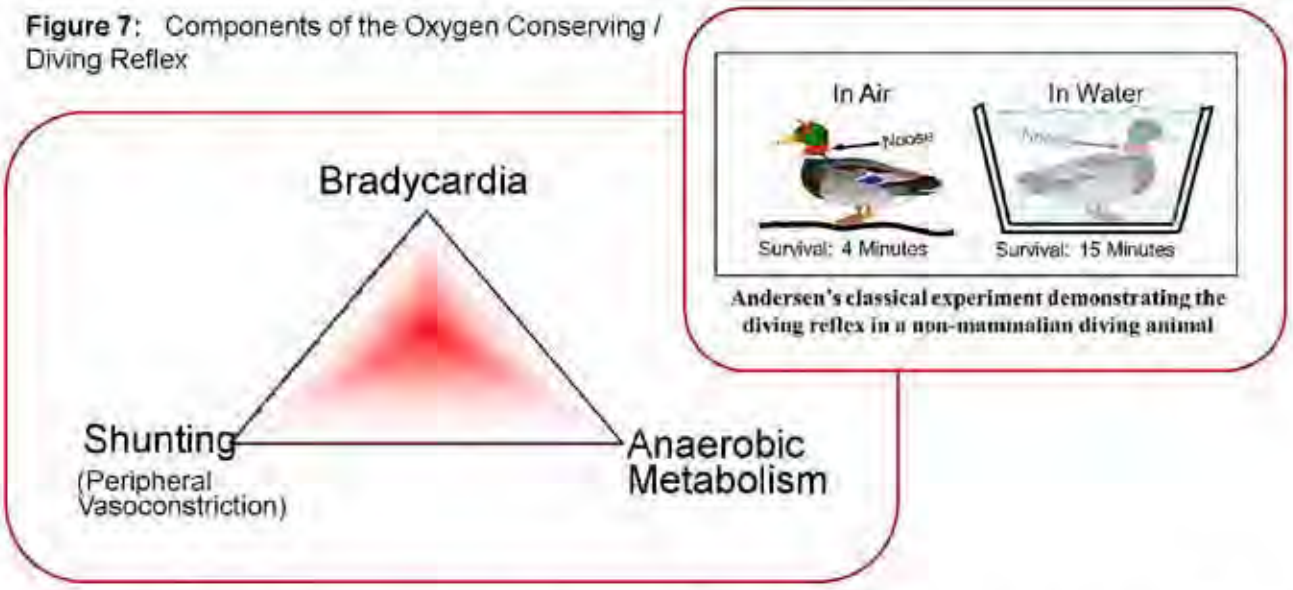
Figure 7: Components of the Oxygen Conserving / Diving Reflex



Legend: The diving/O_2 conserving reflex is initiated by water coming in contact with the terminal branches of the trigeminal nerve. Andersen's classic experiment with the duck shows how profound it is in prolonging survival with immersion apnea.[31] It has been observed in a variety animals, but best appreciated in the diving mammals. Components of the diving reflex can be observed in humans. The reflex is obliterated by struggling and panic.

diac response is likely to be ventricular fibrillation, which is rapidly fatal if not immediately corrected. Once advanced support with an automated external defibrillator is available and intravenous (IV) access established, rewarming while continuing resuscitation should be done. Rewarming techniques include removal of wet garments to prevent evaporative heat loss, shielding from wind, covering with warm blankets or clothing, application of warm water bottles to axillary and groin areas, instillation of warm IV fluids, and inhalation of warm, humidified air. Usually multiple techniques are used for additive warming effects. Heimlich "hugs" (abdominal thrusts) are not recommended for drowning victims as the maneuver is not effective in expelling water from the lungs and may precipitate vomiting that can lead to lung aspiration.

We do not advocate in-water resuscitation although a published report indicates otherwise.[32] In-water resuscitation—alternating mouth-to-mouth breaths with repeated chest level "bear hugs"—is not only difficult to do in the water, but is ineffective. Consequently, we advocate getting the victim to a stable platform as soon as possible where effective cardiopulmonary resuscitation can be done. Cold water immersion (i.e., in water substantially below body temperature) may slow metabolism as well as augment the diving reflex to improve the chances of survival.

Because of differential cooling of the core, which tends to remain warmer than the extremities whose temperatures approach that of the surrounding water, tourniquet use might be considered during the rewarming process. This is to prevent the rush of cold blood from the extremities from entering the core as a consequence of the obliteration of the diving reflex, which occurs with rewarming. Remember with vasoconstriction as a component of the diving reflex, warm core blood flow to the extremities is reduced, which lessens heat loss through the extremities due to their large surface area to mass ratio as compared to the core.

With core temperature monitoring and use of extremity tourniquets, as the victim of near-drowning plus hypothermia warms, the extremity tourniquets are reduced in a serial fashion. This is believed to prevent the temperature afterdrop observed with the rewarming of the hypothermic victim.

Table 4: Highlights in the History of Cardiopulmonary Resuscitation

1. **In the beginning…**He (God) breathed the breath of life into Adam's nostrils and the man became a living creature. (Genesis 2:7)
2. There are **anecdotal commentaries** throughout the ages for recovery of drowning victims
   - Mothers breathing life into their drowned children's nostrils
   - Restoring life to drowned victims by teeter-tottering them
   - Drowned victims coming back to life when transported on a wagon over a bumpy road
3. **1740 Paris Academy of Science**: Mouth-to-mouth resuscitation recommended for drowning victims
4. **1767 Amsterdam Society for Recovery of Drowned Victims**
   - Advocated resuscitation by means of mouth-to-mouth breathing or use of bellows
   - Utilized "fumigation" to stimulate the victim with orally or rectally insufflated tobacco smoke
5. **1903 George Crille**, a surgeon, introduced the technique of **external cardiac compression**
6. **1950-1973 Various techniques for artificial resuscitation**
   - Back pressure technique (victim prone, resuscitator straddles the victim's thighs and rhythmically applies pressure to the lower rib cage
   - Holger-Nielsen back pressure plus arm lift with resuscitator straddling the victim's head
7. **1954 James Elam** reported that **expired air was adequate to maintain adequate oxygenation**
8. **1964-1963 cardiopulmonary resuscitation was developed under the auspices of the American Heart Association headed by Leonard Scheris**
9. 1**973 to present National Conferences on CPR and ECC approximately every four years** with refinement of techniques and simplification of applications. Items such as **rhythms**, **exchanges** of rescuers, **ABCs** (airway, breathing, cardiac compressions), activation of the **ERS** (Emergency Response System), **advanced life support and pediatric advanced life suppor**t qualifications, and use of the **AED** (Automated Electrical Defibrillator). Heimlich maneuver for expelling foreign objects reported in 1974
10. **2010 American Heart Association Guidelines with CAB** (chest compressions, airway, and breathing) management, nearly **uniform rates**, **simplified exchanges** of rescuers, improved AEDs, and higher **quality training manikins**.

The unconscious SCUBA diver imposes additional challenges. If the diver is unconscious on the surface, measures as just described are appropriate. Inflating the buoyancy compensator (BC) and ditching gear in the water will lessen the exertion required by the rescuer to bring the victim to shore or the diving platform. More controversial is the management of the SCUBA diver who is found unconscious on the bottom. Three different "schools of thought" exist for handling this challenge:

First Option: Replace the regulator in the victim's mouth, gain control of the head with the head carry (as has been taught in Red Cross lifesaving and water safety courses), cradle the head on the rescuer's chest while extending the victim's neck, then perform a slow swimming ascent aided, possibly, by improving buoyancy through judicious inflation of the rescuer's BC. Once on the surface, the measures described for the near-drowning victim found on the surface are employed.

Second Option: Swim the victim to the surface with or without ditching the SCUBA tanks and regulator by grasping the victim's BC straps with or without improving buoyancy with the rescuer's BC. A variation of this is placing the rescuer's second stage octopus regulator into the victim's mouth before ascending.

Third Option: Swim the victim to the surface with or without ditching the victim's gear by grasping the victim's fins and letting the head dangle in the downward position during the ascent. Again, inflation of the rescuer's BC can be used to improve buoyancy.

We advocate the head control option for several reasons. With head control and neck extension of the unconscious victim, there is less chance of an arterial gas embolism occurring during ascent from retained air in the lungs whose egress is blocked by the flexed neck. Should the unconscious victim still execute agonal respiratory efforts, the regulator in the mouth may prevent aspiration of water. Finally, should spontaneous breathing resume during the ascent process, the regulator will provide an air supply and allow resumption of oxygen delivery to the brain and other tissues.

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Unconscious SCUBA divers using closed circuit rebreathers (CCR) present additional challenges.[33] Should water enter the breathing circuit, its reaction with the soda lime in the carbon dioxide scrubber will cause the breathing mixture to become caustic, which could cause severe respiratory system injury if breathing is resumed. Because of the special techniques utilized for CCR SCUBA, additional hazards must be considered.[2] These include seizure from oxygen toxicity and loss of consciousness from hypoxia. If the patient is seizing, is it recommended that ascent not be initiated until the seizure has ended.[33] With blackout from hypoxia breathing efforts may continue, so it is important that an air supply be maintained for the victim during ascent. The supply would preferably be from the open circuit pony bottle that CCR divers are recommended to carry. Finally, because of the ability for long, deep dives with CCR, it is likely that a decompression obligation or manifested decompression sickness will occur once the victim is brought to the surface. This possibility must be considered in the definitive management of the victim, as will be discussed in the next section.

Definitive Management of Near-drownings

After the unconscious diver is at a medical center advanced life-support measures are initiated, or if already started, continued with intubation, artificial ventilation, and intravenous fluids. If no spontaneous heartbeat or breathing is present on arrival at the emergency department, the prognosis for recovery is bleak. A decision to pronounce the victim dead may be made by a physician at that time. However, if the period of immersion was relatively short, that is to say 60 minutes or less, and the victim is markedly hypothermic, rewarming while continuing advanced life support should be considered.[7] When normothermic, a decision to continue life support measures should then be made. Medications as indicated are administered, e.g., anti-arrhythmic medications for irregular heartbeat, steroids if aspiration is apparent on chest x-ray, antibiotics if lung infection is a concern (the victim was in polluted water), and sedatives/paralyzing agents if the victim is resisting the ventilator. Blood tests and x-rays will help in decision making for management at this stage.

Once advanced life-support measures are established, it is necessary to decide whether hyperbaric oxygen (HBO) recompression is needed. If decompression was omitted or arterial gas embolism is likely, HBO recompression should be started as soon as possible. Another consideration (which is controversial) is whether to use HBO for brain resuscitation for the anoxic brain insult. If HBO is to be used as an adjunct for acute ischemic brain injury, the decision must be made jointly by the family, the attending physician, and hyperbaric medicine specialist.

A retrospective study that reviewed hospital admissions for drownings reported that 100 percent recovery was observed if the victim arrived in the emergency department with an intact heartbeat. However, if in-hospital cardiopulmonary resuscitation (CPR) was required, 47 percent of the patients died, 33 percent had residual neurological deficits, and 20 percent had full recovery.[34]

Comment: The target group for the acute use of hyperbaric oxygen treatments is, of course, the 33 percent group that has residual neurological deficits. Two problems arise with deciding which group requiring in-hospital CPR should receive HBO treatments. First, the group that has residual neurological deficits might not be readily identifiable for hours or days after the restoration of heart function as a result of anoxic insult-related or medically induced coma. Second, if the acute use of HBO is to be effective for brain injury, it needs to be started during the "golden period," which is thought to be in the two to six hour range after the time of the anoxic insult.

As soon as the near-drowning victim has stabilized, decisions for subsequent management are required. If the person is recovering well, rehabilitation is started for residuals of the neurological injury. If the victim remains in a persistent vegetative state, typical interventions in preparation for transfer to a long-term care facility include tracheotomy, percutaneous endoscopic gastrostomy tube placement, management of contractures, and prevention of pressure ulcers. The prognosis for recovery depends almost entirely on the severity of the anoxic brain injury, and the electroencephalogram may be helpful for determining long-term progno-

sis. Usually critical care management can resolve the lung injury regardless of the severity of the brain injury. The off-label use of HBO for victims of near-drowning who have serious residuals, but have plateaued with their rehabilitation, remains controversial.

The use of HBO in near-drownings (as well as with cerebral palsy and traumatic brain injuries) with significant residual brain morbidity has been anecdotally reported. HBO is used to try to mitigate the residual brain injury after recovery from the acute effects of the anoxic brain insult and after victims plateau with respect to rehabilitation efforts. Although some of the experiences with HBO are positive, the minimal functional changes we have observed have not materially affected the victim's quality of life.

The justification for using HBO in these situations is that there are "idling" neurons in a penumbra zone of injury that regain function with HBO treatments coupled with angiogenesis into the sites resulting in sustained oxygenation of the neurons previously impaired by hypoxia.[35]

Other controversies of using HBO include the number of treatments that are ideal, which range from 14 (angiogenesis effect) to 100 or more; depths of treatments that range from 1.5 atmospheres absolute (16 FSW) to 2.5 ATA (45 FSW); and whether repetitive series of treatments have benefits.

Favorable Prognostic Signs in Victims of Near-drowning

A number of favorable prognostic signs have been associated with near-drownings. First, the shorter the period of immersion, the better the victim's prognosis. Szpilman et al. reported that the risk of severe neurological impairment after hospital discharge was 10 percent if the period of immersion were zero to five minutes, 56 percent if six to ten minutes, 88 percent if eleven to twenty-five minutes and nearly 100 percent if greater than twenty-five minutes.[7] Other favorable prognostic indicators include immersion in water temperatures less than 50°F, a core temperature of less than 95° F, young age, and time to effective BLS less than 10 minutes.[6] Victims of near-drownings who arrive in the emergency department with a spontaneous heartbeat have better than a 50 percent chance of survival, whereas those without spontaneous cardiac activity have less than a 12 percent chance of survival.[7] Factors that complement the diving reflex, such as absence of panic, young age, hypothermia, and avoidance of extremity movements, also favor survival. If the victim is alert at the time of the arrival to the emergency department, the chance of survival without neurological residuals approaches 100 percent.[23] Other factors that favor a good prognosis at the time of arrival at the emergency department include the female gender, absence of aspiration, time to basic life support of less than 10 minutes, blood pH greater than 7.1, blood glucose greater 112 mg percent, Glasgow Coma Score greater than 6, and the presence of the pupillary response.[6]

Myths and Unresolved Questions about Near-drownings and Drownings

1. Precise definitions that are established by experts should always be used for a victim who has experienced a loss of consciousness in the water.

Comment: We advocate simplicity; "drowning" if the victim is dead and "near-drowning" if alive after recovery and resuscitation efforts. However, the severity of residual neurological injury in near-drowning ranges on a continuum from none to persistent vegetative state (Figure 1).

2. Drowning is a sufficient diagnosis for anyone who has lost consciousness in the water.

Comment: As mentioned before, "never say drowned." It sounds like a paradox to acknowledge this adage, but then use the term drowning. The message is that the underlying cause of the loss of consciousness in water needs to be ascertained, be it from decompression illness, breath-hold blackout, cardiac arrest, seizure, trauma, etc. The cause dictates the optimal management for the near-drowning treatment.

3. Usually near-drownings and drownings occur in the absence of risk factors.

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Comment: An analysis of the circumstances surrounding the drowning will usually identify underlying risk factors that lead to the loss of consciousness such as a heart attack from coronary artery disease, excessive hyperventilation leading to blackout, violation of decompression practices, etc. (Table 1).

4. All victims of near-drowning, even if asymptomatic, need to be observed for 24 hours before discharge from the medical center where initially evaluated.

Comment: Although delayed onset of pulmonary edema may occur within the first 24-48 hours after the near-drowning event, if the patient is asymptomatic with normal ventilatory functions observation in the hospital setting is not necessary. However, at discharge the patient and/or family should be aware of delayed onset of pulmonary edema associated with near-drowning and instructed to return the patient to the medical facility immediately if shortness of breath, cough, or other respiratory symptoms occur.

5. The primary concern in near-drownings is the management of the respiratory insult.

Comment: Although adequate ventilation is essential for recovering the victim, management of the possible anoxic brain insult must not be overlooked. Whereas respiratory function will inevitably recover, the neurological injury is more likely to be irreversible.

6. If the period of loss of consciousness while immersed is greater than four minutes, resuscitation efforts should not be initiated.

Comment: Although death of brain neurons occurs after four minutes of anoxia, with intact heart function oxygen physically dissolved in the blood is still being delivered to the brain. This is complemented by the diving reflex, which is initiated by immersion. This prolongs survival and promotes full recovery after immersion exceeding four minutes. In addition, hypothermia can slow metabolism and reduce brain oxygen demands.

7. In the USA children's near-drownings are usually in open waters such as lakes and oceans.

Comment: In the USA, the most common cause of near-drownings and drownings in children occurs when they fall, unwitnessed, into a backyard swimming pool, which is an event typically associated with families affluent enough to have swimming pools. "Water safe" in a child does not guarantee that with clothes, shoes, and possibly restrictive garments on, he/she can swim to safety. Pediatricians have said that the only safe thing to fill a backyard pool with is sand! In third world countries, drownings are usually from play activities in (usually polluted) rivers.

8. In most near-drownings and drownings no water enters the lungs due to the laryngospasm reflex.

Comment: Although the laryngospasm reflex is very profound, and one of the last to be lost with impending death, in 85-90 percent of near-drownings and drownings water is aspirated. This qualifies the drowning as a "wet" type. Even small amounts of aspirated water can alter pulmonary functions and require critical care management.

9. Autopsies of drowning victims always have pathognomonic findings.

Comment: Often autopsies of drowning victims demonstrate no specific findings for the cause of death. Aspiration of sea water may show diatoms in the lung tissues. Heart disease can be confirmed by atherosclerosis of coronary arteries, but deaths from arrhythmias are usually undiagnosable. Thoracic squeeze injury can show edema and blood in the alveoli.

10. Near-drownings and drownings associated with SCUBA diving need not be managed any differently than the problems from other causes.

Comment: Special training and techniques are needed to manage SCUBA divers found unconscious on the bottom. Once at a medical center, a decision needs to be made as to whether hyperbaric oxygen recompression is needed for omitted decompression and/or arterial gas embolism in addition to the usual advanced life support measures.

Conclusions

The management of near-drowning has made noteworthy advancement in both basic life support as well as advanced life support. Unfortunately, the human only has limited reflexes/responses to the hypoxic insult associated with immersion in contrast to almost all of the other stresses associated with diving. The diving reflex and induced hypothermia by the surrounding water environment are the limited responses the body has to deal with the anoxic immersion stress. Although lung function is usually recoverable with advanced life support interventions, neurological injury is often not recoverable. Consequently, attention to rescue and management must always be directed to preventing anoxic brain injury. Usually, underlying causes lead to unconsciousness in the water, hence, "never say drowned" as the cause. Rather, seek the underlying cause and remember that seemingly miraculous recoveries have occurred after recovering a victim from a presumed drowning. This directs treatment interventions from heart attack management to recompression treatments and from observation only for delayed effects of the immersion to physical therapy. Finally, the best measure for near-drownings and drownings is prevention through water safety and diving knowledge. In no water activities is this truer than in SCUBA and breath-hold diving.

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Dr. Michael B. Strauss has been "brainwashed" about near-drowning and drowning since taking his first Red Cross Junior Life Saving Course in 1948 from his father, a self-learned "professor" of swimming-related activities. From his mother, a high school graduate genius, he was cautioned about risk taking in water activities such as never to swim hard after eating a big meal (a subject for another paper). In medical school Dr. Strauss remembers being tested on the differences (now anathema) between fresh and salt water drownings. Later, in his associations with Dr. Ronald Samson, who worked with Dr. Jerome Model, he learned of the hypoxemic/loss of consciousness insults of water immersion. Then from Dr. George Hart, his "mentor of all mentors," he learned about the mechanisms of hyperbaric oxygen and how they can mitigate hypoxic insults. Finally, Dr. Thomas Asciuto, a hyperbaric medicine colleague and critical care specialist, "fine-tuned" him on the contemporary understanding of the pathophysiology and management of near-drownings. Consequently, these mentors and colleagues deserve special recognition for the genesis of this article.

Phi-Nga Jeannie Le, MD is fellowship trained and board certified in Undersea and Hyperbaric Medicine. This is her first of many planned collaborations with the prolific Dr. Strauss. For Dr. Le, any collaboration with the experts and teachers at the Long Beach Memorial Medical Center Hyperbaric Medicine Program is a continuation of lifelong education in the specialty of undersea and hyperbaric medicine that began at the University of Pennsylvania. Though total physical undersea submersion herself is not her passion—nor will her middle and labyrinth of the ear permit such activity—the clinical science and advancement of the field is. Dr. Le enjoys being the one on the surface taking care of the intrepid land mammals who get themselves into trouble playing sea creatures.

Stuart S. Miller, MD is the associate medical director and director of education of the Hyperbaric Medicine Department at Long Beach Memorial Medical Center. He is board certified in Emergency Medicine and fellowship trained/board certified in Undersea & Hyperbaric Medicine. He has co-authored over 40 articles, posters, and book chapters on hyperbaric medicine, wound care, and diving medicine. He has given numerous lectures and is the course director for undersea and diving medicine CME conferences. He has been an avid SCUBA diver for over 25 years.

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Wound Care & Hyperbaric Medicine magazine

Volume 6 Issue 1 • Spring 2015

Sinus and Ear Disorders that Take Place during Hyperbaric Oxygen Therapy

Emanuele Nasole, MD; Antonio Paoli, MD, BSc; Gerardo Bosco, MD, PhD; and Enrico Camporesi, MD

Sinus and internal and external ear disorders are the most common side effects of hyperbaric oxygen therapy (HBO_2).[1] These spaces are the cranium's pneumatic sockets and, particularly those of the middle and inner ear, are most frequently involved in the pressure stress caused by compression and decompression maneuvers during exposure to altered pressures in the hyperbaric chamber. Barotrauma is the mechanical tissue damage produced by environmental pressure variation, and the middle ear is the most frequently involved structure in this kind of damage. According to Boyle's law (the product of pressure and volume is a constant for a given mass of confined gas) it is easy to understand why all enclosed air cavities are more susceptible to this kind of lesion. Barotraumas can occur due to an increase or decrease of gas volume. To avoid gas volume decrease during the compression phase, the patient must perform some compensatory maneuvers aimed at inhaling and forcing gas (air or oxygen) into the nasal and sinus cavities. During decompression in the chamber or even underwater, the body's gas expands and is expelled from cavities to the outside, usually without any active maneuver.

It is essential to teach the patient about the functions of the hyperbaric chamber and the correct maneuvers of baro compensation. In this article, we will describe the main barotraumas that can occur during HBO_2.

External Ear Barotrauma (EEB)

In normal conditions, the caliber of the external auditory canal is sufficient to allow immediate pressure compensation. An EEB can only happen if the external auditory meatus is blocked by an obstacle, such as impacted earwax, external otitis, or ear plugs, during chamber compression. In this case during compression the obstacle prevents the equilibration between outside and inside pressure. During the compression phase there is a reduction of volume both in the plugged external ear and in the middle ear spaces. This leads to edema and petechial hemorrhages of the auditory tube, middle ear, and tympanic membrane. Rare complications include ear bleeding and tympanic perforation. Symptoms include acute pain, deafness, vertigo, nausea, and ear bleeding. The treatment therapy consists of antibiotics and topical steroids.

Middle Ear Barotrauma (MEB)

The most common side effect of HBO_2 is middle ear barotrauma.[2,3] MEB is more common during compression, while during decompression it is less likely to be reported. MEB during compression is a more pathological event and is related to a failed compensatory maneuver to relieve pressure between the middle ear and Eustachian tubes. The incidence of reported MEB varies between different hyperbaric centers from 5 to 66.7 percent.[4,5] This difference is due to the heterogeneous population sample (intubated patients vs. spontaneous breathing patients) or to other causes.

Other common causes of MEB include the presence of diseases of the upper respiratory tract

that generate obstructions of the Eustachian tube (e.g., infectious rhinitis and allergic and nonallergic rhinitis; ingestion of alcohol, which causes nasal congestion; large nasal polyps; large septal deviations; tobacco smoke; use of beta blockers and parasympathomimetic drugs; etc.) and incorrect or delayed compensation techniques.

Depression of the middle ear tympanic membrane during the chamber compression begins to appear at a pressure of 1.2–1.3 atmospheres absolute (ATA) with reduction of the volume of endotympanic gas by 20-30 percent of the initial volume. There is a retraction of the tympanic membrane with pain, hyperemia, and edema of the ME mucosa that could lead to hemorrhage and cause the tympanic membrane to rupture. This generally produces a grade 1 or 2 MEB (according to the Teed classification) in more than 90 percent of patients; about 20 percent of patients with ear pain show tympanic membrane alterations short of perforation (MEB >grade 1).

MEB is prevented in most patients by teaching autoinflation techniques or by use of tympanostomy tubes for those who cannot autoinflate their middle ear compartment. A prospective study in patients treated with HBO_2 demonstrated that many patients develop serous otitis media during serial treatments. A history of Eustachian tube dysfunction predicted serous otitis media.[6] Pseudoephedrine has been demonstrated to be effective in preventing barotitis media in a double-blind randomized controlled clinical trial in underwater divers.[7] Conversely, topical nasal oxymetazoline hydrochloride was found to be ineffective in preventing middle ear barotrauma during HBO_2.[8]

Other Complications

Sinus squeeze is the second most common in-chamber complication[4] and usually occurs in patients with upper respiratory tract infections or allergic rhinitis. Usually a program of decongestant nasal spray, antihistamines, and/or steroid nasal spray just before compression allows the hyperbaric therapy to continue.

Serous otitis has been reported in patients receiving HBO_2 therapy.[6] Although once thought to be due to reduced middle ear pressure by oxygen resorption, there is evidence to suggest that HBO_2 might cause a reversible derangement in a middle ear chemoreceptor reflex arc that may regulate middle ear aeration.[9]

Hyperbaric Experience in Monoplace Chambers

Specific considerations for patients treated in monoplace chambers have been recently summarized in an analysis of adverse events using data from all Diversified Clinical Services centers operating for the period of 2009–2010.[1] Diversified Clinical Services (now Healogics) provides management services to 340 hospital-based outpatient wound care centers, of which 89 percent provide outpatient hyperbaric oxygen treatment to diagnoses limited to those listed in the UHMS *Hyperbaric Oxygen Therapy Indications*. Adverse event data was collected concurrently in a central proprietary database.[1]

The primary adverse event categories were ear pain, confinement anxiety, hypoglycemic events, shortness of breath, seizures (including both oxygen toxicity and hypoglycemic event seizures), sinus pain, and chest pain. Reporting data was reviewed from 463,293 monoplace hyperbaric oxygen treatments provided in hospital-based outpatient settings involving 17,267 patients (an average of 27 treatments per patient). The majority of these patients received hyperbaric oxygen treatment for diabetic limb salvage or complications associated with prior radiation therapy.

In 2009 there were 916 adverse events reported for 207,479 treatments in 7,871 patients, an overall adverse event rate of 0.44 percent. In 2010 there were 954 adverse events reported for 255,814 treatments in 9,396 patients, an overall adverse event rate of 0.37 percent. In order of decreasing rate of occurrence were ear pain (of any description), confinement anxiety, hypoglycemic events, shortness of breath, seizures (including both oxygen toxicity and hypoglycemia-related seizures), sinus pain (of any description), and shortness of breath. There was no significant difference in the number or ranking of adverse events between 2009 and 2010. In this series of

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treatments, all patients received a standardized medical evaluation prior to initiating treatment, standardized pretreatment education, and a standardized assessment prior to each treatment. The consistent attention to detail may be the cause of such a low rate of complications.

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Clinics In Focus
Memorial Hospital Wound Clinic/ Hyperbaric Medicine Department, University of Colorado Health

Continuing our series of interviews featuring outstanding hyperbaric and wound care centers around the world, we spoke with Courtney S. Hoffbauer, RN, MSN, Clinical Nurse Manager of the Orthopedic/ Neurosurgery Unit, HBO, and Wound Care at Memorial Hospital, University of Colorado Health, which is accredited with distinction by the UHMS.

How has seeking UHMS accreditation affected your clinic?

Our accreditation raises the standard of practice to the next level. UHMS accreditation sets a standard for continuous improvement in regard to quality and patient care. Continuing education throughout the year is a mandate for all staff in order to continue accreditation. Quality improvement for patient care is at the forefront of accreditation. Memorial Hospital's HBO department continues to not only meet our patients' expectations, but exceed them.

The staff at Memorial Hospital Wound Clinic/Hyperbaric Medicine Department in Colorado Springs, Colorado.

What are the most common indications treated at your clinic?

The top five indications we treat most often are: diabetic foot wound, radionecrosis, carbon monoxide poising, necrotizing fasciitis, and decompression sickness.

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

We treated a patient who demonstrated one of the best limb salvage cases we have seen. As the nurse presented to the room for a consult, a surgeon was at the bedside informing the patient of the risks of refusing an amputation. The nurse vividly recalls the patient stating: "I have to keep trying before I opt to lose my leg." It was that moment the nurse told the patient that, "We will do everything in our power to salvage your leg." After a series of 30 daily treatments, this same nurse happened to encounter this patient several months later, as the patient walked by on both legs. The patient was so very grateful for one more chance.

Do you work with a management company?

We work with a consulting team from South Carolina, National Baromedical Services—the best in the country.

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

The team that works every day with the patients in the unit is the leading factor of success for us. The team members know each other well and work side by side to ensure patients are receiving the best care. The team at the HBO unit has been working together for over five years, which is demonstrated by the cohesive nature of the group.

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

Dr. Rob Price, our medical director, who also is an active-duty Lieutenant Colonel for the U.S. Army, plays a vital role in the success of our unit. One of his many strong attributes is the ability to network with and educate community physicians. We feel that community reputation is key to increasing patient census. We have a relationship with both the local U.S. Air Force Academy and Fort Carson Army Base to ensure we are available for their cadets or soldiers when in need.

Are there are any additional questions you'd like to answer, or is there any other information about your clinic you would like to showcase?

One of the most important facts about our HBO clinic is we are the only 24/7/365 HBO department left in the state of Colorado. Unfortunately, 24/7 HBO facilities are growing scarce; however, because of our relationship with our armed forces, we feel this is critical in our community.

Our Wound Care Clinic saw a record 9,192 patients in 2014. The clinic has a variety of physician specialties available for our community, and in conjunction with HBO has saved many patients from losing limbs. The wound clinic uses cutting-edge technology to ensure we provide our patients with the very best. We were the first in the state of Colorado to utilize Cellutome, which is a skin-grafting outpatient procedure.

CLINIC DETAILS

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Dr. Rob Price, Medical Director

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How to Make Your Hyperbaric Medicine Practice Stand out from the Pack

From the Best Publishing Company *DEPTH* Blog

Each of us in the field of hyperbaric medicine wants to create an exceptional practice. Part of creating a practice that makes you "stand out from the pack" is finding a continuing education program to keep you up-to-date on cutting edge medical advances in hyperbaric medicine and growing your network of industry colleagues to help take your practice to the next level of success.

Our colleagues at the Mayo Clinic have organized a team of experts in hyperbaric medicine for a unique two-day 16.0 hour CME/MOC accredited event on April 17-18, 2015 in Rochester, Minnesota titled Hyperbaric Medicine 2015.

CONFERENCE DETAILS

Hyperbaric Medicine 2015

April 17-18, 2015

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The Mayo Clinic in Minnesota has been recognized as the best hospital in the nation for 2014-15 by U.S. News and World Report. They are a nonprofit worldwide leader in medical care, research, and education for people from all walks of life.

Conference topics will include:

- HBO_2 therapy in lower limb crush injury, compromised flaps, and deep tissue infections
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Clarity
The Importance of Patient Education

Darren Mazza, EMT, CHT

One of the most important components of hyperbaric therapy is providing complete and informative patient education. Simply speaking to patients about risks and benefits alone doesn't constitute thorough education or ensue that the patients understand their responsibilities. It is also necessary to educate patients on the importance of maintaining both good nutrition and hygiene, as they both have a direct impact on patient safety, health, and healing ability.

When possible, encourage a family member to participate in the patients' education. This can be a great way to encourage patient compliance. Family members are often times the only source of encouragement patients have aside from health care providers. Be clear when educating both the patients and their family members about safety measures.

As a CHT/safety director, I have an obligation to provide the safest environment possible for both the patients and staff in the clinic. Hyperbaric patients also have an obligation to both themselves and the clinic staff to comply with all aspects of the instructions provided to them. One of my goals is to encourage patients to take ownership of their health and their care to achieve their goals towards healing.

Always provide exceptional hyperbaric patient education. This lends the CHT credibility and demonstrates his or her competency and commitment to patient safety. This in turn gains patient trust and confidence in both the CHT and clinic.

One great example of providing good patient education is on the use of the air break line and mask. Don't just hand the air break line and mask to the patient and send him into the chamber. The purpose of thorough patient education is to properly inform the patient of what the equipment is and how it works. The patient needs to be familiar as well as competent with the air break equipment. When teaching the patient how to properly use the air break equipment, instruct him to take two breaths from the air break line. Confirm that the mask creates an adequate seal and that the regulator provides proper air flow to the patient. This will ensure patient clarity and confirm that he has been prepared for treatment with adequate education and safety training.

Final Note: Don't cut corners with patient education. It's absolutely crucial for us as care providers to take the time and properly prepare patients for all aspects of the treatment they are about to receive.

About the Author

Darren Mazza is the CHT and Safety Director at the Center for Wound Healing and Hyperbarics at Swedish/Edmonds, located in the greater Seattle area. He has 20 years of experience in healthcare, which includes 8 years as an EMT in the greater Sacramento region. Darren also worked as a preceptor trauma tech in a Sacramento hospital for several years. After leaving California and moving to Idaho in 2005, his hyperbaric career began after becoming the department head of an outpatient wound care and hyperbaric center. His hobbies include fly fishing and fly tying.

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Oxygen, Hyperbaric Oxygen, and Free Radicals

Wound Management Considerations

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

Introduction

No one questions the roles oxygen has in wound healing. In almost every aspect of wound healing, from the inflammatory process to remodeling and from tissue survival to infection control, adequate tissue oxygen tensions are essential. The roles of hyperbaric oxygen (HBO), however, are not as well defined. When wound healing is not progressing in a normal fashion and ischemia/hypoxia is a contributing factor, it makes sense to use HBO as an adjunct to manage this aspect of the problem. Even more controversial is the role of free radicals in wound healing and whether the production of free radicals by HBO is detrimental to healing and survival. This article addresses these three aspects of the oxygen molecule spectrum and dispels misconceptions about the harmful nature of free radicals.

Things to Know about Oxygen

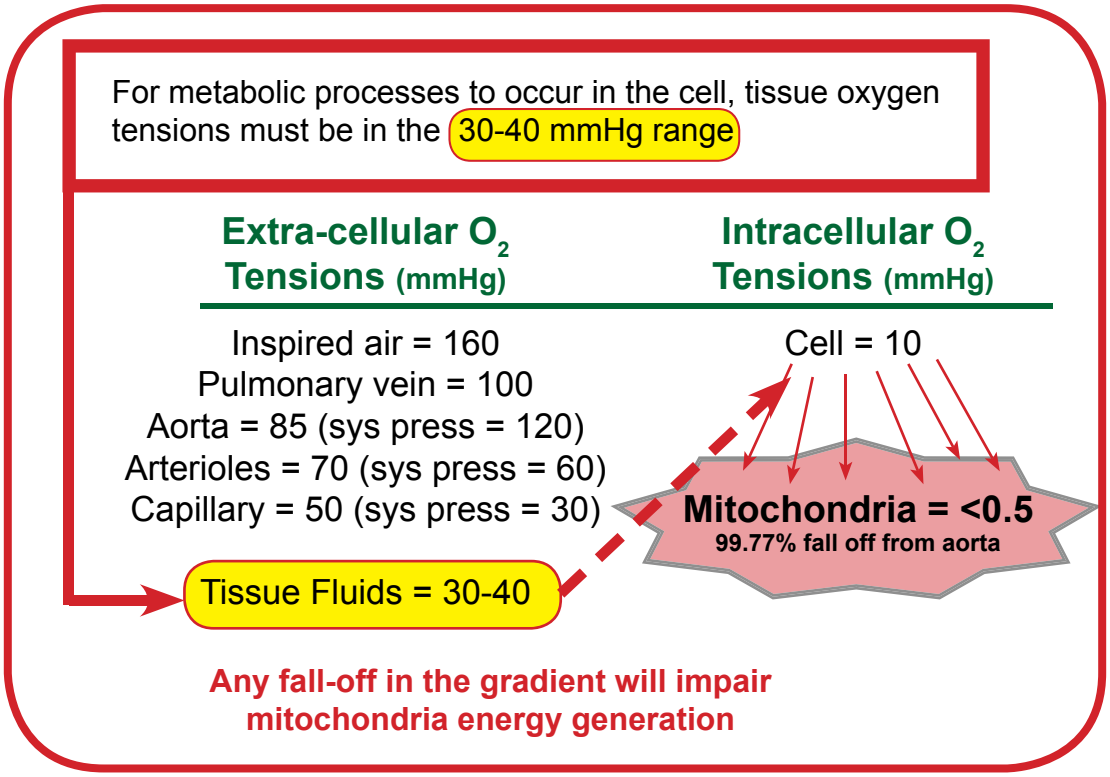
The chemical element oxygen has many unique features. It has the ability to oxidize almost any organic substance to modify it. It does this by "gobbling up" electrons from the compound, which defines oxidation. It also actively forms oxides with inorganic elements such as iron, which results in the rusting process. No other element is so active in the degrading/oxidizing process. As "destructive" as oxygen is, life as we know it would not exist on earth without oxygen. The question is how is this paradox resolved? The answer is that defenses against oxygen were developed as evolution of life on Earth progressed.As Nick Lane said *in Oxygen: The Molecule that Made the World*, oxygen is the "elixir of life—and death."[1] Much is known about

The chemical structure of oxygen is such that its nucleus of 8 protons and 8 neutrons sits in a protective shell (also seen in helium, calcium, nickel, tin, lead, and other heavier elements), which makes its molecular form very stable and prevents decay into other elements. This accounts for oxygen being the third most common element in the universe, second only to hydrogen and helium.

What is more pertinent with respect to oxygen's reactivity is its electron cloud. The lowest tier energy level, the s-shell, is spherical and filled with two electrons. The next energy level/shell, the p-shell, can hold 8 electrons. However, in oxygen's case, it has 6 electrons. Consequently, it aggressively seeks 2 additional electrons to fill this shell and stabilize the shell's energy state. This makes oxygen the most active chemical element in "gobbling up" electrons to combine with or to degrade other elements and compounds.

In contrast, carbon forms bonds with itself and other elements because carbon (element 6) has 4 electrons left over and needs 4 more electrons to fill its p-shell. This explains the variety of permutations and combinations of carbon bonding and the ability to form an almost infinite number of organic compounds.

Table 1: Oxygen Fall-off from Inspired Air to the Mitochondria



the role of oxygen in wound healing. A gradient exists from inspired oxygen tensions of 160 mmHg while breathing air to 0.5 mmHg in the mitochondria (Table 1). This is a 99.7 percent decrease in oxygen tensions from the start to the end of the gradient. Without the 0.5 mmHg oxygen tensions in the mitochondria, energy for cell metabolism will not occur. Consequently, any interference in the oxygen tension at each step of the gradient can be detrimental to mitochondrial function. Clinical conditions that ultimately interfere with oxygen delivery to the mitochondria occur at each level of the gradient (Table 2).

From the wound healing consideration, the tissue fluid oxygen tension is the one that is so critical for healing. Hunt (1969) confirmed that in order for fibroblasts to function and for wounds to heal, the tissue oxygen tension needs to be in the 30-40 mmHg range (Figure 1).[2] Healing is unlikely to occur below 30 mmH; above 40 mmHg healing is likely. Failure for wounds to heal with oxygen tensions 40 mmHg or greater indicate that other potential causes of non-healing, such as bioburden, deformity, inadequate protection/stabilization, malnutrition, cicatrix/bursa barriers to angiogenesis, matrix metalloproteins, and/or inadequate protection/stabilization, are also present.

It is remarkable that oxygen and its compounds carbon dioxide and water are the critical substances that maintain the higher forms of life on Earth. While oxygen is necessary for generating energy through ATP (adenosine triphosphate) as is needed for all cellular processes in higher organisms, its waste product is carbon dioxide.

It is almost mind-boggling that carbon dioxide, water, and sunlight are the essential ingredient of photosynthesis, and the end products of photosynthesis are oxygen and glucose. A structure somewhat analogous to the mitochondrion, the chloroplast, a member of the plasmid family, is responsible for this remarkable conversion. Chloroplasts are the energy generating organelles of plants and generate energy by chemiosmotic mechanism similar to mitochondria. Consequently, while oxygen is essential for animal metabolism, carbon dioxide is essential for plants. Together, they help maintain the Earth's oxygen/carbon dioxide atmosphere in balance.

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Table 2: Conditions that Interfere with Oxygen Availability to the Mitochondria

Tissue Level	Conditions
Lungs	Ventilation-perfusion inequalities, obstructive lung disease
Arteries	Atherosclerosis, vasoconstriction, shunting, anemia
Capillaries/Red Blood Cells	Thickening of the basement membrane, sludging, hemoglobinopathies
Tissue fluids	Relative barriers; e.g., edema, cicatrix, bursa, exudates
Cells	Neoplasms

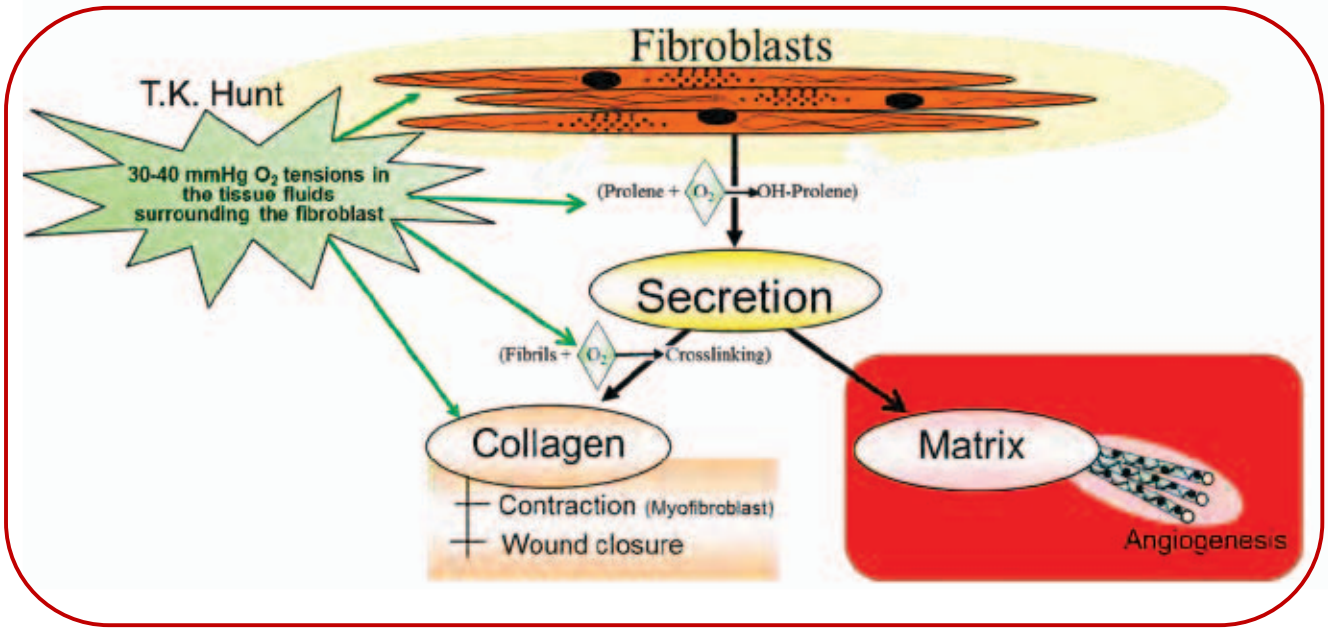
Oxygen, the "elixir of life," is not only required for wound healing—its intracellular tensions and radicals generated by biochemical reactions are an integral part of many other cellular processes. These range from maintaining consciousness to initiating angiogenesis and from intermediary metabolism (Krebs cycle) to white blood cell oxidative killing. For example, during the oxidative burst in the neutrophil phagocytic vesicle, oxygen consumption increases 100-fold or more to kill bacteria through the generation of superoxides and peroxides.[3,4] Oxygen is carried to every cell in the body by the bloodstream through a "loose" attachment to the iron in the hemoglobin molecule. With high oxygen tensions, as in the lungs, oxygen attaches to the hemoglobin molecule. In the tissues, oxygen diffuses through the capillary to the lower oxygen tension tissue fluids and into the cell. The physiology is explained by the Henderson-Hasselbalch equation and the biochemistry by oxygen attachment to iron to convert it to the ferric state in the oxygen-rich lung environment and release of oxygen and conversion to the ferrous state because of the lower oxygen tension tissue fluids.

As the "elixir...of death," oxygen, when not physiologically regulated, is harmful and can kill every cell in the body. Obviously, too low oxygen tensions result in interference with cell function, which has important ramifications for wound healing. With low oxygen tensions, the cell goes into a state of suspended animation (hibernation). While not dead, it is not functioning at a level where it can continue its physiological functions. For the fibroblast, this means the arrest of wound healing; however, with restoration of adequate oxygen tensions it can recover these functions. This, of course, is the justification for revascularization and hyperbaric oxygen therapy (discussed in the next section). Oxygen tensions below a certain point for a sustained period of time result in cell death and no chance of return of function. A big question is whether oxygen utilization for metabolism, analogous to combustion, is ultimately responsible for the cell's death just as after the consumption of all combustible material, the fire ceases to burn. Cells are programmed to die, i.e., apoptosis, at specific times. Is this because oxidation eventually "burns out" every cell in the body and results in the death of the organism?

Oxygen percentages over 17 percent are required for a fire to burn regardless of the oxygen tension. This contrasts to the extraction of oxygen from inhaled air in the lungs, where the partial pressure of oxygen (reflected in the number of molecules breathed) is essential for adequate oxygenation of tissues. The following examples further illustrate this chemistry and physiology.

In 1967 three astronauts were cremated when the space module (which was on the ground at the time) was pressurized with pure oxygen at 0.4 ATA (atmospheres absolute), and a spark set off an explosion from the combusti-

Figure 1: Oxygen Tensions Necessary for Wound Healing



Legend: Oxygen is required for fibroblasts to elaborate their functions of secretion and collagen formation. If deficient, the fibroblast may remain viable but not function. For angiogenesis, a matrix needs to be generated by the fibroblast so capillary budding can grown into it and advance the blood supply.

ble materials in the capsule. Not only did the combustible articles burn in the pure oxygen environment, they were explosive even though the partial pressure of oxygen was 0.4 of an atmosphere. The reason for using hypobaric pure oxygen was to reduce the additional weight of the 80 percent nitrogen contained in air.

In contrast, for diving studies at a thousand-foot depth, 1 percent oxygen is breathed while the remainder of the gas mixture is helium with a little added nitrogen At this depth the partial pressure of oxygen is more than 160 mmHg—sufficient to meet the lungs' requirement for ventilation. Conversely, it would not be possible to kindle a flame in this 1 percent oxygen mixture.

Things to Know about Hyperbaric Oxygen

Too much oxygen is likewise harmful, the other aspect of the "elixir...of death." Sustained periods of breathing 100 percent oxygen can cause pulmonary edema. Hyper-physiological doses of oxygen, as achieved with hyperbaric oxygen, are toxic to every cell in the body. The target tissue for this side effect is the brain, and the consequence of this toxic insult is the oxygen-induced seizure. Intermittent exposures and air breaks while breathing HBO mitigate this side effect. Why seizures occur with increased oxygen tensions of the brain is not clear. Some attribute it to hyper-metabolism just as increased oxygen percentages cause a fire to burn more vigorously. Another idea (which will be further elaborated later) is that the scavengers of the reactive oxygen species are overwhelmed by the free radicals generated by the hyperbaric oxygen exposure.

The mechanisms of hyperbaric oxygen are supported by physics and physiology.[5,6] Hyperbaric oxygen at 2 ATA (33 FSW) increases the inhaled oxygen partial pressure tenfold from 160 mmHg partial pressure of oxygen to over 1600 mmHg. This increases the oxygen diffused from the alveolus to the plasma of the alveolar capillary tenfold but does not change the hemoglobin-carried oxygen of the red blood cell since in the absence of lung disease and/or red blood cell diseases, it would already be approaching 100 percent.

In room air, 97.5 percent of the oxygen in the blood is carried by the hemoglobin in the red blood cell and

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2.5 percent is physically dissolved in plasma. With hyperbaric oxygen, the tenfold increase in plasma oxygen adds 25 percent to the blood's oxygen carrying capacity. All oxygen delivery (as well as nutrients) to cells, be it from hemoglobin or physically dissolved in the plasma, must first diffuse through the capillary, then through tissue fluids, to the cell. The oxygen diffusion is in response to gradients, which are high in the blood, lower in the tissue fluids, and lowest in the cell. With HBO the tenfold increase in plasma oxygen content supplements the hemoglobin-carried oxygen and mitigates conditions where blood flow, hemoglobin-carried oxygen, or diffusion distance problems interfere with oxygen delivery to the cell (Figure 2). Under HBO conditions enough oxygen is physically dissolved in the tissue fluids to meet cellular oxygen requirements in the absence of hemoglobin-carried oxygen.[7]

Another method of increasing the partial pressures of oxygen above physiological levels is through SCUBA diving. SCUBA diving to 100 feet of sea water (FSW) increases the oxygen partial pressure of the inhaled air fourfold, which is nearly equivalent to breathing pure oxygen at sea level. While this is tolerated for the relatively short durations of the SCUBA dive, saturation diving (in an underwater habitat) at 100 FSW requires reducing the oxygen percentages of the breathing gas in order to prevent oxygen toxicity.

With closed circuit rebreathers the likelihood of oxygen toxicity, especially seizures, is much greater. When breathing pure oxygen in a rebreather unit, depth and time durations are strictly limited. For example, a 30 FSW dive is limited to 30 minutes, while shallower dives can have longer durations.

Mixed-gas closed circuit rebreathers are designed to provide a constant partial pressure of oxygen regardless of the depth. To accommodate the increased ambient pressures with descent, increased percentages of the diluent gas are added to the breathing loop. Seizures may occur from errors in setting the oxygen partial pressures. In addition, the breathing mixture may be switched to pure oxygen near the surface to hasten off-gassing of the inert gas. However, if done prematurely, for example at a depth greater than 30 FSW, an oxygen seizure may occur.

In 1959 Boerema et al. demonstrated that piglets who had their red blood cells removed could be kept alive and functioning for brief periods (15 minutes) with physically dissolved oxygen in their plasma.[7] The end-point of the study was carbon dioxide accumulation rather than oxygen deficiency.

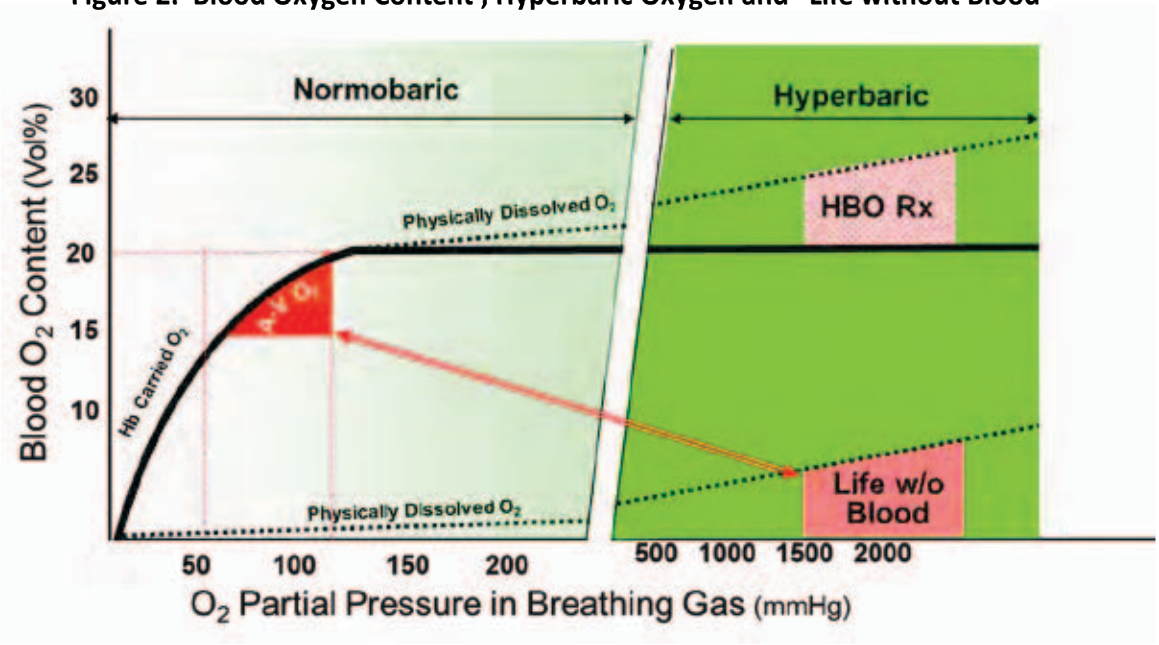
This study dispelled the "Haldane hex," which contended that cells could only utilize oxygen that was hemoglobin borne. Boerema's study gave the use of hyperbaric oxygen a solid physiological basis and ushered in the modern area of HBO therapy. For this seminal contribution, we refer to Dr. Boerema as the father of hyperbaric medicine.

Oxygen diffuses through relative barriers poorly, especially as compared to carbon dioxide. Carbon dioxide's ability to diffuse through tissue fluid is 20-times greater than that of oxygen. Commonly encountered relative barriers include atherosclerotic vessels, which interfere with perfusion; thickened capillary membranes, which slow diffusion through the endothelium; edema fluid, which increases the diffusion distance from the capillary to the cell; and cicatrix, which acts as an obstruction (Table 2). The tenfold increase in tissue fluids achieved with HBO promotes oxygen tissue diffusion through these barriers, which are considered relative because they can vary in extent from inconsequential to totally obstructing oxygen availability to the cell.

An example of a relative barrier is that of edema associated with stasis ulcers. The more severe the edema, the more likely a stasis ulcer will develop. The pathophysiology of the ulcer etiology can be multifactorial such as from trauma, venous stasis disease, atrophic/friable skin, loss of skin elasticity with aging, hemosiderin deposition in the subcutaneous tissues, cicatrix formation from ischemia/hypoxia of the underlying tissues, and/or insufficient perfusion to allow healing.

Regardless of the cause, a primary intervention in managing the stasis ulcer is that of reducing edema through use of elastic wraps, elastic support hose, leg compression pumps, and/or

Figure 2: Blood Oxygen Content , Hyperbaric Oxygen and "Life without Blood"



Legend: The physically dissolved oxygen from the hyperbaric oxygen exposure adds to the hemoglobin-carried oxygen. Once the hemoglobin-carried oxygen becomes fully saturated it cannot carry additional oxygen. At about a 2000 mmHg oxygen partial pressure there is enough oxygen content in the plasma to meet oxygenation requirements without hemoglobin-carried oxygen. This was demonstrated by Boerema's "Life without Blood" experiment.

Key: **A-V O_2 =** arterial-venous oxygen extraction, **HBO** = hyperbaric oxygen, **Rx** = treatment; **w/o** = without

diuretics. The reduction of edema reduces the distance oxygen has to diffuse through tissue fluids to reach the ulcer wound bed.

While everyone attests to the benefit of edema reduction in managing stasis ulcers, the beneficial role of reducing oxygen diffusion distance through tissue fluids to improve oxygen delivery to the ulcer and promote healing must not be overlooked.

In our experience, there are three fundamental reasons why wounds, especially diabetic foot ulcers, do not heal in the usual and customary fashion.[8] These are failure to address adequately the underlying deformity; persistence of deep infection of bone, cicatrix, and/or bursa; and ischemia/hypoxia. Of the three, the confirmation of ischemia/hypoxia is perhaps the easiest. This can be ascertained with the clinical exam (e.g., palpable pulses, Doppler pulses, skin coloration and temperature, and capillary refill time), imaging studies, and juxta-wound transcutaneous oxygen measurements (TCOMs). As mentioned before, tissue fluid oxygen tensions in the 30-40 mmHg range are needed for wounds to heal.[2] Juxta-wound TCOMs measure and reflect the tissue fluid oxygen tensions and can be used as a guide to determine whether the ischemic/hypoxic wound will heal or if measures to increase perfusion/oxygenation are needed.

Hyperbaric oxygen is an intervention to increase wound oxygenation (in addition to revascularization, edema reduction, improved cardiac function, and pharmacological agents).[9] The value of using TCOMs to predict healing with use of HBO as an adjunct to manage wound hypoxia and achieve healing is established. We reported that hypoxic wounds (that is, wounds with juxta-wound TCOM levels in room air of less than 40 mmHg) heal in 87 percent of cases if the TCOMs increase to 200 mmHg or greater with HBO exposure and HBO treatments are subsequently used in wound management.[12] This information using TCOMs objectifies the indications for HBO in problem wounds.

Hyperbaric oxygen has applications to many other medical conditions in addition to hypoxic wounds. The indications for using HBO in other conditions

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The origin of the 200 mmHg oxygen tension with HBO for predicting healing of the hypoxic wound is attributed to Dr. George B. Hart. In the 1990s TCOMs became available but predictions for healing with HBO ranged from 300 to 900 mmHg oxygen tensions.

In the late 1990s Dr. Hart was queried as to what juxta-wound TCOM value was needed for healing to occur with HBO treatments. From his keen observations he gave the number of 200 mmHg. In the 1997 and 1998 Annual Undersea and Hyperbaric Medical Society meetings we presented posters from our observations demonstrating the validity of the 200 mmHg number in increasing series of patients.[11,12] This work culminated in our 2002 *Foot & Ankle International* prospective peer reviewed publication with a study group of 82 patients who had TCOMs less than 30 mmHg in room air.[12]

In reviewing the history of the predictive value of the 200 mmHg number for healing of the hypoxic wound with HBO, we found that this value was also used in a paper by Fife et al. in 2002.[12] In their review of over 1100 patients with many permutations such as using 100 percent surface oxygen, leg elevations tests, etc., it was unclear how they derived the 200 mmHg number stated in their conclusions.

are based on its mechanisms. We divide the mechanisms into primary and secondary.[5,6] The primary mechanisms hyperoxygenation and pressurization (to reduce bubble size) are immediate and act in a drug dose-duration fashion. Applications in addition to decompression sickness and arterial gas embolism include threatened flaps, acute blood loss anemia, acute peripheral ischemia, burns, crush injuries and compartment syndromes, and central retinal artery occlusions. Secondary mechanisms occur as a result of hyperoxygenation and pressurization acting on body tissues and microorganisms. In contrast to the dose-duration effects of the primary mechanisms, the effects of the secondary mechanisms tend to be additive and require repetitive HBO treatments. They include edema reduction, stimulation of host healing responses (including fibroblast function and angiogenesis), gas washout (for carbon monoxide poisoning and decompression sickness), reperfusion injury, delayed radiation damage of soft tissue and bone, cerebral abscess, refractory osteomyelitis, gas gangrene, and necrotizing soft tissue infections. Awareness of the mechanisms of HBO helps justify its use. In addition, mechanisms of HBO may have applications to current off-label uses of HBO such as acute brain and spinal cord events, sports injuries, osteonecrosis, and fracture healing.

Things to Know about Free Radicals and Hyperbaric Oxygen

For decades researchers have known that highly reactive molecules called free radicals cause aging by damaging the DNA (deoxyribonucleic acid) of cells and thus disturbing the carefully regulated functions of tissues and organs. Free radical formation is an integral part of intermediary metabolism (Krebs cycle) and, with glucose plus oxygen, provides the energy for cell metabolism, cell/tissue function, and generation of cell products. In addition, free radicals generated by the phagosomes in the neutrophil kill bacteria. However, reactive oxygen species in the wrong place or in super-physiological numbers damage tissues. This is observed with radiation injury and ischemia-reperfusion injury. There is increasing recognition that cardiovascular diseases, neurodegenerative diseases such as Alzheimer's and chronic inflammation, apoptosis, and necrosis have oxidative stress components.

With evolution, cells have generated antioxidants (oxygen radical scavengers) to mitigate oxidative stresses. This phenomenon is so important that Lane asserts that life would not have evolved to its present form without the generation of antioxidants to "tame" the highly reactive oxygen radicals it generates.[1] Defenses to mitigate the oxidative stresses include vesicles such as the mitochondrion shell, which contains the reactive oxygen species as glucose is metabolized to generate energy, and allows the train of reactions to proceed in a regulated fashion. The other mechanism to handle reactive oxygen species is the generation of antioxidants such as glutathione peroxidase and superoxide dismutase.

While HBO is believed to generate reactive oxygen species, oxygen is required for the generation of

oxygen radical scavengers.[14] Consequently, in the hypoxic environment insufficient oxygen to generate the oxygen radical scavengers may cause damage such as cell death and tissue necrosis, which is always of concern in problem wounds. Much is known about oxygen radical scavengers/antioxidants, oxygen transporters, and reactive oxygen species.[15] For example, 84 genes in the human genome are related to oxygen stresses.

A number of vitamins have been promoted as antioxidants and purported to be useful in preventing aging such as vitamins E, C, and A. Vitamin E in particular was promoted for this purpose. It also had been used in conjunction with HBO treatments to prevent oxygen seizures.

Vitamin E is no longer recommended for preventing seizures with HBO, because seizure rates are so low with clinical HBO treatments that they are almost a non-occurrence. When a seizure does occur, it is usually associated with hypoglycemia in the diabetic patient or non-therapeutic doses of anticonvulsants in the patient with a seizure history.

Reactive oxygen species may have salubrious effects with respect to longevity and disease prevention. There is increasing appreciation of noncoding DNA sequences (epigenes, or "junk" genes) as being able to influence genetic information through the expression of DNA. The physiological reactive oxygen species generated by HBO could be one of the "missing links" in our understanding of how they are beneficial to the organism. Reactive oxygen species may remove damaged DNA segments that lead to aging, diseases, neoplasms, or prevent wound healing.

A "take home" observation of this is seen in the exuberant callus formation that recurs debridement after debridement, especially in the diabetic foot ulcer. Even after the wound heals, the exuberant callus returns as if the message system to form callus persists, suggesting that epigenes have influenced the DNA associated with callus formation.

Animal studies have shown that longevity is increased in animals genetically altered and missing antioxidant enzymes.[16] In addition, those animals that overproduced superoxides lived 32 percent longer than the controls. The longest living rodent, the naked mole rat, is able to survive 25 to 30 years, has lower levels of antioxidants than similar sized rodents, and remains disease free eight times longer. The hypothesis to explain this observation was that the mole rat accumulates more oxidative damage to their tissues at an earlier age, so only the most healthy individuals survive. A herbicide that generates free radicals resulted in worms living 58 percent longer than untreated animals.

This may support the "survival of the fitness" concept, that the young organism should be exposed to a constellation of diseases to "train" their immune systems. Epigenes may influence DNA to generate the antibodies, etc., and free radicals may "turn on" the epigenes so at a young age the organism becomes immunologically privileged, ensuring the longest possible survival.

Conclusions

Oxygen is a remarkable molecule; too little is lethal and too much is lethal. Organisms have generated remarkable mechanisms to maintain oxygen in physiological settings. Hyperbaric oxygen may alter these protective responses on one hand and on the other may make them more effective. The role of reactive oxygen in wound healing has hardly been addressed, but much is known about its role in killing bacteria. We are not quite ready to recommend sleeping in the pressurized HBO chamber, but if HBO modestly generates reactive oxygen species to turn on epigenes to influence DNA messaging which, in turn, mitigate disease processes, many new roles for HBO can be expected.

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