

# Wound Care & Hyperbaric Medicine magazine

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**MATRIX**



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## Note from the Editor

There had been much discussion this November about diabetes mellitus, and with good reason. Nearly 30 million people in America alone have diabetes, a debilitating disease with complications that include blindness, cardiovascular disease, stroke, and lower-extremity wounds and amputations. An increasingly sedentary lifestyle, poor food choices, and stress are factors that leave many individuals vulnerable to developing diabetes, and it is projected that its prevalence will continue to rise worldwide. The direct and indirect costs related to diabetes are also expected to rise; in the US in 2012 this number was estimated at a whopping \$245 billion. This also means we can expect an increase in diabetic foot ulcers (DFUs) requiring treatment in wound care and hyperbaric centers.<sup>1</sup>

In support of Diabetes Awareness Month, Best Publishing Company teamed up with Wound Care Education Partners to create a 21-Day Diabetes and Wound Care Challenge. The educational challenge was designed to help practitioners sharpen their skills and expand their knowledge of wound management for patients with diabetes, and foster thought and discussion about its clinical implications. We are continuing this important discussion about diabetes in this issue of *WCHM*, where we've asked a few of our authors to weigh in on issues of diabetes prevention and management.

We welcome Dr. Ryan Fitzgerald, who has submitted a case study on limb salvage and the interdisciplinary diabetic assessment rapid response treatment team. Darren Mazza looks at the safety implications surrounding diabetic patients undergoing hyperbaric treatments, and Gretchen Dixon has outlined some ways in which individuals can make healthy life choices that will offset the risk of diabetes development.

We also welcome Dr. Keith Van Meter, who has submitted an article outlining the use of hyperbaric therapy for the approved indication of severe anemia, and Dr. Strauss et al. continue with their assessment of diving stresses in Part IV of this series, which focuses on the "no panic" syndromes.

Also included is this issue's Clinic In Focus, the Center for Wound Care and Hyperbaric Medicine at Springhill Medical Center in Mobile, Alabama. 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 revamped Map of Wound Care and HBO Centers. Take advantage of this free resource and make your clinic easy to find for prospective clients and referral physicians.

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

1. Centers for Disease Control and Prevention. National Diabetes Statistics Report: Estimates of Diabetes and Its Burden in the United States, 2014. Atlanta, GA: U.S. Department of Health and Human Services; 2014.

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# Wound Care & Hyperbaric Medicine

## Now is a Good Time to Change Your Lifestyle

Gretchen Dixon, MBA, CCS, CPCO

Diabetes ranks in the top five chronic medical conditions we in the United States may develop over our lifetime; however, there are life-altering practices we can do daily to decrease our chances of developing this disease. Diabetes is a serious medical condition that affects people young and old. Now is a good time to think about the ramifications of this medical condition and what we can do to reduce our risk of developing it.

Is it possible you may develop diabetes? YES! The Centers for Disease Control and Prevention's National Diabetes Statistics Report of 2014 documents the following 2012 statistics<sup>3</sup> for all ages:

- Diagnosed with diabetes = 21.0 million people
- Undiagnosed as having diabetes = 8.1 million, or approximately 28% of people with diabetes have not yet been diagnosed
- The total = over 29 million people, or more than 9% of our population, have diabetes

### Why is This a Concern?

Diabetes is a chronic medical condition that, if left undiagnosed, untreated, or poorly controlled, is the leading cause of kidney failure, lower limb amputations, and blindness among adults. And there is a cost ratio that affects each person's lifestyle. The total cost of diagnosed diabetes in 2012 was \$245 billion, broken out as \$176 billion in direct medical care costs (doctor's appointments, medications, complications, sickness) and \$69 billion in a decrease in work productivity, which includes missing work, less productivity at work, or not able to work at all.

Did you know that 37% of adults ages 20 years and older are considered pre-diabetic? This percentage then increases to 51% for adults 65 years and older. Why does this happen?

It is a result of our daily lifestyle and dietary habits, from our fast and processed food as sources of nutri-

tion to our sedentary activities to stress and a lack of adequate sleep.

### Simple Steps to Take Control of Your Life

Let's be proactive by being aware that we have to start making better lifestyle decisions. Where do we start?

#### 1. Sleep

It's more important than you know, yet we never seem to be able to "catch-up" on missed sleep. If we think of the human body as a super computer, it needs 8 hours of total shutdown to regenerate and fix all of its programs and functions down to the cellular level. Each component, including cells, organs, and hormones, requires this reboot to carry on the internal teamwork needed for us to function like we do each day.

**Keep a schedule** —just like going to work on a schedule, practice going to bed and waking up at the same time every day. Your body will adjust rather quickly, and you will begin to feel less tired and actually have more energy for everyday activities. Your habit of falling asleep on schedule won't happen overnight, but don't give up. **WARNING:** Don't change your wake up times for the weekend; keep to your schedule. Be prepared to keep your bedtime a ritual just like you would for children—start the downtime about 30 minutes before your actual bedtime.

**Falling asleep in your bed** means turning off all electronics, including the TV, iPad, mobile phone, or any other device. As with all activities we have goals; when in bed at night the goal is to fall asleep. This is a component of training your body to slow down and regroup. It is ok to read since reading for some people is a way to wind down after a hectic day. Just keep the reading to no more than 15 minutes. Plan to keep the room cool and dark, and reduce the amount of light, sound, and other distractions that may stimulate your mind.

## 2. Exercise Daily

This is a cheap way to reap benefits only for you. Starting early in the morning each day will increase your metabolism, resulting in higher energy for your daily activities. Walking or riding a bike at a moderate pace is a great way to increase your heart rate and respiration, thus oxygenating all your cells to help your body optimize its work in keeping you healthy. Get a buddy to walk with each morning and make it a daily habit. Sure it will be slow starting, but the idea is to increase your endurance as you increase your distance. I call my morning walk/run with my dog my “natural mojo” because it increases my energy for the day. In the beginning I would sometimes skip a day or two (or three) and feel guilty, so I reminded myself that I was doing this for my own benefit and had better stick to my routine. The cost for me is to wake up at 5:15 am and get out the door by 5:30 am. You can start with a brisk 30-minute walk for the first two weeks, then increase your time and distance by 15 minutes every two weeks until you build up to a 60-minute walk. If you can’t walk every day, set a goal of every other day as a minimum. Remember, you are doing this for YOU!

It is great to be exercising outdoors in the fall and springtime and hard to stick to your routine in the summer and winter months. Remember that 30 minutes is not an eternity, and we all can find empty time (non-productive activities) to go for a walk. I think of non-productive activities as playing games on a phone or iPad, answering emails, surfing the web, or watching TV for hours, all of which have led to our sedentary lifestyles. These activities are causing our bodies to become weak and less efficient, and the resulting costs are high! SO GET OUT OF BED or OFF THE COUCH and go for a walk in your neighborhood. You will be surprised by what you can do on your own to improve your health.

### Benefits of Exercise

- An invigorating day results in a better night’s sleep.
- Your body will become more sensitive to insulin; however, this effect is only temporary.
- There is NO benefit from trying a weekend catch-up for missed exercising days.

## 3. Get Some Sun

Sunlight brings happiness when we see it and feel its warmth. It also provides vitamin D, which is absorbed through our skin. There are a plethora of articles from around the world on how sunshine may be related to the slowing of the development of obesity and the prevention of diabetes.

## 4. Reduce Stress

Be calm since stress causes an increase in the hormone cortisol, which affects the insulin resistance in the body leading to diabetes. What is insulin resistance?

First, the purpose of the hormone insulin is to regulate the delivery of glucose into our cells to provide them with energy. Insulin resistance occurs when our cells cannot use insulin as effectively, resulting in high glucose/blood sugar levels or hyperglycemia. Other organs involved in this process, such as the pancreas, use their cells to increase insulin production, which results in a domino effect of high blood sugar/ glucose levels.

**How to minimize stress**—We all experience stress at times in our lives, but managing it is sometimes difficult. Here are a few easy ways to engage control over stress, which can be used in any lifestyle or at any activity.

*Focus on the present* through meditation, which only takes a couple of minutes.

- Correct your sitting posture—sit up straight with both feet on the floor
- Close your eyes and focus on a positive thought, like walking on the beach and hearing the waves and seagulls
- Set your phone on a timer, with soothing music to signify when your time is up

*Focus on deep breathing* during a 2-minute break. Breathe deeply through your nose, pulling air in into your lower abdomen and working your diaphragm. Hold the breath for a few seconds and slowly exhale, pushing your diaphragm as far up towards your lungs and ridding your lungs of impure air. Oh this feels so good.

*Tune into your body* by being aware of those situations or places that make you feel uncomfortable, without trying to change anything. Take a deep breath and focus on the task at hand.

*Reach out* to your inner circle of friends to share thoughts that are causing stress and receive their different perspectives on the situation. Talk, cry, laugh, and express anger as these are healthy expressions and emotions that will help to reduce your stress level.

## 5. Healthy Eating

Eating healthy may cost more at the grocery store; however, the cost of your health care tomorrow, in five years, and in ten years can be beyond your wildest imagination. Cost is not just money; it is how drastically diabetes can affect and change a person’s lifestyle from independence to dependence if it is not controlled.



- AVOID red meat, white bread, and fried foods.
- Grocery Shopping: When you go to the grocery store think about only shopping the outer aisles for fresh fruits and vegetables, fish, legumes, nuts, and whole grains.

## 6. Blood Sugar Level Awareness

Knowing blood sugar numbers is important to help you stay healthy. Keeping your blood sugar numbers close to the normal range reduces diabetes-related health risk factors like eye diseases, circulation problems, heart attacks, kidney failure, and nerve damage, especially in your feet. Two of the most common methods to test your blood sugar levels are either with a self-monitoring blood glucose device or with the A1C test performed at your doctor's office.<sup>5</sup>

The most common times and ranges for self-testing at home are:

Before meals	After meals
90-130	Less than 180

The A1C test identifies how your blood glucose averages over the past 3-month period with a goal of being normal at level of 6 or less. Any reading over 7 requires action to be taken as determined by your physician. **Fact: the A1C level does correlate with your blood glucose self-test results as follows in this chart.**

A1C Level Numbers	Average Self-test Glucose Numbers
6	135
7	170
8	205
9	240
10	275
11	310
12	345

We need to recognize this important factor in making lifestyle changes happen: We are the conduits of change, and we must hold ourselves accountable to make the necessary changes. There are no magic pills or foods that will cure diabetes, but by using our knowledge we can make a change in ourselves today so we may be able to enjoy what tomorrow brings.

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**Gretchen Dixon** is the owner of Professional Compliance Strategies LLC and consults on outpatient departments and physician services. She provides revenue cycle compliance reviews of services with the focus on wound care department operations for over 9 years. She holds several credentials: MBA in Healthcare Management, Registered Nurse with practicing licenses in NY and a multi-state license from Virginia, AHIMA Certified Coding Specialist (CCS), AAPC Certified Professional Compliance Officer (CPCO), and is an AHIMA Approved ICD-10-CM/PCS Trainer/Ambassador. Being a longtime internal healthcare compliance auditor, she identifies issues through audits of D, C, Bs (documentation, coding & billing) of provided services. The outcomes of each audit determines the topics of education to be provided to staff and physicians as she believes education is the KEY to having accurate, complete and consistent documentation for accurate reimbursement of billed services. If you are interested in the services offered, contact her at [gmdixon@cox.net](mailto:gmdixon@cox.net) or 615 210 7476.



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# Wound Care & Hyperbaric Medicine

## Stresses in SCUBA and Breath-Hold Diving Part IV: The No Panic Syndromes

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

### Introduction

In the three previous issues of *Wound Care & Hyperbaric Medicine* we introduced the subject of stimulus/stress—response/resolution and used this as the basis for discussing the physical, physiological, and psychological stresses of diving.<sup>1-3</sup> In this issue we discuss a different presentation of diving stresses and a group of stresses that are almost the antithesis of the psychological stresses. These are the stresses that lead to sudden and unrecognized scenarios that can cause loss of consciousness, and for this reason we label them the “no panic” syndromes (NPS).<sup>4</sup>

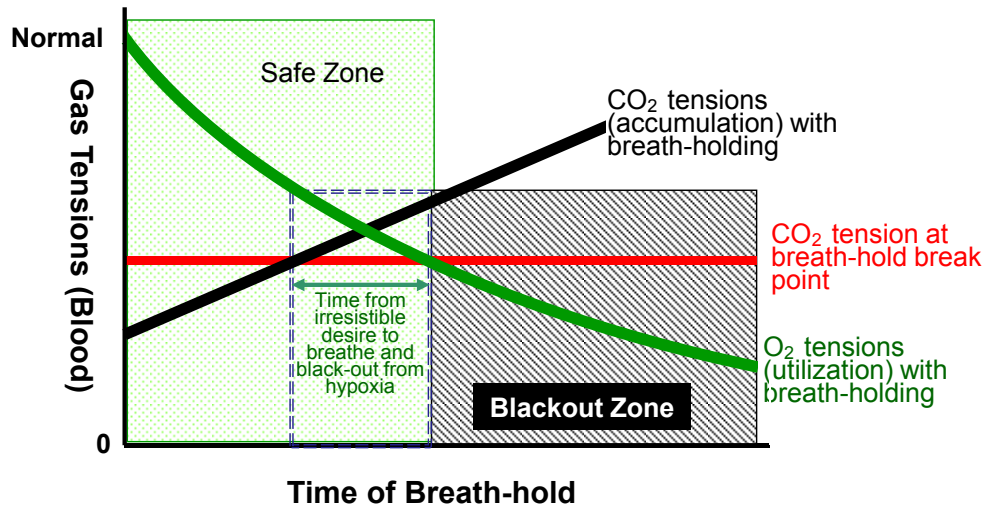
The NPS represent stimuli, primarily low oxygen tensions in the bloodstream and insufficient oxygenation of the brain, with the responses being blunted or totally obscured by the loss of consciousness. Even though a diver may breath-hold for a minute or longer, a moment's interruption of oxygen availability to the brain will lead to loss of consciousness. This can occur so suddenly that the usual warning symptoms of hypoxia typically signaled by the stimulus-response mechanism of elevated carbon dioxide tensions do not manifest themselves. The consequence is that the diver loses consciousness before corrective actions are initiated. Thus, this is the reason for labeling this group of medical problems of diving the NPS.

The consequences of a momentary interruption in the brain's oxygen supply leads to loss of consciousness. Because of the extraordinarily high oxygen consumption of the higher function neurons (i.e., consciousness and cognitive function centers) of the brain, a one or two second interruption in oxygenation of these cells interrupts their metabolism enough that they stop functioning properly and consciousness is lost. The cells themselves may remain viable for three or four minutes before the anoxic insult leads to irreparable damage to the brain cells. Remember with breath-holding, blood is still flowing to the brain with resultant oxygenation of the brain during this activity.

Several clinical observations substantiate the above information. First, with the “choke hold,” the blood supply to the brain is interrupted. This results in immediate loss of consciousness. Similarly, with third degree heart block, the lag in ventricular contraction before the idioventricular heart pacemaker takes over sometimes causes instantaneous syncope.<sup>5</sup> Third, after extreme hyperventilation (to the point the fingers and toes develop paresthesias), a forceful Valsalva maneuver leads to unconsciousness. The physiology to explain this is hyperventilation lowers carbon dioxide tensions. It appears that the hypocapnia causes activation/excitability of sensory and muscle neurons. This causes the paresthesias and may even lead to tetany.<sup>5</sup> In addition, cerebral blood flow is reduced 30 to 40 percent so with each heart beat there is less oxygen getting to the brain.<sup>6</sup> The forceful Valsalva maneuver impairs cardiac filling and thereby lowers cardiac output and oxygen tension enough (to the already reduced blood flow to the higher centers of the brain) that consciousness is lost.

The underlying pathophysiology is due to blunting or elimination of the air hunger stimulus. There are more than a dozen reasons loss of consciousness occurs in the diver before the usual warning systems alert the diver to the impending danger. In contrast to many of the other medical problems of diving where inexperience can often be a contributing factor, the NPS are more likely to occur in the experienced diver. Also, there is a propensity for these problems to occur with breath-hold and special equipment diving in contrast to open circuit SCUBA diving. Finally, any loss of consciousness in water is an extremely serious event, with drowning being the consequence without immediate rescue and resuscitation. This article examines the great variety of presentations of the NPS and describes the physiology that leads to the loss of consciousness for each.

**Figure 1: Gas Changes with Breath-holding, the Breath-hold Break Point and the Safe Interval from the Irresistible Desire to Breathe and Blackout from Hypoxia**



**Legend:** With breath-holding, oxygen (O<sub>2</sub>) is utilized while carbon dioxide (CO<sub>2</sub>) accumulates. At a certain level of CO<sub>2</sub> accumulation, an irresistible desire to breathe occurs. This is the breath-hold break point, which occurs before the O<sub>2</sub> tensions decrease to the point of blackout from hypoxia. These curves vary from person to person and with training, experience, hyperventilation and other situations.

## Alterations of Blood Oxygen and Carbon Dioxide Tensions with Breath-Holding

The physiological effects of breath-holding are well understood. The two principal considerations are the changes that occur with blood oxygen (O<sub>2</sub>) and carbon dioxide (CO<sub>2</sub>) tensions. With breath-holding, O<sub>2</sub> is consumed by cell metabolism while CO<sub>2</sub> is generated as a waste product. Oxygen content in the bloodstream decreases from 20 Vol% to 15 Vol% with normal breathing.<sup>8</sup> This 5 Vol% represents the amount of O<sub>2</sub> that diffuses through the capillary from its arterial to venous ends, diffuses through tissue fluids and then is utilized by cells for metabolism. Conversely, as a waste product of metabolism, CO<sub>2</sub> diffuses into the capillary and is carried by the bloodstream to the lungs where it is expelled with respiration. Whereas the percentage of CO<sub>2</sub> in air and inhaled with each breath is 0.05 percent, the percentage of CO<sub>2</sub> in each exhaled breath is 5 percent. This represents a hundred-fold increase and exemplifies the amount of waste product generated by metabolism.

Diving mammals as well as human divers have adaptations to maximize the efficiency of the 20 Vol% of O<sub>2</sub> that is carried in the arterial side of the bloodstream. These adaptations are collectively labeled the diving reflex. The diving reflex is a homeostatic mechanism to maximize oxygen delivery to the brain and heart when oxygen interruption is impeded or totally interrupted as with breath-holding. For organism

survival, these two critical organs must continue to function with breath-holding to maintain function and avoid death.

The three components of the diving reflex associated with breath-holding include: 1) bradycardia, the slowing of the heart rate to lessen O<sub>2</sub> requirements for this critical organ; 2) vasoconstriction to shunt blood away from non-critical organs and maintain adequate perfusion to the brain; and 3) anaerobic metabolism in the organs from which the blood supply has been shunted. As a subsidiary mechanism to ensure adequate O<sub>2</sub> availability as CO<sub>2</sub> accumulates in the bloodstream, the blood becomes more acidotic and shifts the arterial-venous capillary O<sub>2</sub> extraction curve to the right. From this effect more O<sub>2</sub> attached to the hemoglobin molecule can be released. This results in a greater than 5 Vol% extraction of oxygen from the bloodstream for the critical brain and heart organs.

With breath-holding, O<sub>2</sub> consumption continues unabated while CO<sub>2</sub> tensions increase in the bloodstream since this waste product of metabolism is no longer being expelled (Figure 1). As a component of the body's homeostasis mechanisms (as explained in reference 1), the stimulus to breathe is an indirect one. It arises principally from elevated CO<sub>2</sub> tensions in the bloodstream, which activate sensors and initiate reflexes to breathe. In normal situations, low O<sub>2</sub> tensions do not provide a significant stimulus to breathe. As CO<sub>2</sub> accumulates in the bloodstream, there is an increasing desire to breathe. Under normal circum-

**Table 1: Categories of Blackouts that Occur in Divers**

Category	Components	Comments
<b>Hypoxic</b>	<b>Breath-holding</b> after hyperventilation <b>Distractional</b> (disregard of B-H breakpoint) <b>Diffusional</b> (deep B-H dives) <b>Dilutional</b> (rebreather equipment) <b>Tank</b> (rust consumption of O <sub>2</sub> )	Except for diffusional blackouts, the blackouts in this category are due to altering the normal CO <sub>2</sub> elevation signaling mechanism to breathe
<b>Cardiogenic</b>	<b>Cardiac arrest</b> <b>Arrhythmias</b> <b>Carotogenic</b> <b>Valsalva</b>	All these conditions interfere with O <sub>2</sub> delivery to the brain mediated by heart activity
<b>Altered Level of Consciousness</b>	<b>Nitrogen narcosis</b> <b>Downer drugs/depressants</b> <b>Carbon monoxide poisoning</b> <b>CO<sub>2</sub> &amp; O<sub>2</sub> Toxicities</b> <b>Hypothermia</b>	All the components interfere with the loss of consciousness and can be additive with each other or other categories of blackout
<b>Ascent Associated</b>	<b>The chokes</b> <b>Decompression illness</b> <b>Alternobaric vertigo</b>	Associated with explosive decompressions and/or ascents
<b>Miscellaneous</b>	<b>Head injury</b> <b>Marine animal envenomations</b> <b>Hypoglycemia / seizure</b> <b>Electric shock</b>	Unusual causes of NPS, but are other considerations for loss of consciousness in divers

stances, this goes unrecognized and a breath is taken without conscious awareness. As CO<sub>2</sub> tensions elevate, eventually a point is reached where breath-holding is no longer possible. This point is termed the breath-hold breakpoint. This point varies from person to person, can be overwhelmed in certain situations and is altered by physiological mechanisms. Categories of the no panic syndromes include: **1) hypoxic, 2) cardiogenic, 3) altered awareness situations, 4) ascent associated, and 5) miscellaneous reasons** (Table 1). Each category has one or more specific causes. The remainder of this article describes each of the components of the five categories of the NPS, their pathophysiology, and the measures to prevent their occurrences.

The human body has, in fact, sensors to detect low O<sub>2</sub> tensions in the blood. These chemoreceptors are located in the carotid bodies of the carotid arteries in the neck. However, their ability to stimulate breathing is much less effective than elevated CO<sub>2</sub> tensions, which initiate strong responses to breathe through central chemoreceptors located in the brainstem.

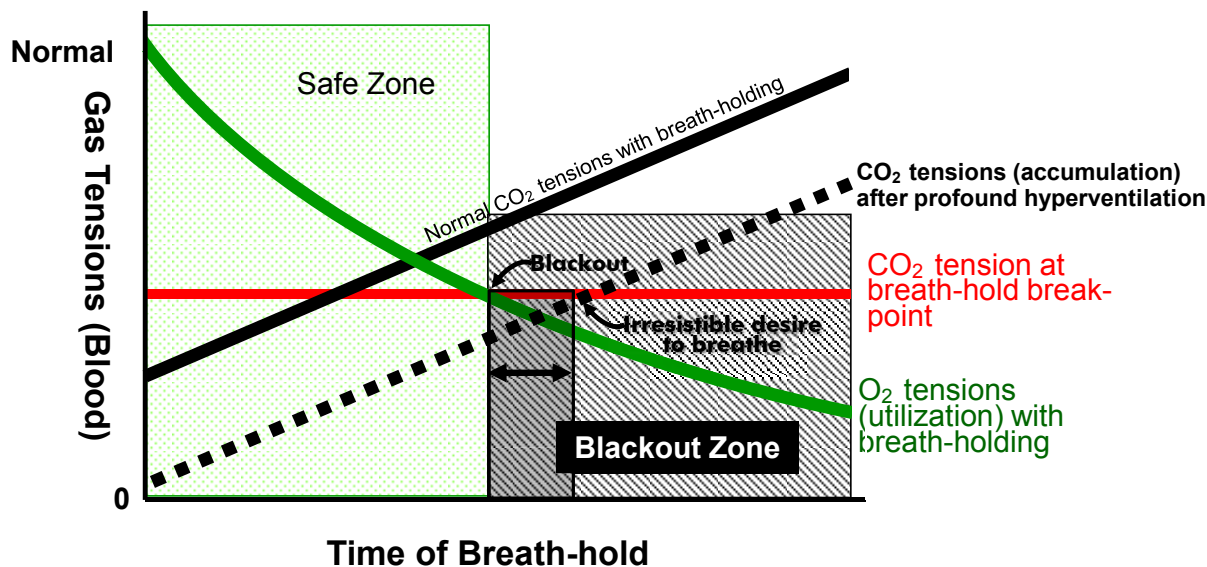
Clinical correlations are observed in divers using rebreather diving gear. If, by accident, the diver is breathing a hypoxic mixture while CO<sub>2</sub> is maintained at normal

levels by reabsorption through the scrubber, the diver will continue to breathe the hypoxic gas mixture. The diver continues to breathe at a normal rate from the low O<sub>2</sub> stimulus, but acceleration of breathing/air hunger, as seen with elevated CO<sub>2</sub> blood tensions, does not occur. Quickly, the diver loses consciousness because of lack of O<sub>2</sub> to the brain from breathing the hypoxic gas mixture.

Another example of the O<sub>2</sub> stimulus to breathe is seen in patients with advanced chronic obstructive pulmonary disease (COPD) who retain CO<sub>2</sub>. Their respiratory systems no longer respond to elevated CO<sub>2</sub> tensions in their blood; rather, their respirations are driven by low O<sub>2</sub> tensions in the blood. Treating these patients with hyperbaric oxygen (HBO) then becomes somewhat problematic. Breathing may cease, especially if the patient falls asleep, due to the high O<sub>2</sub> tensions that occur during the HBO exposure. This is because these patients' primary stimulus to breathe is hypoxia. When patients with COPD have HBO treatments, they need to remain awake and constantly monitored and told to breathe in order to ensure adequate ventilation.

**Part IV: The No Panic Syndromes\***  
\*Parts I, II, and III, Physical, Physiological, and Psychological Stresses of Diving were published in the three previous issues of *Wound Care & Hyperbaric Medicine*.<sup>1-3</sup>

**Figure 2: Hyperventilation, Breath-Holding Blackout**



**Legend:** Profound hyperventilation markedly lowers the CO<sub>2</sub> tensions in the blood whereas O<sub>2</sub> utilization remains relatively constant. This can delay the breath-hold breakpoint time beyond the time the diver will blackout from hypoxia as indicated by " ↔ " in the darker cross-hatched rectangle.

## Section A: Hypoxic Blackouts

Hypoxic blackouts occur due to mechanisms that alter carbon dioxide (CO<sub>2</sub>) tensions. The normal stimulus to breathe from elevated CO<sub>2</sub> tensions and physiological blood oxygen (O<sub>2</sub>) tensions is mitigated or totally absent in the hypoxic blackouts except for the diffusional category. When blood O<sub>2</sub> tensions that normally decrease with O<sub>2</sub> utilization for metabolism reach a critical point, consciousness is lost. Five types of hypoxic blackout occur. Three are associated with breath-hold diving (hypoxic, distractional, and diffusional) and typically occur in the experienced diver. Dilutional blackout occurs with use of rebreather equipment while tank blackout could occur with any type of SCUBA diving.

Historically, the prototype blackout was labeled "shallow water blackout" in 1944 by Barlow and Macintosh for their Royal Navy Personnel Research Committee. Royal Navy divers were experiencing sudden losses of consciousness at depths less than 25 feet when using O<sub>2</sub> rebreathers, which limits diving to shallow depths. Oxygen toxicity was initially proposed as the cause of the blackouts. This was unlikely due to the shallow depths of the dive. Subsequently it was determined that the cause was CO<sub>2</sub> toxicity that occurred because the CO<sub>2</sub> filters were inadequate to remove this waste gas of metabolism with prolonged, vigorous activity. Later the term shallow water blackout became associated with shallow breath-hold dives after hyperventilation leading to hypocapnea. This delayed the central nervous system drive to breathe. The result was hypoxia leading to blackout.

Subsequently, the first author proposed *hyperventilation breath-holding blackout* be used as the preferred terminology.<sup>9</sup> This terminology encompassed blackouts that occurred both from underwater surface swims for distance and shallow water as well deep depth breath-hold dives. In addition, it was realized that hypoxic blackout could occur with SCUBA gear, especially rebreather types, as well.

The next chapter in the terminology evolution of blackout recognition in divers was the appreciation that other causes of loss of consciousness occur in divers.<sup>9,10</sup> Since almost all of these blackouts can occur without conscious awareness, and in direct contrast to the panic syndromes (see reference 3), we group them together as the no panic syndromes.<sup>4</sup>

Other terminologies that have been used to describe these conditions include surface blackout, deep water blackout, free diver blackout, and in-water blackout. When in reality, all the blackouts are manifested as losses of consciousness without seeming awareness of the impending disaster, the no panic syndromes terminology is an appropriate one.

**Breath-holding after Hyperventilation Blackout** Of the hypoxic blackouts, breath-holding after hyperventilation is the benchmark no panic syndrome (NPS). The precipitating cause is the lowering of blood CO<sub>2</sub> levels with hyperventilation (Figure 2).<sup>11,12</sup> This significantly lowers the CO<sub>2</sub> tensions in the blood. As the breath-holding activity is continued, O<sub>2</sub> is utilized for metabolic activity while CO<sub>2</sub> accumulates as a waste

product of metabolism. With the lowered starting point of CO<sub>2</sub> in the blood, the decreasing O<sub>2</sub> tensions in the blood reach a point where loss of consciousness occurs from hypoxemia before the breath-hold break point from CO<sub>2</sub> accumulation is reached. Consequently, the diver (or swimmer) loses consciousness without the irresistible desire to breathe. Once consciousness is lost, the victim will breathe spontaneously and, being submerged, will aspirate water. Without rescue and restoration of oxygenation to the brain, drowning occurs.

While hyperventilation can prolong the breath-hold time, as noted above, it is risky and should not be done excessively before any breath-hold diving activity. The safer approach is to take a few deep breaths—but not to the point of numbness—before beginning a breath-hold dive, while always employing the buddy system. In addition, breath-hold dives should be timed so that surfacing is initiated before hypoxemia may place the diver at risk of losing consciousness before the breath-hold breakpoint stimulus is approached and/or the surface is reached.

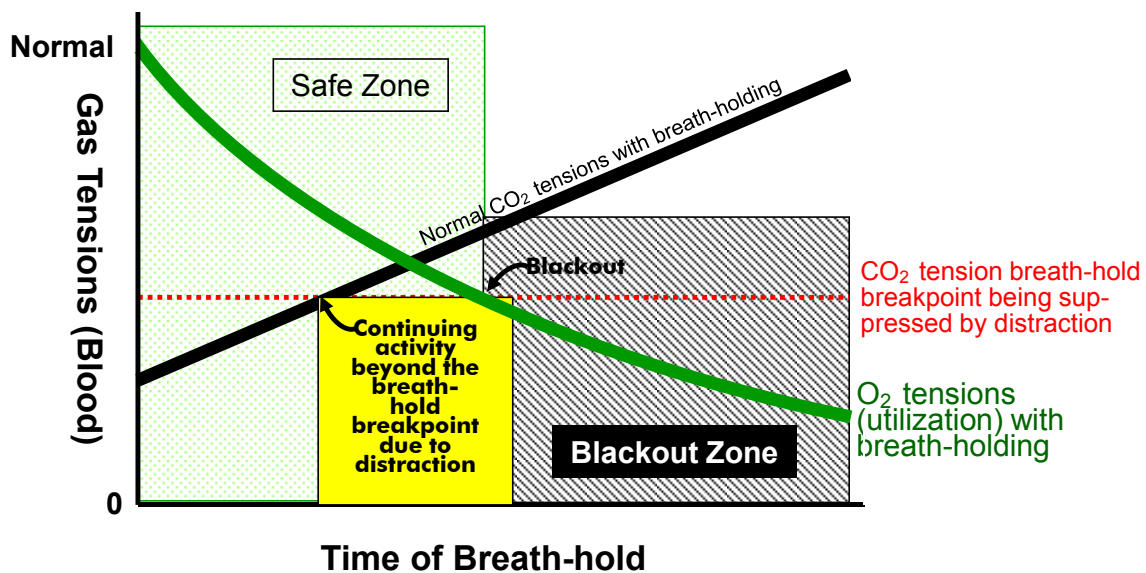
**Dive Scenario** An athletic 16-year-old male wanted to break his 50 yard underwater swim record and heard that hyperventilation would delay the desire to breathe. However, he was vaguely aware that it could also lead to drowning. After hyperventilating to the point of numbness in his hands and feet, he began his underwater swim with a buddy watching from the pool deck. At the 50-yard mark he had no “air hunger,” but his arms and legs felt heavy. After he touched the wall at the 60-yard mark, he sank to the bottom. Fortunately, his buddy realized what happened and brought the diver to the surface. After coughing and retching, the swimmer’s consciousness returned.

**Comment** Without the prompt rescue, a tragic outcome could have arisen. The coughing and retching occurred because water had been aspirated when the swimmer inhaled spontaneously after losing consciousness while submerged. Fortunately, the swimmer was well enough informed to have a buddy watch him—which always should be the rule with breath-hold diving activities.

**Distractional Blackout** This variation of the hypoxic blackouts occurs when the victim approaches the breath-hold break point but continues breath-holding due to “mind over matter” (Figure 3). This type of blackout can occur with intense focusing on collecting game such as retrieving a lobster from a crevice, spear fishing, prying an abalone off a rock, etc.; during breast stroke swim competition; and in children with breath-holding spells during some temper tantrums.<sup>13</sup> The latter situation can be an attention getting device where the child cries/acts out when denied his/her wishes, breath-holds, then can pass out from hypoxia. This is a great attention getting device for the child! Perhaps the breath-hold breakpoint is at a higher threshold in children than adults, which allows them to blackout from hypoxia before their breath-hold breakpoint is reached more easily.

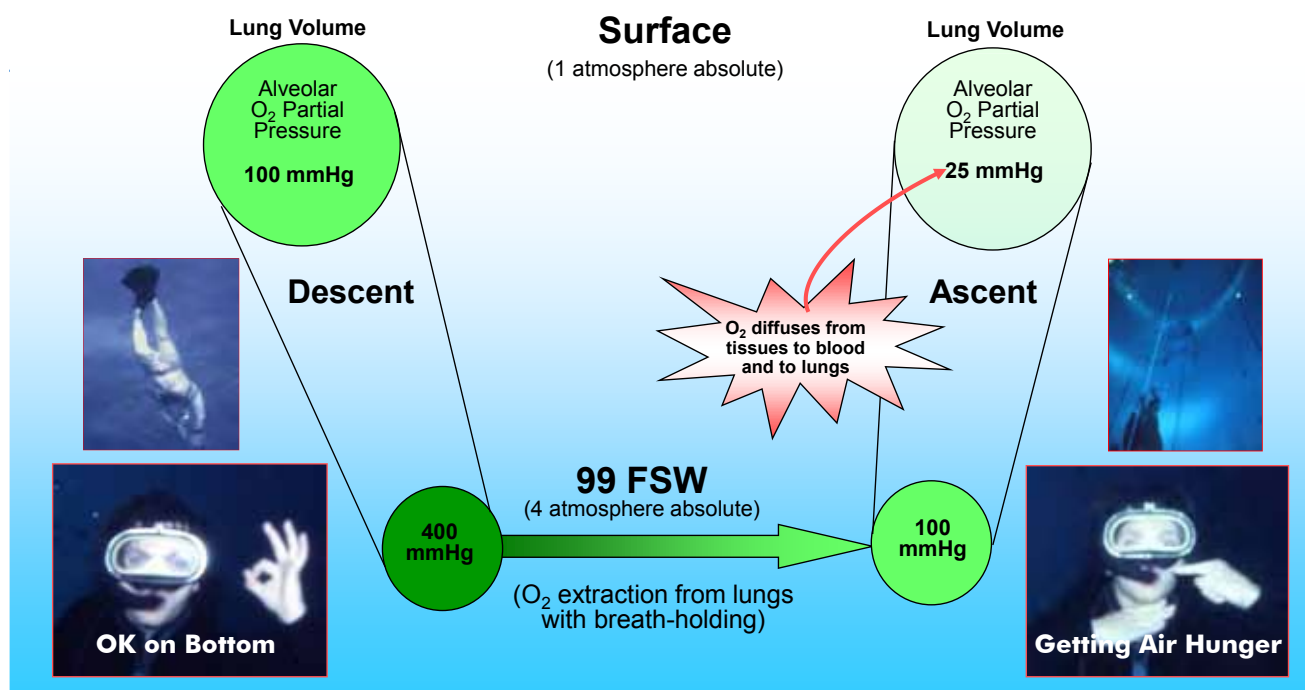
In the 1950s competitive breast stroke swimmers would take a breath at the last turn of a race, submerge, and swim the last 25 yards underwater. Not infrequently,

**Figure 3: Distractional Blackout**



**Legend:** The breath-hold breakpoint (before blackout occurs from hypoxia) may be suppressed by distractions, which overcome the breath-hold breakpoint. When this occurs, the diver/swimmer may continue the activity until consciousness is lost.

**Figure 4: The Physics of Diffusional Blackout**



**Legend:** With descent while breath-holding, the lungs (alveoli) decrease in volume while the partial pressure of the gas in the alveoli increases, as quantified by Boyle's law ( $V_1P_1 = V_2P_2$ ) and Dalton's law. With ascent the opposite happens, and the  $O_2$  tensions may become so low in the blood that  $O_2$  diffuses from the brain and other tissues in the reverse direction into the bloodstream because of the gradient.

when reaching the finishing line they would gray-out or completely blackout. For those breast stroke swimmers who used this technique, spotters would always be placed at the end of the pool to retrieve the grayed-out/blacked-out swimmer. Obviously the competitiveness of the race was a greater distraction than the breath-hold breakpoint. This type of blackout needs to be differentiated from breath-holding after hyperventilation blackout since blood  $CO_2$  levels would already be maximized from the swimming efforts that preceded the last length of the race. Subsequently, submerging for breast stroke races disqualified the swimmer. Now water is allowed to pass over the breast stroker's head and breaths are taken with each stroke so this variation of distractional blackout is no longer observed.

**Dive Scenario** A spear fisherman in his late 20s "bagged" a 3-foot mackerel, but the attached line got caught around a rock in about 30 feet of water. The diver did a breath-hold swimming descent to free the line and retrieve the speared fish as two buddies watched from the surface. Much difficulty was encountered in freeing the entanglement, but the diver persevered and struggled with the line. After 45 seconds or so the struggling ceased and the diver floated motionless a few feet above the entanglement. Fortunately, the buddies recognized the predicament, rescued the unconscious diver, and after a few seconds on the surface breathing surface air, the diver regained consciousness.

**Comments** The most likely explanation for what happened in this scenario is that it was a case of distractional blackout. The swimming descent and struggling consumed oxygen while rapidly generating  $CO_2$  which accounted for the relatively short 45-second bottom time before losing consciousness. When questioned after his revival, the rescued diver said that he was so preoccupied with freeing his prized catch that he suppressed the intense urge to breathe with what he said was going to be one last heroic effort and apparently lost consciousness (from hypoxia).

Distractional blackout is avoided by heeding the body's  $CO_2$  elevation stimulus, surfacing, and responding by breathing air on the surface. While setting time limits for breath-hold dives after hyperventilation to avoid blackout is effective for that type of diving activity, it is not effective for distractional blackout. This is because struggling as in the above scenario may markedly shorten the breath-hold time, and the distraction itself may be profound enough to allay the stimulus to breathe.

**Diffusional Blackout** This is the third of the breath-hold hypoxic blackouts. It occurs with deep breath-hold dives and its physics and physiology are well understood (Figures 4 and 5).<sup>14-15</sup> While on the surface, the lung alveolar  $O_2$  tension is about 100 mmHg at the end of each breath, and  $O_2$  extraction from the

blood decreases it to about 50 mmHg before the next breath is taken. This is sufficient to avoid hypoxemia leading to unconsciousness. When the breath-hold diver descends to 100 feet of sea water, the pressurization effect (4 atmospheres absolute) causes lung O<sub>2</sub> tensions to increase about four-fold and approach 400 mmHg (Dalton's law). Concomitant with the increase in partial pressure and directly related to it is a reduction in volume of the alveoli in accordance with Boyle's law ( $V_1 \times P_1 = V_2 \times P_2$ ; i.e., as the pressure increases in a contained flexible-wall configuration, the volume decreases in an inverse proportion). Oxygen extraction continues based on the partial pressure of O<sub>2</sub> until it approaches 50 mmHg, while CO<sub>2</sub> tensions in the blood only increase as a result of metabolism, as they would on the surface. This may give the diver a false sense of comfort and absence of air hunger while on the bottom and feel that it is OK to stay underwater longer.

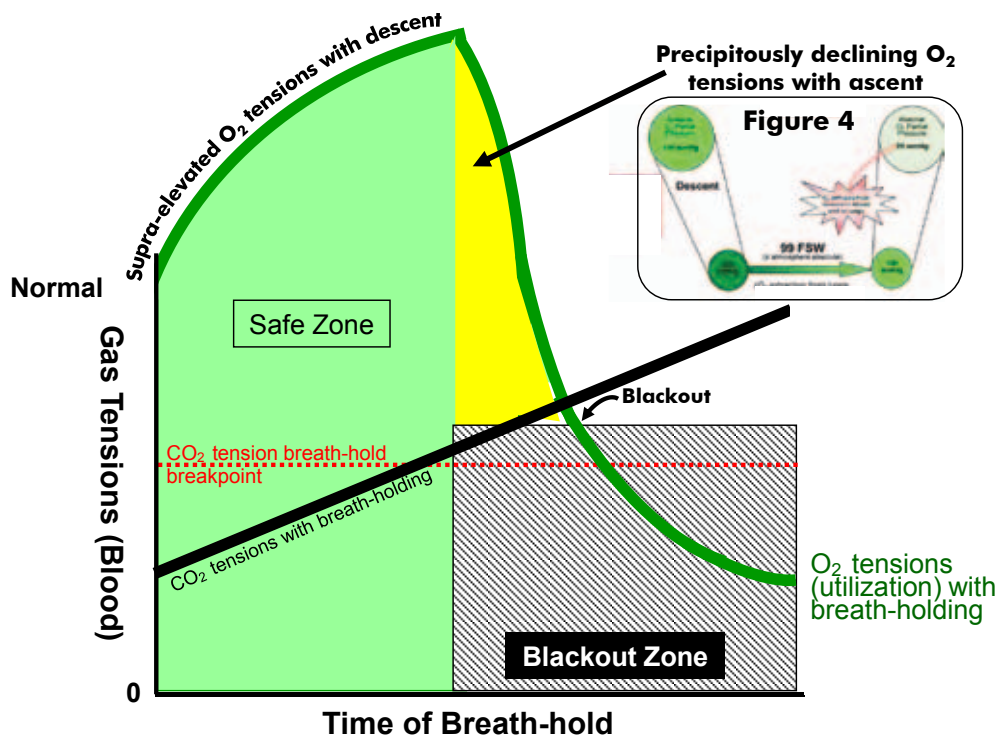
However, potential problems arise with ascent. With the decreasing ambient pressures associated with ascent, the alveoli expand and partial pressures of O<sub>2</sub> in the lungs decrease proportionately. The result is hypoxia and the probability that a reverse gradient will develop such that the now higher O<sub>2</sub> concentrations in the tissues move to the blood and the alveoli. This further aggravates the hypoxia situation and leads to loss of consciousness as the breath-hold diver nears the surface (Figure 5).

The lungs, the incredible organs that they are, only require a narrow range of O<sub>2</sub> partial pressures to provide sufficient oxygenation of the blood. Another way to consider this is that the lungs only require a finite number of molecules (as reflected in partial pressures) of O<sub>2</sub> to meet blood oxygenation needs. The partial pressures are independent of the percentage of O<sub>2</sub> breathed. For example, at a thousand foot equivalent depth chamber dive, the O<sub>2</sub> percentage of the gas mixture may only be one percent whereas the O<sub>2</sub> partial pressure (number of gas molecules) is sufficient to meet blood oxygenation requirements. This reflects what magnificent filters for O<sub>2</sub> diffusion the alveoli of the lungs are.

**Dive Scenario** A diver was doing deep breath-hold dives with a buddy in 80 feet of water. During ascent on his first dive, he lost consciousness about 10 feet from the surface and gradually began to sink. His dive buddy retrieved him at about a 50-foot depth and brought him, unconscious, to the surface. Shortly thereafter the victim regained consciousness. He reported that he felt OK until he neared the surface when he felt light headed and then apparently blacked-out.

**Comments** Although the diagnosis could be considered a near drowning, analysis of the history conforms to the loss of consciousness being due to a diffusional hypoxia event.

**Figure 5: The Physiology of Diffusional Blackout**



**Legend:** With deep breath-hold dives, O<sub>2</sub> partial pressures increase in the alveoli as expressed by Boyle's law and Dalton's law. With ascent, the opposite occurs, with precipitous declines in O<sub>2</sub> alveolar tensions. If alveolar O<sub>2</sub> tensions on the bottom approach blackout tensions, then with ascent and lung expansion, the tensions may fall to blackout levels and there may even be reverse gradients, which moves brain O<sub>2</sub> into the bloodstream.



Experienced breath-hold divers need to be aware of the pathophysiology of diffusional blackout. All breath-hold dives deeper than snorkeling depths should only be done with a dive buddy of equal skill. With dives deeper than 20 feet, it is crucial that the dive be planned and the signal to leave the bottom and start the ascent be based on a predetermined time and not air hunger, i.e., the breath-hold breakpoint.

**Dilutional Blackout** This type of blackout is associated with special types of diving equipment including trimix SCUBA, closed circuit 100 percent oxygen gear, and closed circuit rebreathers (Figure 6). The unifying problem is that of breathing a hypoxic gas mixture in the absence of CO<sub>2</sub> elevations and their stress-response mechanism, which normally stimulates breathing and maintains adequate oxygenation of the brain.<sup>2,4,16</sup> Trimix SCUBA diving utilizes SCUBA tanks with different gas mixtures to increase dive depths and reduce decompression requirements. A potential problem arises when the diver accidentally switches to a hypoxic (less than 50-60 mmHg O<sub>2</sub> partial pressure for the particular depth) gas mix. Since trimix is a variation of open circuit SCUBA diving and CO<sub>2</sub> is exhaled with each breath cycle, it does not accumulate to warn indirectly that the diver is breathing a hypoxic gas mixture that could lead to blackout.

**Dive Scenario** A possible trimix diving profile might read as follows: The dive starts with breathing air to a 100-foot depth. Then, the diver switches to a helium-oxygen mixture with 5 percent O<sub>2</sub> to the 300-foot depth. During ascent, the diver had intended to switch back to air at the 100-foot depth and pure oxygen at 33 feet but is distracted and continues to breathe the heliox mixture while decompressing. At the 16.5-foot depth the O<sub>2</sub> partial pressure of the 5 percent heliox mix would be less than 40 mmHg. This would subject the diver to lightheadedness, and he would almost immediately black out without “air hunger.”

**Comments** Since the diver was using open circuit SCUBA gear, CO<sub>2</sub> accumulation did not occur to signal the diver that something was wrong. Loss of consciousness can be very insidious when hypoxia occurs in the absence of elevated CO<sub>2</sub> tensions in the blood, as this scenario illustrates.

Closed circuit 100 percent oxygen gear and closed circuit rebreathers cause dilutional anoxia through a different mechanism than with trimix diving. With these diving rigs, gas is rebreathed through a closed circuit.<sup>2</sup> Oxygen is added to meet metabolic needs while CO<sub>2</sub> is reabsorbed in a scrubber unit. If O<sub>2</sub> addition is insufficient to meet metabolic demands, con-

**Figure 6: Special Equipment that Can Lead to Dilutional Blackout**



**Closed Circuit Rebreather**



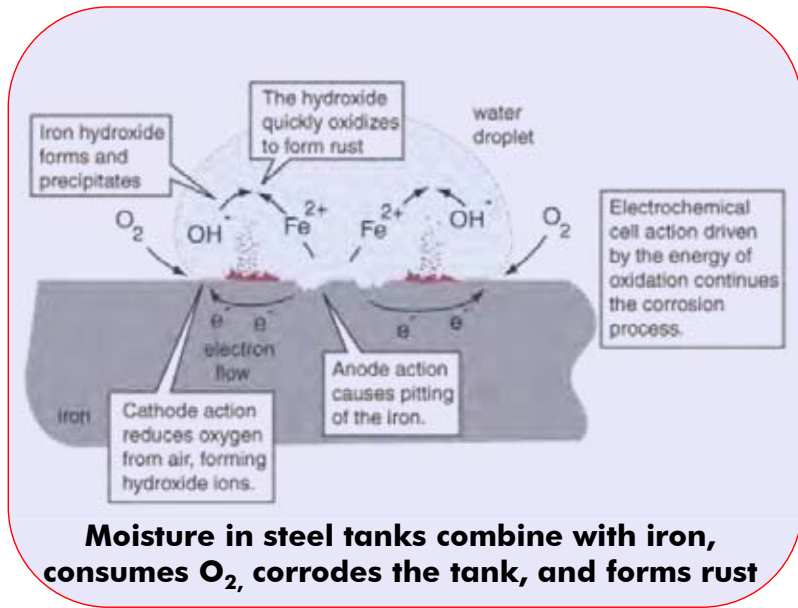
**Tri-mix Open Circuit SCUBA**

**Legend:** With reabsorption of CO<sub>2</sub> in a scrubber during closed circuit SCUBA or exhalation of CO<sub>2</sub> with open circuit tri-mix SCUBA, the alarm signs from elevated CO<sub>2</sub> are not present to stimulate breathing. If O<sub>2</sub> delivery is diluted due to breathing a hypoxic mixture of gas, consciousness will be lost without warning signs. This problem can occur when diving with the above types of equipment.

**Figure 7: Rust Leading to Tank Blackout**



**Rust in SCUBA Tank**



**Legend:** Proper maintenance and periodic inspections of SCUBA tanks prevent this problem. Again, hypoxia is insidious in this situation because the signal to breathe from elevated  $CO_2$  does not occur because it is breathed out to the ambient environment with each exhalation.

consciousness is lost without the warning signals that increase the breathing rate and signal that something is wrong. The outcome is loss of consciousness without appreciating an impending life threatening problem.

To avoid hypoxia blackout with rebreather diving equipment, training, pre-dive briefings, and verification of breathing mixtures is essential. With modern rebreather equipment, a constant partial pressure of oxygen can be maintained regardless of depth.<sup>2</sup> To ensure that the correct  $O_2$  partial pressure is maintained, three sensors in the inhalation component of the closed circuit are used. The sensors are checked frequently during the dive, typically at 1-minute intervals. If one sensor fails, the diver usually completes the planned dive. If two sensors fail, the dive needs to be aborted immediately in a safe and controlled fashion. When this type of equipment is used, buddy diving is essential. During training with closed circuit, 100 percent  $O_2$  rebreather equipment in the US Navy, buddy lines were tied between the divers. This reflects the respect dive trainers and supervisors have for the potential hypoxia hazard that can occur using this equipment.

**Dive Scenario** During a training dive using closed circuit, 100 percent oxygen gear in 10 feet of water, the dive buddy notices that the buddy line becomes taut and he

begins to drag his dive partner. When the dive buddy realizes that his buddy is unconscious, he brings the victim to the surface. After breathing surface air directly, the victim immediately regains consciousness without incident.

Later with debriefing and testing of the gas in the rebreather tank, it was found that the victim was breathing air in the tank instead of 100 percent  $O_2$ .

**Comments** Since gas was only being added at the calculated  $O_2$  consumption rate based on breathing pure  $O_2$ , the victim was only getting about one-fifth the amount of oxygen necessary to maintain consciousness. With  $CO_2$  resorption in the scrubber there was no awareness by the victim that he was breathing a hypoxic mixture. Fortunately, the buddy system averted a potential tragedy.

**Tank Blackout** This type of blackout is rare, with only one documented report, but for completeness is included as one of the hypoxic blackouts.<sup>17</sup> More importantly, it again demonstrates how the body's stress response mechanisms to breathe are hindered by eliminating  $CO_2$  elevations and how insidious hypoxia leading to loss of consciousness can be.

If moisture accidentally enters the galvanized SCUBA tank and the tank is stored for a long time, rust formation occurs (Figure 7). In the process, the  $O_2$  and iron in the tank combine, which results in the lowering of

the O<sub>2</sub> percentage in the tank. With SCUBA diving using the rusted tank, the partial pressure in the tank may be so low with the reduced oxygen percentage that consciousness is lost without awareness. Since CO<sub>2</sub> is exhaled with each breath, there is no awareness that the diver may be breathing a hypoxic mixture.

To avoid this type of blackout, SCUBA tanks need to be properly stored and inspected each year. If an O<sub>2</sub> sensor is available as needed for nitrox diving, the tank's O<sub>2</sub> percentage can be checked to be sure it approximates 21 percent.

## Section B: Other Causes of Blackout

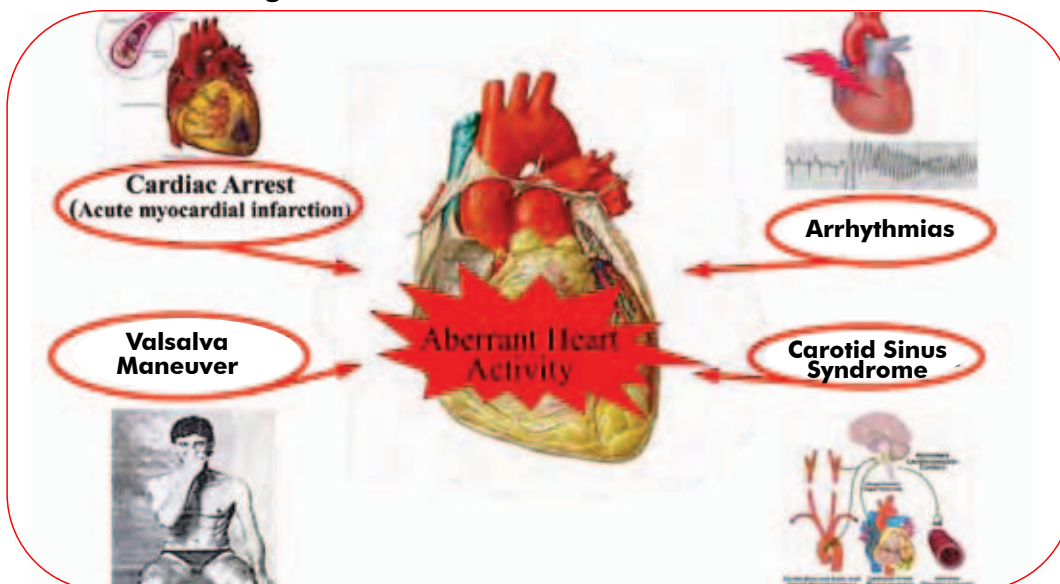
As noted previously, there are four other causes of blackout in addition to the hypoxic blackouts that can occur in divers (Table 1). Some may cause prodromal symptoms prior to the actual loss of consciousness, but the symptoms may occur so suddenly or be so inconspicuous that panic is usually not associated with the blackout. These other causes of blackout/no panic syndromes have a variety of etiologies. Hence, it is important to establish the cause of the problem that leads to the blackout so appropriate interventions, definitive management, and decisions about returning to diving can be made.

**Cardiogenic Blackout** This type of blackout is associated with cardiac events such as infarction and arrhythmia (Figure 8). The stresses of donning div-

ing gear, getting to the water entry site, and making it through the surf zone are usually the greatest of any phase of the dive.<sup>1</sup> Frequently significant risk factors for cardiac events are present such as obesity, hypertension, hyperlipidemia, smoking, and poor physical condition. Consequently, many of the cardiogenic blackouts occur before the actual SCUBA dive commences, especially with **acute myocardial infarction**. When a diver loses consciousness at these times, the *index of suspicion* should be highly weighted toward a cardiac event. Since retrieval from underwater is not the usual situation with this cardiac cause, standard cardiopulmonary resuscitation (CPR) efforts can be initiated immediately along with activating the Emergency Medical System (EMS). About a third of the deaths associated with SCUBA diving are attributable to cardiac causes and are the third leading cause of death in recreational SCUBA divers.<sup>18</sup>

**Dive Scenario** An overweight, poorly conditioned, cigarette smoking male in his late 40s decides to SCUBA dive. He believes it will be a good way to lose weight as well as be an enjoyable recreational activity. After pool training, his first open water dive requires a beach entry. With moderate difficulty and noticeable shortness of breath, he dons his diving gear and descends a 60-foot trail to the water's edge. After wading into waist deep water, a wave crests over him and he collapses. The diver is brought to shore, the EMS system activated and CPR started. Later at a hospital the diver is pronounced dead.

**Figure 8: Cardiac Causes of Blackout in Divers**



**Legend:** There are four main causes of cardiac-related causes of blackout in divers. Pre-dive medical examinations can identify patients with increased likelihood of cardiac arrest and for generating arrhythmias. Education can prevent Valsalva maneuver- and carotid sinus syndrome-related blackouts. The vasovagal reflex may be related to blackouts thought to be due to the carotid sinus syndrome.

An autopsy showed advanced atherosclerotic changes of his coronary arteries.

**Comments** Prior to commencing diving, the diver should have had a comprehensive pre-dive evaluation including history and physical, laboratory studies including lipid profile and electrocardiogram with stress testing, and a chest x-ray. In addition, to clear the diver to begin dive training should be contingent on weight reduction, smoking cessation, and initiating a fitness program.

This scenario is noteworthy for three reasons. First, it demonstrates the importance of the pre-dive medical evaluation, especially in the older aged adult. Second, it confirms that the time before beginning the dive can be the most stressful, energy-demanding phase of the dive. Third, it is wishful thinking on the diver's part that SCUBA diving is a good way to lose weight. With controlled SCUBA diving activities, the energy requirements after the water entry should be less than almost any land-based activity.

Sudden death from **cardiac arrhythmia** is another cardiac related no panic syndrome. Cold water immersion may induce a cardiac arrhythmia; if the arrhythmia significantly decreases cardiac output, loss of consciousness will occur abruptly. World class breath-hold divers as well as runners are often found to have bradycardia at rest with increased P-R intervals on their electrocardiograms.<sup>19</sup> Some of the heart rate slowing is present even to the extent of having first degree heart blocks from their prolonged P-R intervals. When losses of consciousness or drownings occur in this cohort of experienced, well-conditioned divers, this type of arrhythmia should be suspected.

A third type of cardiogenic blackout is that associated with the **Valsalva** maneuver.<sup>20</sup> As explained in the first text box at the beginning of this article, consciousness can be lost with a forceful Valsalva maneuver. This may be associated with increased expiratory resistance in the regulator or monitored breathing (to conserve gas supply). In this latter situation the SCUBA diver refrains from exhaling, then inhales until almost the breath-hold breakpoint. The forceful exhalation, especially with expiratory resistance in the regulator, may be enough of a delay that consciousness is lost. When a drowning death in a SCUBA diver occurs for no apparent reason, this type of blackout should be suspected. Sometimes clues from the history can be helpful such as the divers bragging about how much air they conserve with monitored breathing or boasting about how much remaining air is in their tanks when all of the other divers needed to surface due to low air supplies.

**Carotogenic** (our terminology) is the fourth cause of cardiogenic blackouts in divers. It is more commonly referred to as the carotid sinus syndrome.<sup>21</sup> Pressure and/or massage on the carotid sinuses, which contain pressure sensors (baroreceptors) in the carotid arteries of the neck, leads to slowing of the heart. A tight neckband of a neoprene hood may cause a similar effect. This has been hypothesized to cause enough slowing of the heart to lead to cardiac arrest and may be the explanation for seemingly unexplainable deaths in divers with no findings other than a tight hood. During dive training programs, this cause of blackout should be mentioned and the divers advised that any development of a headache associated with wearing a tight neoprene hood is sufficient reason to terminate the dive and obtain a better fitting hood. If neoprene diving hoods are being used during a dive, this potential problem from a tight hood should be mentioned in the pre-dive brief. Finally, the onset of a headache is an important warning system that something is wrong and the diver should immediately terminate the dive in a safe fashion. Attempts should then be made to ascertain why the headache occurred and determine how to relieve it before resuming the dive.

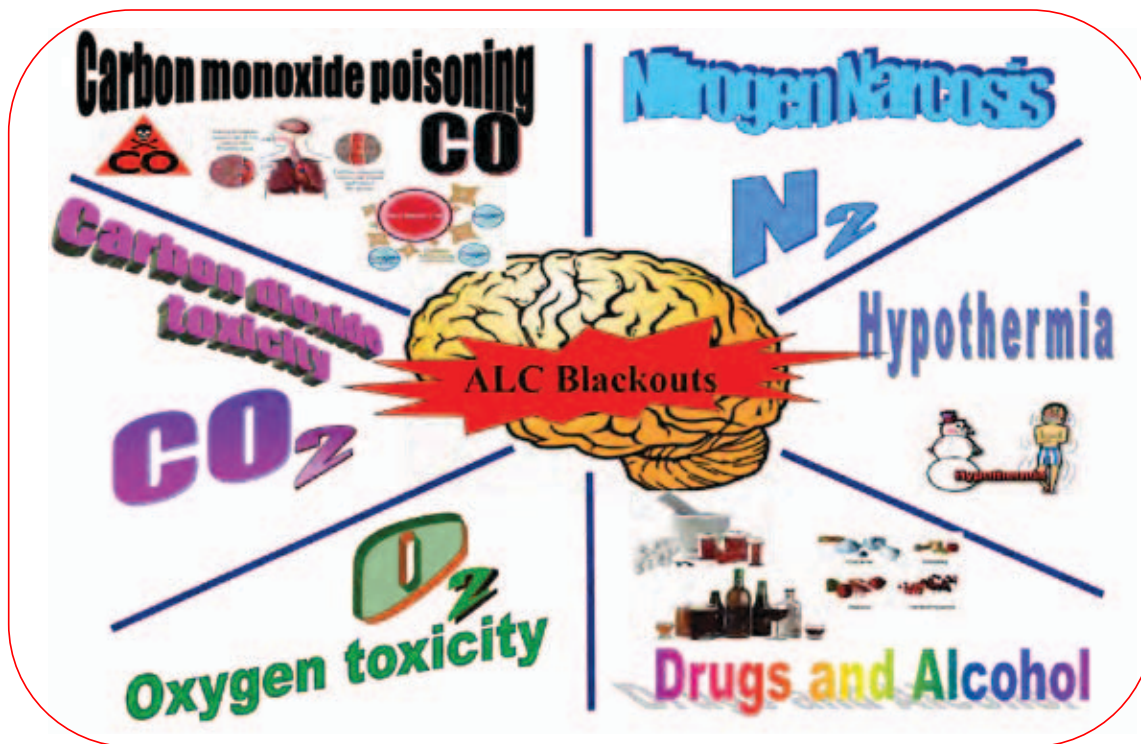
We postulate that a different mechanism occurs for this type of blackout. The mechanism is that the tight hood interferes with venous return from the brain. This increases intracranial pressures, which can lead to a cascade of effects. The initial symptoms are headache, then lightheadedness followed by blackout.

This postulation is based on observations where divers complained of headaches leading to lightheadedness during the surface phase of the dive who returned to shore before starting to descend. When they removed their tight hoods, their symptoms cleared and they resumed their SCUBA dives, sans hoods, without incident. Stimulation of the carotid sinus may initiate a vasovagal response, which may be the actual cause of loss of consciousness.

A more common cause of headache in divers is that of a sinus squeeze. This usually has its onset during the descent phase of the dive, is localized over a sinus cavity, and does not proceed to lightheadedness and loss of consciousness. In contrast, the headache associated with the "tight hood" syndrome is global in presentation and usually occurs early in the dive, often while the diver is still on the surface.

**Altered Mental Status and Loss of Consciousness Blackouts** There are hundreds of causes that lead to altered mental status, which can evolve to loss of consciousness. The causes range from drugs to poisons and from disease processes to environmental

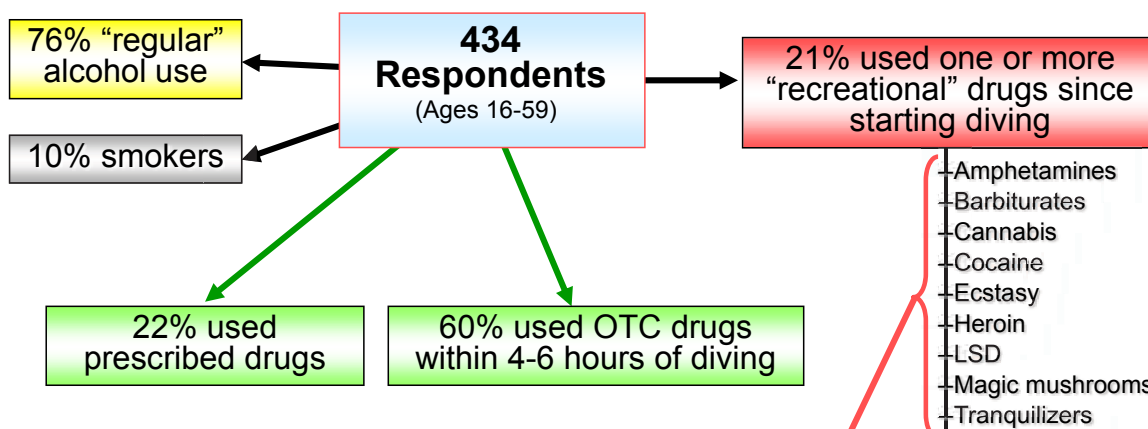
**Figure 9: Altered Level of Consciousness (ALC) Blackouts**



**Legend:** There are five main causes of altered levels of consciousness (ALC) in divers. In contrast to the other causes of loss of consciousness in divers, where a single event is likely to be the cause, the ALC losses of consciousness tend to be additive.

**Figure 10: Recreational Drug Use in Sports Divers**

**A Study from the United Kingdom by Dowse, et al., Ref. 24**



**Wow!**

- 40% of the recreational drug use group used schedule 1 (illegal) drugs
- 20% of the recreational drug use group used schedule 1 or 2 (prescribed drugs with potential for high abuse) drugs, from 5 minutes to 24 hours before diving.

**Legend:** This study indicates that drug use is widespread in the study population and possibly can be generalized to all recreational divers. Even with such alarming numbers, the incidences of drowning and episodes of serious diving problems requiring hyperbaric oxygen recompression are remarkably low. Nonetheless, any person in diving activity leadership should advise against recreational drug use in connection with diving.

**Table 2:** Causes of Altered Levels of Consciousness in Divers and How They Contribute to Blackout

Cause	Mechanisms & Effects	Comments
<b>Nitrogen Narcosis</b>	Under increased ambient pressure, nitrogen acts like a narcotic. The greater the depth, the more likely the effect  Before loss of consciousness, the diver is likely to become confused and do things that could endanger himself/herself	The analogy that each 50 feet of descent is equivalent to drinking one martini is used to describe nitrogen narcosis effects <sup>2</sup>
<b>“Downer” Effects of Drugs</b>	Analgesics, sedatives/tranquilizers, hypnotic agents, antihistamines, marijuana, muscle relaxants, and alcohol have side effects of altered levels of consciousness; all can make the diver more susceptible to nitrogen narcosis	If the diver has medical conditions serious enough to require these agents, they should not dive. There are great differences in susceptibilities to downer drugs
<b>Fatigue</b>	Decreases mental acuity and leads to sleepiness, delays reflexes, interferes with muscle function, strength, coordination, and endurance	May render the diver unable to meet the exertion demands of an emergency in the water
<b>Anxiety &amp; Inexperience</b>	Morbid concerns about the dangers of diving, encounters with sea life, etc.; may cause the diver to lose focus on safety aspect of the dive	Diving is not for everyone and should not be forced onto someone who is not comfortable in the water
<b>Toxic and Poisonous Gases</b>	CO <sub>2</sub> and O <sub>2</sub> at non-physiological levels cause problems from confusion to loss of consciousness to seizures. Carbon monoxide leads to headache and loss of consciousness	Toxicities are among the most preventable diving problems when dives are properly planned
<b>Hypothermia<sup>1</sup></b>	A continuum of responses to eventual loss of consciousness occur from hypothermia	Chilling and shivering during a dive should signal the diver to abort the dive; fatigue contributes to hypothermia

stresses. Often times the altered mental status onset is insidious, that is the person is unaware of what is occurring. Any altered mental status that occurs while diving is, of course, undesirable, and the loss of consciousness while underwater can be disastrous. There are a half dozen predominant causes of altered mental status that occur while diving that are different than the other causes of blackout described in this paper (Figures 9 and 10, Table 2).

The most well known cause of altered mental status in diving is **nitrogen narcosis**, popularized as “rapture of the deep” by Jacques Cousteau. What makes the altered mental status and loss of consciousness no panic syndromes of particular concern is that different causes can have additive effects while underwater. The is because nitrogen narcosis, which already alters the level of consciousness, is additive to any “downer” effects of drugs or other causes of altered mental status.<sup>22,23</sup> However, because of the confident image many divers exhibit, some feel that they can “do drugs” and still dive safely. One study showed that up to 40 percent of recreational SCUBA divers in

Great Britain had utilized downer drugs immediately before diving.<sup>24</sup>

The US Navy has a rule that care providers including inside tenders and physicians not make decisions about patient management while pressurized in the multiplace chamber. This is because of concerns that nitrogen narcosis may cloud judgment. Consequently, the inside personnel are directed to report their observations topside, then the topside personnel make decisions as to what needs to be done for the remaining course of the recompression treatment.

Other factors can contribute to altered mental status while underwater including **environmental factors** such as poor visibility and hypothermia, **fatigue, inexperience/anxiety, respiratory gas toxicities, and carbon monoxide poisoning**. Education is essential to prevent the insidious causes of altered mental status with associated losses of consciousness while diving. Such information should be an integral part of any dive training program and again emphasized during pre-

dive briefings, especially if the diving conditions are challenging or deeper than previously experienced, or when new/unfamiliar equipment is being used.

**Ascent Associated Causes of Blackout** The fourth category of blackouts are those associated with ascent. **Massive intrapulmonary** (the chokes) and/or **intravascular bubbling** (decompression illness) can cause the immediate loss of consciousness. The events can be so rapid that they can appropriately be considered in the spectrum of the no panic syndromes. These are rare situations that only occur in extraordinary circumstances such as with explosive decompressions and/or extremely rapid, uncontrolled ascents. Because of the insult imposed by bubbles in the arterial circulation to the brain, brain cells are deprived of oxygen and consciousness is lost. Autochthonous (*in situ*) bubble formation is the term that describes the phenomenon of inert gas coming out of body tissues to form spontaneous bubbles. Contemporary thinking is that bubble formation regularly occurs with ascents; the bubbles are transported in the plasma to the lungs and then eliminated with exhalation by the lungs. In over 99.99 percent of ascents from SCUBA dives, venous bubbles do not lead to decompression problems and are rightly labeled “silent bubbles.”

**Dive Scenario** Two hardhat divers using helium oxygen mixtures at a 280-foot depth lost hardwire communications with the surface tenders. A decision was made to surface the divers as fast as possible and do surface decompression in a hyperbaric chamber. About halfway to the surface, communications were reestablished, but the decision to continue the ascent was followed. The divers arrived at the surface unconscious and efforts to resuscitate them were unsuccessful. Massive intrapulmonary and intravascular bubbling was found at the autopsy.

**Comments** Massive gradients developed during the rapid ascent from the deep dive and precipitated the spontaneous formation of bubbles in the lungs and blood. This overwhelmed perfusion to and oxygenation of critical tissues such as the brain, heart, and lungs so that consciousness was lost almost instantaneously. Recompression to standard treatment table depths would have been ineffective in collapsing the bubbles and restoring perfusion. This is because the gradients for inert gas (helium) to move from the tissues to the blood as well as the spontaneous formation of bubbles in the blood were so great. It is likely that the rapidity of the events caused the divers' loss consciousness before any awareness of their perilous situation.

The carbonated beverage (soda bottle) analogy illustrates the *in situ* (autochthonous) vs. controlled off-gassing of the inert gas in the breathing medium. If the bottle cap is gently removed and the bottle not shaken, the physically dissolved carbonation in the fluid phase offloads without visible bubble formation. Eventually the beverage is no longer carbonated and tastes flat when consumed. Conversely, fizzing and visible bubble formation occur if the bottle is shaken.

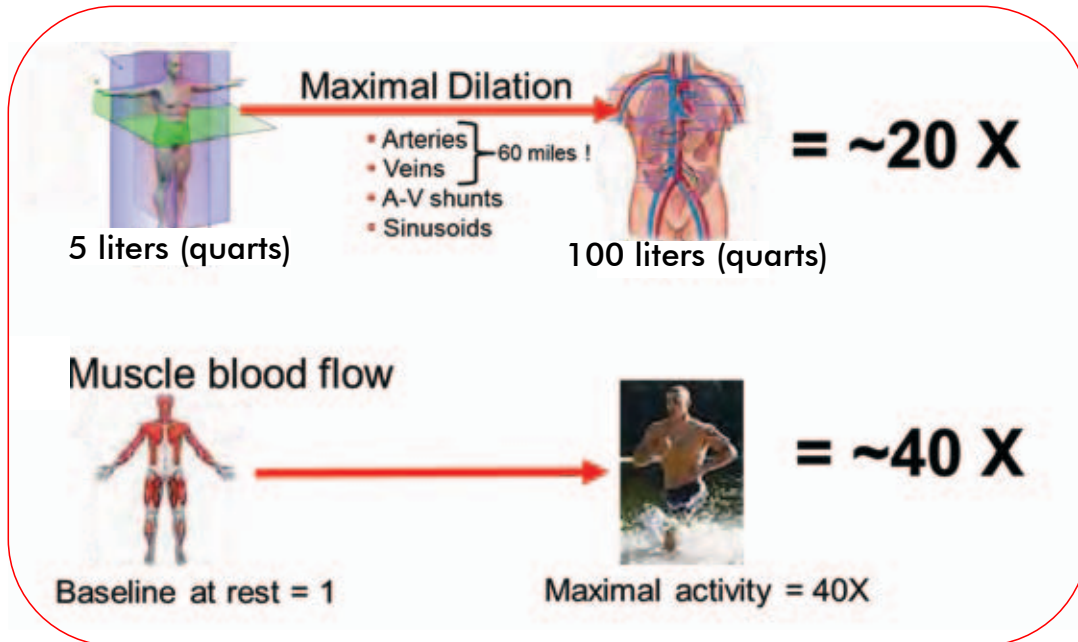
While this analogy is easy to appreciate, it appears that the reality of the situation, as described above, is that even with uneventful decompression from a SCUBA dive, bubble formation occurs in the venous blood and is ordinarily harmlessly off-loaded through the lungs with exhalation.

Problems arise when the gradients are so great that the bubbles coalesce, enlarge with ascent (per Boyle's law), and block the circulation to critical organs such as the brain and heart. In addition, the inert gas bubbles themselves appear to activate neutrophil sensitive adhesion molecules and initiate a reperfusion type insult. Consequently, even if the intravascular bubbles are dissipated with recompression, residuals of the decompression insult are likely to occur from this problem and/or the effects of interruption of perfusion to critical organs.

A second problem can also occur if venous bubbles enter the arterial circulation rather than exit through exhalation by the lungs. This is associated with patent foramen ovale defects of the heart and possibly arteriovenous fistulas in the peripheral circulation.

A second cause of altered mental status and loss of consciousness is due to **disordered decompression**. Whereas the blood volume of the average human is about 5 liters (quarts), the summated capacity (i.e., maximal filling of arteries, veins, sinusoids, arteriovenous fistulas, and storage reservoirs of the liver, spleen, and bone marrow) of the vascular system is about 100 liters or 20 times greater( Figure 11).<sup>25</sup> Critical tissues such as the brain, heart, and lungs have a constant blood supply, whereas non-critical tissues at rest ordinarily have only minimal perfusion needs. In these tissues, perfusion increases as needed for exercise, food digestion, wound healing, and infection management. The limited blood volume is superbly regulated by the sympathetic nervous system and chemoreceptors. If an insult such as a head injury temporarily disables the sympathetic nervous system, the critical tissues, which are fast tissues with respect to on- and off-gassing of inert gas, may no longer receive sufficient blood flow to off-load the inert gas that was on-loaded during the dive and bubbles form as in the massive intrapulmonary and intravas-

**Figure 11: Blood Volume versus Vascular System Capacity**



**Legend:** The vascular system has an enormous capacity, but due to autoregulation (sympathetic nervous system + chemoreceptors), only a small quantity of blood flows to non-critical tissues at rest. This conserves body mass and reduces the need for a “gigantic pumping system” to perfuse all tissues equally in the approximately 100-liter vascular system. With activity, the limited volume of blood is channeled to where it is needed, as, for example, to the muscles with exercise.

cular compartments described in the previous Dive Scenario. While the loss of consciousness may be the presenting sign from the injury or insult, serious residual neurological problems may be the outcome. Dive leaders need to be aware of **disordered decompression**, and if suspected, should immediately initiate oxygen breathing, hydration measures, and possibly administering a single aspirin tablet to the victim while he/she awaits hyperbaric oxygen recompression treatment as well as comprehensive neurological workup and management.

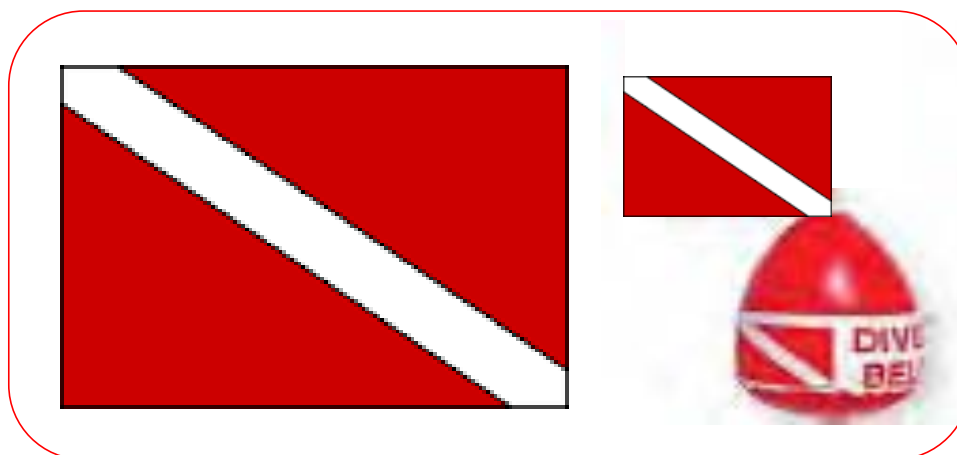
**Dive Scenario** An experienced diver, upon reaching the 100-foot maximum depth of his dive using a 36 percent nitrox mixture, experienced the sudden onset of an excruciating headache. He did not incur ear barotrauma during descent. He signaled to his dive partners that he had to abort the dive and ascended rapidly, but not explosively. He omitted the 15-foot rest stop and near the surface temporarily lost consciousness from what he later described as the severity of the headache. After rescue and oxygen breathing he had significant neurological residuals including severe weakness, balance problems, visual disturbances, and confusion. Even with multiple hyperbaric oxygen recompression treatments, residual neurological problems remained but they improved somewhat with time.

**Comments** The best explanation for this variant of the no panic syndromes was that the severe headache and loss of consciousness event temporarily inactivated the sympathetic nervous system control of perfusion. The result was diffuse bubble formation in the fast brain tissues due to the “steal syndrome” of blood from the brain. This resulted in insufficient blood flow to carry away the inert gas accumulated in the fast brain tissues during the short dive. The temporary loss of sympathetic nervous control of blood flow resulted in insufficient perfusion to the brain to off-gas nitrogen while blood was allowed to flow without sympathetic nervous system control to non-critical tissues.

A third cause of altered mental status and loss of consciousness is attributed to **alternobaric vertigo**. This is a type of ear barotrauma that occurs with ascent.<sup>2</sup>The failure to equilibrate pressures in the middle ear space during ascent cause mixed signals to the equilibrium perceiving mechanisms of the inner ear. The imbalance leads to disequilibrium, confusion, and loss of orientation, especially in turbid water. While technically not a no panic syndrome, the consequences can theoretically lead to uncontrolled descents, loss of consciousness from nitrogen narcosis, and drowning without manifesting a panic syndrome reaction.



**Figure 12: The Red, White-Stripped Dive Flag**



**Legend:** The dive flag is well recognized but not always heeded, especially if the surface craft operator's vigilance is diverted by distractions such as bringing game aboard the craft, socializing, etc. Alcohol consumption also factors in as a distraction. For dive activities not supported by a surface craft with a hoisted dive flag, the divers should use a flag plus buoy to demonstrate they are diving below the buoy.

### **Miscellaneous Causes of Loss of Consciousness Leading to No Panic Syndromes**

For completeness an additional diverse group of situations needs to be mentioned that can lead to loss of consciousness in the water. These include 1) **concussion** from overhead obstructions or boats, 2) **hypoglycemia** in the diabetic, 3) **seizure** in the epileptic, 4) **envenomations** from marine animals, and 5) **electric shock**. Divers must be particularly aware of overhead boat traffic as well as obstructions during their ascents. Many times dive sites are also shared with fishermen who may be oblivious to diving activities below. The red background-white stripe dive flag should alert boat traffic that divers are underwater (Figure 12). However, boat operators may be diverted with their fishing activities and/or inebriated and not appreciate that divers are below. Consequently, it behooves the ascending diver to use an extended upper extremity to protect his/her head during the last few feet of ascent, especially if obstructions, boat traffic, turbid water, or combinations of these are present. If a diver is found unconscious and there is a suspicion (e.g., bleeding head wound, moving boat, or obvious obstruction) of head injury, rescue and resuscitation needs to be followed up with a medical evaluation for brain injury.

Diabetes and epilepsy are medical conditions that present relative and absolute contraindications to diving, respectively. Both can lead to loss of consciousness in the water insidiously and without panic. Typically, they are documented in the pre-dive training

medical screening process and are already appropriately managed with medications. Generally, well controlled and knowledgeable diabetics are OK'd for SCUBA diving, and immediate pre-dive blood glucose checks can confirm that their blood sugars are in the safe range. Should prodromal symptoms of hypoglycemia be observed during a dive, the dive should be terminated, and the diver should drink a sugar laden beverage or receive a glucagon injection, both of which knowledgeable diabetics typically have available. Finally, diving by diabetics should be done in optimal conditions with well-planned dive durations and depths to minimize the risks of hypoglycemia.

The consensus of medical diving experts is that epilepsy is an absolute contraindication to SCUBA diving since a seizure underwater places the victim at a high risk for drowning. In addition, the increased partial pressures of oxygen while underwater may lower the seizure threshold. Although seizures often have prodromal symptoms such as an aura, usually the events proceed so rapidly that there is not time for ascents and other corrective measures. If a seizure occurs while submerged, controversy exists as to what the best management is. We recommend that the unconscious victim be brought to the surface with the neck in the extended position to prevent air trapping in the lungs due to an obstructed glottis. In addition, and what is controversial, is replacing the regulator in the victim's mouth. It is our opinion that inhaling air is a better option than inhaling water with the ascent. A

history of a childhood febrile seizure is not a contra-indication for SCUBA diving. Finally, there are SCUBA divers with a history of epilepsy and on medications who SCUBA dive against medical advice with no apparent problems during the dive.<sup>26</sup>

Envenomations that can lead to loss of consciousness when SCUBA diving are rare to non-existent. More likely they will occur with land-based activities. The two most potent venoms are found in the blue ringed octopus and the sea snake. Both contain potent neurotoxins that can rapidly lead to respiratory arrest. Sea snake bites typically occur when the snakes are removed from fishing nets, whereas blue ringed octopus envenomations occur when the octopus is handled after washing up on shore. Other marine animals such as jellyfish, cone shells, and sculpins can cause envenomations, but usually the toxins cause pain without loss of consciousness. The best way to prevent these injuries is through pre-dive briefings of the marine animal hazards that may be encountered at the dive site and to avoid handling these creatures. With respiratory arrest, cardiopulmonary resuscitation is required. Antivenins for sea snake bites are available from Australian laboratories.

Electric shocks from eels can be potent enough to render a person unconscious, although we are not aware that this has ever happened to a SCUBA diver. Awareness through the pre-dive briefing and avoidance as the advice for marine animal envenomations is the key to eluding this problem.

## Conclusions

Loss of consciousness in the water is always a serious matter because the outcome can be drowning. When warning symptoms of impending loss of consciousness are blunted or non-existent, the definition of a no panic syndrome is met. Because there are at least five different categories of problems that can lead to the no panic syndromes and subsequent loss of consciousness in the water, it is essential that the cause be established so optimal management can be initiated (Table 1). For example, loss of consciousness from decompression illness requires different management than from a hyperventilation induced breath-hold blackout/near drowning. Divers must be aware of the situations that lead to the no panic syndromes.

In contrast to the panic-provoking medical problems of diving, which most often occur in the inexperienced diver, the no panic syndromes are more likely to occur in the experienced diver. This is because attempting to extend breath-hold times or use closed circuit rebreather equipment puts the diver at increased risk

for loss of consciousness without warning symptoms. As in all diving activities, training, fitness, familiarity with equipment, and pre-dive briefings are the best measures for avoiding the no panic syndromes and their associated losses of consciousness in the water.

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**Dr. Michael B. Strauss** has had a long-standing interest in breath-hold diving and one of its consequences, namely blackout. As an adolescent he heard that hyperventilation could extend one's breath-hold time. In attempting to set a new under swim for distance record he experienced a blackout (see Dive Scenario on page 14 of this paper). Upon learning about respiratory physiology in medical school, Dr. Strauss began to

formulate some of the information that is fundamental to this article. While stationed with the US Navy in the Philippine Islands, he made a breath-holding descent to the bottom in 80 feet of water. A week after that another Navy diver was breath-hold diving at the same location and suffered a diffusional blackout on ascent, near-drowning, subsequent evolution to a thoracic squeeze, and death. Later, Dr. Strauss became a voice in abandoning the term *shallow water blackout* in favor of using *breath-holding blackout*. In his 2004 textbook *Diving Science*, he catalogued the hypoxic blackouts but also added other types of blackouts that could occur in divers such as cardiac, altered mental status, etc. This article reflects the further refinement of the cataloging of blackouts and integrates them with the stress-response approach presented in his three previous diving articles to appear in *Wound Care & Hyperbaric Medicine*. In collaboration with his coauthors, this article further clarifies some areas of confusion in blackouts such as why hyperventilation causes numbness in the extremities, hypoxia's role in stimulating respiration, and why distractional/temper tantrum blackouts occur in children.



**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

## Hyperbaric Oxygen Clinical Application in Resuscitation from Insult of Acute Severe Anemia

Keith Van Meter, MD

Hyperbaric oxygen therapy provides a way to practically address acute blood loss anemia when blood transfusion is not available, desired, or safe. The use of hyperbaric oxygen has withstood the test of time to reduce initial rapidly-mounting oxygen debt in the sick or injured before shock becomes irreversibly damaging. The efficacy has been borne out in human and animal research as well as in clinical experience.<sup>1-3</sup> Nearly 25 years ago, Shoemaker observed in adult human patients that if within 4 hours of induction of shock, the oxygen debt were less than 8 liters, the subject experienced at most a mild systemic inflammatory response. If an oxygen debt in the same circumstance were greater than 22 liters, then multi-organ failure would occur and if more than 33 liters, death would inevitably occur.<sup>4</sup>

In a sense, oxygen debt is simply described as a negative value of  $VO_2 - DO_2$  over time (where  $VO_2$  represents oxygen consumption,  $DO_2$  represents oxygen delivery, and the negative subtractive value, if present, is indexed to lapse of time).<sup>5</sup> Inflamed ischemic tissue, especially upon reperfusion, develops an increased metabolic rate that expends available high energy phosphates (i.e., ATP) at a prodigious rate. If oxygen is not made available to the intermitochondrial space within cells, then adequate resupply of ATP simply fails.<sup>6</sup>

The mitochondria are elegant oxygen gas and chemical powered furnaces that produce energy through the compartmentalization of the hydrogen ions, which turn the rotor-stator motor of the ATP-synthase nanomachine to compact inorganic phosphate (Pi) against ADP to produce ATP (Figure 1).<sup>7</sup>

The brain and the heart are most sensitive to drops in oxygen supply (every 100 grams of brain uses 3.5 ml of oxygen per minute, and every 100 grams of heart

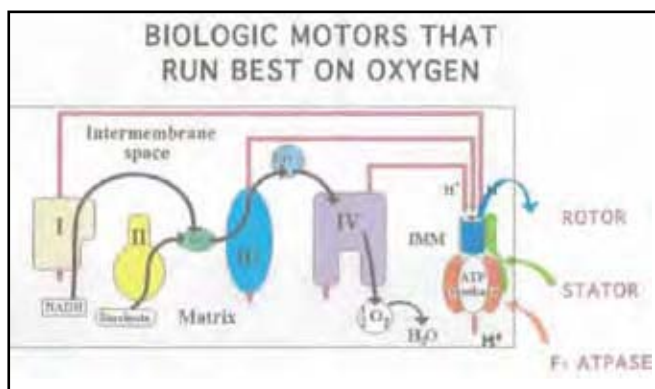


Figure 1. The respiratory enzymatic complex chain in the mitochondrial inner membrane drives the production of ATP by ATP-synthase which, in turn, is empowered by the docking of oxygen molecule on cytochrome aa3 (designated as complex IV in the chain).

uses, at rest, 8 ml of oxygen per minute and up to 70 ml/min in extreme exercise). Plying brain function against blood flow in non-anemic patients, the following brain biologic function follows for blood flow:<sup>8</sup>

1. normal function > 50 ml/100 g/min
2. electrophysiologic dysfunction < 30 ml/100 g/min
3. cationic dyshomeostasis < 20 ml/100 g/min
4. biologic death < 5 ml/100 g/min

Low blood flow rates damage end organs even more pronouncedly in anemia. Cellular function worsens in low blood flow rates and yet, in the short term, hyperbaric oxygen can bolster the oxygen content of blood by allowing the plasma fraction as well as the red blood cell cytosol to carry so much more oxygen that even "life without blood" is possible. Boerema exsanguinated swine by severe bleeds to arrive at Hgb 0.4 g/dL. He then intravenously infused D5 Ringers lactate intravenous solution. Next, the animals were immediately exposed to 3 atmospheres of pressure in a

hyperbaric chamber, while they administered oxygen. The animals were re-infused with their shed blood before being brought back to 1 atmosphere, whereupon they walked off unimpaired.<sup>9</sup>

More recently, our laboratories have been able to reproduce the same experiment exposing normothermic swine to similar drops in hemoglobin by exsanguination. The animals have been monitored with a polarographic oxygen-sensing electrode placed through a brain case burr hole into deep brain structure. Surface animals, ventilated with an FIO<sub>2</sub> of 100% and intravenously fluid resuscitated, had a brain tissue pO<sub>2</sub> of 0 mmHg, while animals treated the same way but pressurized to three atmospheres in a hyperbaric chamber maintained a normal physiologic brain tissue pO<sub>2</sub> of 30 to 40 mmHg. Shed blood was re-infused just at the 1 hour mark before these animals came to surface and as in the Boerema experiment, the animals walked off unimpaired (Figures 2–6).<sup>10</sup>

Additional effects of hyperbaric oxygen beyond its use for bridging therapy in acute blood loss anemia include:

1. Increase in flushing of intramedullary adult stem cells into the central circulation<sup>11</sup>
2. Blunting of B-22 integrin cell surface adhesiveness of white blood cells during post-ischemic reperfusion<sup>12,13</sup>
3. Induction of gene expression of over 8000 documented genes, approximately half of which orchestrate anti-apoptotic/anti-inflammatory function and half of which orchestrate cellular reparative function<sup>14</sup>
4. Improvement of both aerobic and anaerobic bacterial killing by white blood cells<sup>15</sup>
5. Improvement in the bactericidal activity of a selection of antibiotics (vancomycin, gentamycin, clindamycin, erythromycin, and sulfa drugs to name a few)<sup>16</sup>
6. Lessening of all membrane lipid peroxidation in reperfusion injury by truncation by means of quenching lipid endoperoxide radicals<sup>17</sup>
7. Lessening of impact of intravascular microparticles after ischemic insult<sup>18</sup>

By use of post-exsanguination “tailing” hyperbaric oxygen treatments in series, hemoglobin levels have been demonstrated to increase above the expected baseline recovery, and this may have to do with induction of cellular reparative genes.<sup>1</sup>

By way of illustration of the modern clinical use in remote medically based field settings, two cases from the literature are summarized.



Figure 2. In swine, a Licox<sup>®</sup> polarographic electrode is inserted through calvarium burr hole to measure tissue pO<sub>2</sub> in brain tissue during exsanguination.



Figure 3. Blood extractor for a swine exsanguination experiment in a veterinary research facility's hyperbaric chamber in New Orleans, Louisiana.



Figure 4. A swine exsanguination experiment underway at the Baromedical Research Institute in New Orleans, Louisiana.



Figure 5. Hyperbaric oxygen treated swine after hour-long, normothermic exsanguination insult walking in vivarium right after shed blood was returned and the animal came to surface.



Figure 6. The same swine from Figure 5, 1 year after severe exsanguination insult was treated by hyperbaric oxygen for 1 hour without replacement blood (all animals without immediate hyperbaric oxygen resuscitation died).

### Case 1

Forty-five years ago, at the Long Beach Naval Installation in California, a 26-year-old male Jehovah's Witness was treated after a previously undiscovered gastric leiomyoma eroded blood vessels to acutely produce a greater than 40% blood volume blood loss. The patient was brought to a naval yard dock-side hyperbaric chamber. The patient was kept in the chamber for several days and had short-interval hyperbaric oxygen administration periods by compression to 2.8 atmospheres. Progressively the intervals between hyperbaric oxygen breathing periods could be lengthened by responding to ever-increasingly periods of EKG ST-segment elevation normalization by electrocardiographic monitoring. The patient was able to recover after a necessary gastroectomy without need for transfusion.<sup>19</sup>

### Case 2

Shortly after Hurricane Katrina struck New Orleans, Louisiana in the late summer of 2005, the LSU Health Sciences Center Emergency Medicine physicians participated in the treatment of a 42-year-old commercial diver aboard a diving supply ship in the Gulf of Mexico. The diver was in storage in a heliox environment at 300 fsw of simulated pressurization in a deck saturation diving operation set up for repair of deep subsea oil and gas infrastructure. The diver had exsanguinated by erosion of a duodenal artery caused by a duodenal ulcer and was in shock for 8 hours. Onsite diving company medics attempted, unsuccessfully, to gain intravenous access. No interosseous needles were available, and hypodermoclysis with normal saline was ultimately used to slowly replace intravascular volume. The patient was pulsed with short interval oxygen breathing periods at 300 fsw of pressurization utilizing a surface equivalent fraction of inspired oxygen (SEFIO<sub>2</sub>) of 300%. He was set up with chest leads for cardiac monitoring and was able to regain

consciousness as the physician response team was on the way by helicopter. The interval between the enriched hyperbaric oxygen breathing periods was slowly increased, gauged by the non-return of ST-segment elevation on the cardiac monitor. Immediately upon return of any ST-segment elevation, the diver was placed on a SEFIO<sub>2</sub> 300% breathing mix, and the ST-segments would promptly normalize. Progressively the patient became less reliant on the hyperbaric enriched oxygen breathing periods, which next allowed the use of short rounds of enriched oxygen breathing every 6 hours. The patient was orally fed protein broth and given injectable hematinics (iron, erythropoietin, folic acid, and B12 and generic multivitamins) either by subcutaneous, intravenous, or intermuscular route, as an intravenous line could finally be established. Because of foul weather and logistics, type O packed red blood cell units could not be brought to the remote site. The diving support ship made its way to port while the diver underwent an obligatory slow decompression from 300 fsw to surface. At surface and now in port, the diver walked out of the chamber and into a waiting ambulance. His hemoglobin had by then elevated to 4 g/dL after he was transfused in route with type and crossed units of packed red blood cells. He was admitted to a regional medical center, had an endoscopy, and was discharged the next day after discovery of a large, hemastatic duodenal ulcer that was almost completely healed, which had recently eroded a duodenal artery. He had a positive test for *Helicobacter pylori*. In the hospital, he was transfused with additional units of typed and matched packed red blood cells to hemoglobin greater than 8 g/dL. His lab chemistries were normal and he suffered no evidence of multiorgan dysfunction and has been well since (Figures 7–10).<sup>10</sup>



Figure 7. Saturation chamber/diver storage chamber on deck of a commercial diving support ship in which the exsanguinated patient in Case 2 presentation was resuscitated with hyperbaric oxygen therapy.



Figure 8. The patient in Case 2 is featured on the center monitor screen in the saturation gas control shack. The diver was at 300 fsw and had exhibited an Hgb level well under 4 g/dL for days. He was able to read a book and take care of daily self-care activities under his own power by way of intermittent, enriched hyperbaric oxygen breathing periods.



Figure 9. The patient in Case 2 is shown with a nearly healed duodenal ulcer ready for hospital discharge after a diagnostic endoscopy.



Figure 10. Two years after the exsanguination shock insult incident the patient in the Case 2 presentation enjoys a trip to watch the Olympic Games with his family. His duodenal artery had been eroded by a duodenal ulcer while he was on a saturation diving operation in the Gulf of Mexico, and he has had no recurrence.

Hyperbaric oxygen therapy actually does not involve very high technology, and at least one level 1 trauma center has placed a multiplace chamber directly in the emergency department for the purpose of resuscitation translational trials to treat exsanguinated thoracoabdominal gunshot wound patients with loss of vital signs in the field (Figures 11 and 13).<sup>20</sup> Small, two-person transport chambers have been engineered to fit in the aisle of a conventional box ambulance patient compartment to be pressurized by H-flask high pressure air and oxygen without the need for compressors (Figures 12 and 14). In a transport chamber under pressure, a specially trained paramedic would render immediate advanced trauma life support to a patient in the field, and transfer with the patient to the emergency department while still under pressure. At the emergency department, the transfer chamber would be locked to the multiplace chamber for continuation of damage control resuscitation under pressure by emergency physicians and trauma surgeons, where they would then have use of hyperbaric oxygenation for the patient.<sup>10</sup> Only the future will reveal if medical politics will allow this to happen.

The 13th edition of the Undersea and Hyperbaric Medical Society *Hyperbaric Oxygen Therapy Indications* provides additional information on this approved indication.

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Figure 11. A multiplace hyperbaric chamber with SICU/ICU capability in the Emergency Department at Charity Hospital's Trauma Center in 2005 in New Orleans, Louisiana.



Figure 13. A single patient hyperbaric transport chamber capable of fitting in the aisle a standard EMS box ambulance patient compartment (capable of 6 ATA pressurization).



Figure 12. The first floor Emergency Department where the Trauma Center's multiplace hyperbaric unit was located.



Figure 14. A paramedic and patient containing transport chamber, which is also capable of 6 ATA pressurization and able to fit in the patient compartment of a conventional, land-based EMS ambulance.

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# Wound Care & Hyperbaric Medicine

## Clinics In Focus

### Center for Wound Care and Hyperbaric Medicine Springhill Medical Center, Mobile, AL

This issue starts our first interview with some of the most outstanding hyperbaric and wound care centers around the world. We asked Julio R. Garcia, RN, BSN, CHT, ACHRN, Program Director at the Center for Wound Care and Hyperbaric Medicine at Springhill Medical Center in Mobile, AL, to talk about his dynamic clinic.

#### Date of UHMS accreditation

May of 2004 was the year of our initial UHMS accreditation. We were the 33rd accredited center nationally. Currently we are one of the few centers in the US that has been accredited four consecutive times. Our current accreditation expires in 2016.

#### How has seeking UHMS accreditation affected your clinic?

Accreditation demonstrates a commitment to strive for excellence. It demonstrates an adherence to the recognized scientific, medical, and safety standards of the field of hyperbaric medicine. It has helped our unit to be recognized as a leader for our region.

#### What are the most common indications treated at your clinic?

We treat the full scope of UHMS indications. Our highest utilization has been for diabetic foot ulceration, but with the recent changes to the specificity of the definition on national coverage determinations and local coverage determinations, it is harder to demonstrate no clinical signs of improvement in the last 30 days with standard care. Since a Wagner III DFU has an infectious process we do see a good number of fetid diabetic foot ulcerations, necrotizing soft tissue infections, and chronic refractory osteomyelitis. We are also one of the few centers left in the US, especially on the central Gulf Coast, that will treat decompression illness.

#### STATS

Center for Wound Care and Hyperbaric Medicine  
Springhill Medical Center, Mobile, AL 36608  
[www.springhillmedicalcenter.com](http://www.springhillmedicalcenter.com)

Operating since 1988

Number of chambers: 2

Chamber type: Perry Baromedical Sigma II Duo-place chambers

On staff: 2 Physicians/6 CHRN/1 CHT

Dr. Harry E. Studdard, II ABPM/UHM, Medical Director

Julio R. Garcia, CHT, ACHRN, Program Director

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

One can go a whole career in hyperbarics and never see a single arterial gas embolism (AGE). In July of 2014, we had three sent to our center in a span of 2 weeks. One of these patients was paralyzed completely on the left side and on a ventilator. During the second treatment on the US Navy Treatment Table 6, the patient spontaneously began to move his left upper and lower extremities. It was like watching a miracle.

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

The center was managed from September of 1999 to November of 2008 by Diversified Therapy Corporation. Since that time it has been managed by the hospital.



(Top) The two Perry Baromedical Sigma II Duo-place chambers are set up like a large monoplace chambers. The maximum rating is 3 ATA, and the entry lock-up capability allows staff to access the patient on a critical care stretcher.



(Right) The center boasts a fully-certified staff.

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

Always doing what is right for the patient. Focusing on the needs of the patients and the community is our primary driver. Focus on sound medicine with good outcomes and doing the right processes and your reputation will build.

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

Educate, educate, and more educating. This means to the community, the patients, the physicians, and staff at your individual institution. When you think about the explosion in evidence-based medicine for wound care in the last 20 years and the relative infancy of clinical hyperbarics (less than 60 years), education has to be our focus throughout the entire spectrum.

**Is there any additional question you’d like to answer, or any other information about your clinic you’d like to showcase?**

The unit has received the Gulf Coast Chapter Unit Appreciation Award for hosting the chapter meeting in 2006. The entire unit is certified as CHTs or CHRNs. We are a four-time accredited facility by the UHMS. We are a teaching facility for an introductory course in hyperbaric medicine and a safety director course in conjunction with Wound Care Education Partners. We are an approved preceptorship site with the National

Board of Diving and Hyperbaric Medical Technology. The unit director, Julio R. Garcia CHT, ACHRN is a recipient of the Alabama Hospital Association 2011 Hospital Hero Award and 2014 Gulf Coast Chapter Special Services Award.

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# Wound Care & Hyperbaric Medicine

## The Sweet Zone

Darren Mazza, EMT, CHT

Perhaps one of the most important challenges in hyperbaric therapy is to determine a safe, acceptable pretreatment blood glucose level in the diabetic patient. It's been my experience that a pretreatment blood glucose level in one patient may not be adequate for another. It's absolutely imperative, though sometimes difficult, to find what I call the "sweet zone"—the acceptable pretreatment blood glucose level for a diabetic patient. Although our clinic has a written policy that states diabetic patients must have a prehyperbaric treatment blood glucose level of at least 110 mg/dL before they begin their treatment, we have to first consider several preceding factors that take precedence over a number marker. The following questions need to be answered.

- 1. Is the patient's blood glucose under control?**
- 2. Did the patient eat before coming in for HBOT?**
- 3. Did the patient take insulin before coming in for HBOT?**
- 4. How low do patient blood glucose levels typically drop after a treatment?**

Often one of the most difficult tasks for both the patient and provider is to get the patient's blood glucose level under control before beginning hyperbaric therapy. Studies have shown that optimum wound healing in a patient with a blood sugar >200 mg/dL cannot be achieved. During the patient's history and physical exam the hyperbaric provider will determine how uncontrolled the patient's blood sugars are. The provider will help the patient make diet and lifestyle adjustments in an effort to gain control of the patient's blood sugar levels, promote better overall health, and ensure that the patient gets the most out of hyperbaric therapy in order to achieve optimum wound healing.

Diabetic patients should be instructed not to take an insulin shot before hyperbaric therapy. It's much safer for the patient to avoid taking insulin until after receiving HBOT. Blood sugars in the diabetic patient have been known to drop 50 – 100 points during a hyper-

baric treatment having to do with complex changes in glucose metabolism during a hyperbaric therapy treatment; coupled with additional insulin this could have disastrous consequences. I once had a patient with a pretreatment blood glucose level of 211 mg/dL who did take her insulin around an hour and 45 minutes prior to HBOT. The patient told me that after she ate a big lunch, she checked her blood sugar and it was 343 mg/dL, so she took her normal insulin dose. After her treatment I checked her post-HBOT blood sugar and it had dropped to 94 mg/dL! That's just an example of how much a blood sugar can drop after taking insulin and having hyperbaric therapy.

Make sure an interventional care plan is in place for each patient and is updated weekly by the hyperbaric nurse. This will create a weekly trend in a patient's blood sugar levels and allow it to be monitored by both the CHT and hyperbaric nurse. The hyperbaric nurse is the go-to person when it comes to intervention and will be instrumental in ensuring that the diabetic patient maintains good nutrition, particularly during their course of hyperbaric therapy. The hyperbaric nurse can work with both the patient and family in devising a dietary plan that will ensure the patient's meals contain enough protein to sustain their blood sugars during hyperbaric therapy.

### 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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# Wound Care & Hyperbaric Medicine

## Press Release

### The American Diabetes Association Encourages People to Focus on Healthy Living for World Diabetes Day

**Contact:**

**Lauren Gleason, [lgleason@diabetes.org](mailto:lgleason@diabetes.org)  
703-549-1500 ext. 2622**

**Alexandria, Virginia—November 14, 2014**

During American Diabetes Month® this November, the American Diabetes Association will join the International Diabetes Federation to raise awareness of diabetes on Nov. 14 - World Diabetes Day. Nov. 14 is the birthday of Sir Frederick Banting, the co-discoverer of insulin.

The World Diabetes Day 2014 campaign marks the first of a three-year themed focus on healthy living and diabetes. This year's activities will specifically address the topic of healthy eating and its importance both in the prevention of type 2 diabetes and the effective management of all types of diabetes to avoid complications. All campaign activities will be informed by the slogan, "Diabetes: protect our future."

"Diabetes is a serious epidemic facing not only our nation, but the world," said Marjorie Cypress, PhD, C-ANP, CDE, President, Health Care & Education, American Diabetes Association. "But we can make progress in the fight to Stop Diabetes® by educating people about the vital importance healthy living plays in helping to prevent type 2 and manage all types of diabetes. We need to work together to provide people with healthy ideas they can put into action, including resources to help people make the right choice when it comes to what they eat."

Nearly 30 million Americans have diabetes and an additional 86 million have prediabetes, placing them at risk for developing type 2 diabetes. The latest estimates from the IDF Diabetes Atlas estimate there are 387 million people living with diabetes worldwide.

When it comes to healthy living, and making better food decisions, it can often be overwhelming, especially if you are living with diabetes. Here are some tips to follow to learn how to better balance meals throughout the day:

- **Choose healthy fats** like nuts, olive oil, peanut butter and avocado.
- **Eliminate sugar-sweetened beverages** – drink water flavored with fruit and herbs, like mint.
- **Understand carbohydrates** – the balance between how much insulin is in your body and the carbohydrate you eat makes a difference in your blood glucose levels.
- **Incorporate healthy carbohydrates** into your diet, instead of processed.
- **Decrease the amount of sodium in the diet** which can help lower blood pressure. Lowering blood pressure also means you are decreasing your risk for heart attack or stroke, both of which are common diabetes complications.

"As people try to make healthier food decisions each day, don't forget that healthy eating begins with breakfast," said Cypress. "Small steps like being aware of the fats you cook with and using oil instead of butter or lard, as well as incorporating whole grains into your breakfast, can make the meal that much healthier, and it still tastes good."

The American Diabetes Association is leading the fight to Stop Diabetes and its deadly consequences and fighting for those affected by diabetes. The Association funds research to prevent, cure and manage diabetes; delivers services to hundreds of communities; provides objective and credible information; and

# Practice Transformation for Physicians and Health Care Teams

[www.YourDiabetesInfo.org/  
PracticeTransformation](http://www.YourDiabetesInfo.org/PracticeTransformation)

*Practice Transformation for Physicians and Health Care Teams* (formerly called *Better Diabetes Care*) is a free online resource designed for health care professionals and administrators who want to change systems of health care delivery around diabetes.

Newly updated by the NDEP!

Practice change is essential to provide evidence-based care recommended by the Patient-Centered Medical Home (PCMH) model and to manage issues related to diabetes and its complications. This resource provides models, links, and tools to help physicians and health care teams initiate and maintain quality improvements in their health care practice.

Content featured on this site is based on current, peer-reviewed literature and evidence-based clinical practice recommendations.



**NDEP** National Diabetes Education Program  
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gives voice to those denied their rights because of diabetes. Founded in 1940, our mission is to prevent and cure diabetes and to improve the lives of all people affected by diabetes.

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# Wound Care & Hyperbaric Medicine

## A 5-Year Follow Up to the Essential Skills for Targeted Limb Salvage: Being DRRAFTed

Ryan Fitzgerald, DPM, FACFAS and David McArthur, C.Ped, CFo

### Introduction

The incidence of diabetes mellitus worldwide has reached nearly epidemic proportions, with nearly 29 million affected by the disease in the United States alone and more than 366 million people worldwide.<sup>1,2</sup> Current projections suggest that by 2030, at least 550 million people will have diabetes—approximately 10% of the world's adult population. In concert with this increasing incidence, there has been a significant rise in the observed comorbidities commonly associated with the disease process in patients living with diabetes.<sup>3-6</sup> Among these complications, lower-extremity manifestations are a significant source of patient comorbidity, mortality, and healthcare expense.

It has been estimated that the lifetime risk of developing diabetic foot ulceration (DFU) is as high as 25% in patients living with diabetes.<sup>1,7</sup> In addition to the development of DFU, more than 50% of these ulcerations will become infected, accounting for nearly 20% of all diabetes-related hospital admissions, and therefore a significant portion of healthcare-related costs—nearly \$11 billion in 2001.<sup>8-11</sup> In those patients presenting with infected DFUs, underlying osteomyelitis is observed in as many as 65% of cases, and these infected ulcers constitute a major risk factor for non-traumatic lower-extremity amputation (LEA).<sup>7,11,12</sup> Indeed, nearly 83% of all non-traumatic lower-extremity amputations in the United States are secondary to complications associated with diabetes mellitus.<sup>7</sup> It has been well documented that the consequences of major lower-extremity amputation in diabetics are severe, with an estimated 5-year post-operative survival rate of less than 50% suggesting that the mortality rate associated with diabetic LEAs exceeds that of most cancers.<sup>13</sup> It is therefore vital to provide early and effective diagnosis and manage-

ment of patients with lower-extremity complications of diabetes in an effort to stem the current epidemic of limb loss.

Interdisciplinary models have been demonstrated to be highly effective in reducing the incidence of non-traumatic amputations in the diabetic population.<sup>14-19</sup> Considering that the pathophysiology of lower-extremity limb loss is multifactorial and that vasculopathy and neuropathy are critical contributors, it is appropriate to utilize an interdisciplinary team approach to specifically address the varying factors that combine to create this spiral of lower-extremity ulceration, infection, and subsequent amputation. In 2009 the author defined seven vital abilities that a diabetic rapid response acute foot team (DRRAFT) should have in its armamentarium so that it might effectively manage the lower-extremity complications of diabetes.<sup>20</sup> Five years later, it is now prudent to revisit this team model to evaluate the efficacy and real-world effectiveness of such an interdisciplinary team.

### A Model for Change: The Greenville Experience

The diabetic assessment rapid response team is an interdisciplinary team model whose core involves the ability to rapidly diagnose and provide effective treatment to patients presenting with lower-extremity complications of diabetes utilizing basic skill-sets necessary for limb preservation. It has previously been advocated that the “irreducible minimum” regarding interdisciplinary units be oriented around treatment teams that are staffed by members of the vascular surgery and podiatric surgery specialties, with adjunctive team members being added as necessary via judicious use of consultation.<sup>14,16-18</sup> The original DRRAFT concept is the natural extension of this premise:

Bringing the nuances from each individual specialty, the team collectively must possess the ability to perform seven essential skills to be effective in promoting limb preservation. In practice, however, an eighth skill has been identified as critical for successful long term patient outcomes. The Greenville Health System (GHS) Center for Amputation Prevention in Greenville, SC is an interdisciplinary limb salvage team that incorporates the elements of the original DRRAFT concept with podiatric and vascular surgery, as was described in the original DRRAFT article. Unique in this model is the addition of an in-house prosthetics and orthotics team that can provide a long lasting, healthy environment for patients predisposed to ulcerations. An emphasis in aggressive and continued management of the patient's footwear, from custom insole to Charcot restraint orthotic walker (CROW), is critical to the long term success of sustained wound remission and reduced reoccurrence rates.

## DRRAFT 2.0 –8 Essential Skills for Limb Preservation

The management of the lower-extremity manifestations of diabetes mellitus is a complex task, and it is vital that practitioners involved in diabetic limb salvage address both the systemic and local factors that interact to generate significant comorbidity and mortality in this patient population. The major factors include vasculopathy and neuropathy, often in combination with foot deformity, and lead to the development of DFU.<sup>21-23</sup> The literature is clear that infected diabetic ulcerations present a major risk factor for lower-extremity amputation, and therefore it is necessary to appropriately manage DFUs when they occur. This includes addressing the underlying etiology as well as dealing with any infection that may be present.<sup>24</sup> Eight essential skills have been identified that are utilized in combination by DRRAFT members to effectively manage DFUs when they occur and prevent progression to lower-extremity amputation.

The DRRAFT team model was originally designed to provide for seven specific skill-sets: 1. the ability to perform hemodynamic and anatomic vascular assessment with revascularization, as necessary; 2. the ability to perform neurologic workup; 3. the ability to perform site-appropriate culture technique; 4. the ability to perform wound assessment and staging/grading of infection and ischemia; 5. the ability to perform site-specific bedside and intra-operative incision and debridement; 6. the ability to initiate and modify culture-specific and patient-appropriate antibi-

otic therapy; 7. the ability to perform appropriate post-operative monitoring to reduce risk of reulceration and infection. These skills address the predominant issues commonly observed in chronic, non-healing DFUs.

Patients with diabetes often suffer from peripheral arterial disease with elements of micro-vascular and macro-vascular disease,<sup>25-27</sup> although it is predominantly macrovascular disease that produces critical limb ischemia (CLI). Patients with CLI are at significant risk for limb loss and require timely intervention to improve distal lower-extremity perfusion.<sup>28,29</sup> Delay in the recognition and treatment of macrovascular occlusive disease compromises outcomes, delays wound healing, prolongs hospital stay, and unnecessarily increases the risk of major limb amputation.<sup>25</sup>

In addition to circulatory issues, diabetic patients often develop neurologic symptoms as a consequence of long-standing hyperglycemia; these include motor, sensory, and autonomic neuropathy.<sup>21,22,36-38</sup> These symptoms are involved in many levels in the development of lower-extremity ulcerations. Perhaps the most widely recognized of the neurologic symptoms common to diabetics is sensory neuropathy with loss of protective sensation (LOPS).<sup>3</sup> These patients lose the "gift of pain." In the absence of pain, diabetic patients are far more likely to develop ulcerations due to LOPS in the context of increased shearing forces. Additionally, motor neuropathy in the intrinsic musculature can lead to muscle imbalance, which creates deformity that, in conjunction with sensory neuropathy, can lead to the development of areas of increased forces, which can progress to ulceration. DRRAFT members can appropriately evaluate the patient's neurological status to identify LOPS via sensory neuropathy as well as any elements of motor or autonomic neuropathy present that can contribute to the development of lower-extremity ulceration.<sup>1,40</sup>

Considering the morbidity and mortality associated with infected DFUs, it is vital that the clinician be able to reliably obtain useful culture data. The literature demonstrates that diabetic lower-extremity infections are often polymicrobial, with an average of 2.25 pathogens per patient.<sup>10,42</sup> Furthermore, superficial swab cultures taken from a wound are notoriously unreliable; one study demonstrated that superficial swabs of infected ulcerations identified deep soft tissue pathogens in only 75% of cases.<sup>43</sup> It is necessary that suitable deep cultures be obtained to appropriately direct antibiotic therapy. Almost all patients present-

ing with mild (and some moderate) infections can be treated using oral antibiotics with fairly specific activity against aerobic gram positive organisms. Patients presenting with more severe infections should initially be placed on empiric, broad antibiotic coverage until more focused therapy can be initiated based on appropriate culture results.<sup>10,50</sup> The majority of the moderate to severe lower-extremity infections in this patient population are polymicrobial, and considering the increasing rates of antibiotic resistant strains of pathogens, it is vital that these patients receive appropriate antibiotic coverage. Toward this end, it is vital that DRRAFT members be able to effectively select appropriate empirical therapy and modify patient antibiotic regimens in response to accurate culture and sensitivity data.

Following appropriate assessment of vascular status and assessment of potential infection, it is necessary that DRRAFT members be able to provide timely incision and drainage to decompress areas of abscess formation as well as to provide appropriate debridement to remove all infected, nonviable, and necrotic soft tissue and bone. Such debridement allows the clinician to limit the proximal spread of infection and obtain deep specimens for culture as well as allow for tissue demarcation in those zones of tissue compromise.<sup>15,22</sup> Appropriate and timely tissue debridement has the ability to turn the tide of infection and to set the patient down the road toward reconstructive efforts, particularly in the context of staged lower-extremity reconstruction and wound healing.<sup>53-56</sup>

Initial wound healing is winning the battle; however, the war rages on in these high-risk patient populations and it is necessary that DRRAFT members be able to provide continued offloading solutions for DFU patients. It is well understood in the literature that diabetic patients with previous ulceration have a significantly increased risk for reulceration, and it is necessary that DRRAFT members have the ability to actively follow these high-risk patients throughout the acute post wound healing phase into continued wound remission.<sup>58-60</sup> The use of an in-house orthotic and prosthetic department with imbedded pedorthists and prosthetists allows for protective offloading through all phases of wound healing and subsequent wound remission. From custom offloading insoles and offloading postoperative shoes to CROW boots and partial foot prostheses, these team members provide continued and comprehensive offloading solutions to reduce the risk of subsequent ulceration.

## Conclusion

The DRRAFT model proposes the essential skills that form a necessary core of the interdisciplinary limb salvage model. These skills provide for the rapid diagnosis and timely surgical management of diabetic patients presenting with lower-extremity compromise and should be the foundation upon which any interdisciplinary team be built. As the population ages and lifestyles change, the incidence and prevalence of diabetes mellitus are increasing, and therefore it is incumbent upon clinicians involved in the care of patients living with diabetes be adequately prepared to provide efficient, quality care to prevent lower-extremity ulceration, infection, and progression toward amputation. The development of an ulcer is a pivotal event in the life of the patient, and it is necessary that DRRAFT members provide the necessary management post-ulceration to address the specific ulcer while also reducing risk for further ulceration.

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