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WOUND CARE AND HYPERBARIC MEDICINE

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INSIDE THIS ISSUE

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NOTE FROM THE PUBLISHER

Publisher
John S. Peters, FACHE

In This Issue…

You will notice our new look in this issue of *WCHM* magazine. As we continue to search out practical applied topics in wound care, diving, and hyperbaric medicine, we strive to provide you with the most current, up-to-date information, and our new format reflects that endeavor.

The series on physiological and psychological diving stresses returns on Page 4, with Drs. Alcock, Khalsa, and Strauss addressing the travel and surface stresses affecting divers in the predive phase of a dive trip.

Dr. James explains the unique role of hyperbaric oxygen treatment for stroke patients on Page 15.

We welcome Dr. Heather Hettrick with Part 1 of her Wound Geography and Tissue Types series. Her journey begins on Page 22. The video course is also available for 1.0 nursing CE credits. See the article for all the details.

On Page 24 Dr. Strauss contributes another article in this issue in which he describes his experiences with gentian violet as a wound dressing agent.

We polled continuing medical education (CME) accreditation experts for their insight on whether to provide courses in person or online for CME credits. Read the responses from the Undersea and Hyperbaric Medical Society (UHMS) and the Wilderness Medical Society (WMS) on Page 30.

An excerpt from *Hyperbaric Facility Safety: A Practical Guide* by Rudy Prunea on Page 33 provides insight and guidance for hyperbaric facilities. The article discusses staffing needs and selection, center design, equipment, and marketing. If you are starting a new center, this is a must-read article.

As we focus on advancing the knowledge and practice of wound care, diving, and hyperbaric medicine, join us in delivering the highest-quality publication in the industry by sharing *WCHM* magazine with colleagues and clients. Add your clinic to our Map of Wound Care and HBO Centers (www.bestpub.com). If you are a part of an exceptional hyperbaric or wound care center, contact us today to be our next featured clinic.

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WCHM
WOUND CARE AND HYPERBARIC MEDICINE

Stresses in Scuba and Breath-Hold Diving

Part VI: Predive and Surface Problems of Divers

By Dr. Joe Alcock, MD, MSCR, FAAEM; Dr. Satkirin Khalsa, MD;
and Dr. Michael Strauss, MD, FACS, AAOS

This series of *Wound Care and Hyperbaric Medicine* has focused on the various physiological and psychological stresses that affect divers. This section concerns the travel and surface stresses affecting divers in the predive phase of a dive trip. We will review the behavioral and physiological responses to travel and jet lag, travel-related infections, and sun and heat disorders. We will also discuss the most common cause of death for travelers on dive trips: cardiovascular disease. We suggest ways to mitigate these stresses to improve the health of divers and allow them to get the most out of their dive experience.

Travel Stresses

Divers frequently travel long distances to dive locations. Travel itself can be experienced as pleasurable or stressful or both, although recent studies suggest that air travel in particular is often experienced as a negative and stressful experience.[1,2] Features that predict air-travel stress include (a) anxious reactions to air-travel events, (b) negative interactions with other travelers, and (c) a lack of trust in the ability of airlines/airports to provide comfort and safety.[1] Travel itself can elicit a physiological stress response involving sympathetic nervous system activation and cortisol production.[3]

Laboratory simulations of long-haul flights, using crowded conditions and hypobaric or normobaric hypoxia, have been shown to result in increased stress catecholamines, transient immune impairment, sleep disturbance, fatigue, and mood changes.[4-6] Although aircraft pressurization and crowding are unlikely to change, careful planning and other coping strategies can mitigate the adverse effects of travel. Successfully resolving these stresses can increase the pleasure from diving and leisure activities, the enjoyment of which has been associated to increased psychological and physical well-being.[7]

Just as safety concerns can add to air-travel stress, the safety capabilities of dive boats can raise concerns. Most North American dive operators have sophisticated navigation, emergency communication, and safety equipment on board. In developing worlds, where many popular diving destinations are located, a wide degree of variation may be observed for boat maintenance, communication capacity, and safety and emergency medical equipment. Combined with language barriers, these features can vastly influence whether a dive trip is experienced as stressful or pleasurable.

Is Jet Lag an Issue for Divers?

Jet lag is an underappreciated source of stress that can affect a dive trip. Circadian rhythm disruption and jet lag are often unavoidable during long-distance travel over many time zones. The normal pattern of sleep involves an eight-hour sleep cycle with cycles of REM and deeper sleep. The normal timing of sleep is determined by circadian physiology and circadian stimuli known as *zeitgebers* ("time givers" in German). The most important zeitgeber is light exposure, which sends signals to the suprachiasmatic nucleus (SCN). Output rhythms from the SCN affect physiology and behavior, including the timing of sleep. Changes in light exposure accompanying travel over many time zones causes circadian disruption and sleep loss, as well as daytime sleepiness.[8]

Sleep restriction causes weight gain and is linked to systemic inflammation. Long-term sleep loss has been associated with a variety of chronic diseases, including hypertension, increased risk of cardiovascular events, and cancer.[9] In the shorter term, circadian disruption is associated with mood disturbances, impairment of concentration, and mental fatigue.[10] These occur with sleep of shorter duration and increased awakenings, both of which are described in jet lag. Jet lag can induce depressive symptoms that can be treated or prevented with melatonin and melatonin analogs.[11] Findings indicate that even a one-hour change in sleep duration can make a big difference.[9]

The stress of jet lag can be ameliorated by several modalities. Melatonin taken at the sleep time of the destination for several days prior to travel has been shown to make a difference in acclimatization to the new time zone.[8] Mainly, recovery from jet lag is accomplished with exposure to daytime light to provide circadian input to the brain and suprachiasmatic nucleus.

Two other stimuli that have been recognized as important for entraining circadian rhythm are social activity and food. Socializing during the day hours also helps entrain a new circadian rhythm. A nap can reduce jet lag and help adjust the circadian rhythm if daytime sleep is not excessively long. Exercise, especially outdoors, is recommended.[12] Yoga and meditation can also help resolve the stress of travel and time change. Finally, an increasing body of work has linked the gut and intestinal microbes to circadian rhythm and the physiological changes seen in jet lag.[13] Recovery from jet lag may occur faster by eating at mealtimes appropriate for the new time zone, ensuring adequate dietary fiber, and by avoiding a high-fat diet and excessive alcohol.[8]

Does Stress Predispose Divers to Decompression Illness?

Besides determining whether a trip is pleasurable or not, physical and psychological stresses have hematologic and immune effects that may alter the risk of decompression illness (DCI). Increased DCI from emotional stress has not yet been demonstrated, but evidence suggests a plausible mechanism for such a link.

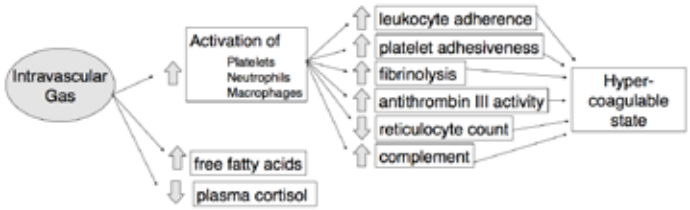
1. Emotional stress and sleep disturbance are linked with platelet activation, resulting from increased exposure to the catecholamine stress hormones norepinephrine and epinephrine.[14]

2. Catecholamines, e.g. epinephrine, are well-known stimuli of platelet activation and aggregation when microbubbles are present.[15]

3. Platelets are critically important in promoting thrombosis from bubbles during decompression illness.[15] Bubbles cause platelet aggregation and clotting in animal models of decompression[16,17] and in humans.[18] After decompression, platelet adhesiveness increases, free platelet counts decreases, antithrombin III activity decreases, fibrinolysis increases, all markers of a hypercoagulable state[16] (*Figure 1*). In addition to promoting thrombosis, bubbles activate immune pathways that induce inflammation,[19] exacerbating tissue damage during DCI.[20,21]

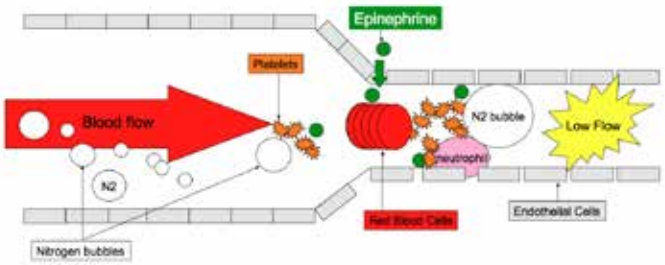
Because bubbles and catecholamine exposure are potent activators of platelets, travel stresses outlined in this section have the potential to increase harm from activated platelets in DCI. A model showing this hypothesized mechanism is shown in Figure 2. Whether stress hormones worsen DCI has yet to be proven. Either way, it probably makes for a more enjoyable dive when stress and stress hormones are low.

FIGURE 1. Activation of platelets and clotting by bubbles



Gas bubbles activate immune cells, complement, and platelets, causing a hypercoagulable state.

FIGURE 2. A model for how exposure to stress hormones interact with bubbles to increase risk for DCI



Nitrogen bubbles interact and stress catecholamines (epinephrine) activates platelets, white blood cells, and endothelial cells, resulting in vasoconstriction, microvascular hemostasis, and tissue hypoxia in DCI.

FIGURE 3. Yoga for divers

Yoga and breathing exercises (usually performed above water!) are a good way to resolve diving and travel stresses.

Travel Medicine: What's New in Travel-Related Disease?

One important concern for travelers is the potential exposure to travel-related diseases. Because travel medicine risks vary by location, the reader is urged to consult *CDC Health Information for International Travel* ("The Yellow Book") before planning a dive trip.[22] Because of space constraints, we limit this discussion to travelers' diarrhea and the mosquito-borne diseases dengue fever and chikungunya. Adequate preparation for these infectious threats can help prevent a trip-ending illness.

Travelers' Diarrhea

Gastrointestinal upset and diarrhea are common infectious complications that can occur to any traveler in the predive stage or at any other time on the trip. Travelers' diarrhea (TD) occurs in 30-50% of travelers and may be exacerbated by dietary changes, exposure to infectious disease, as well as physiological stress of travel and jet lag. TD is most often caused by enterotoxigenic *E. coli* (ETEC). ETEC and other bacteria account for 80-90% of TD. *Campylobacter* is a common cause of TD in some destinations, including Thailand. Other causes include the protozoan *Giardia intestinalis*, responsible for a more indolent and persistent diarrheal illness. A notable viral cause of TD is norovirus, which causes epidemic gastroenteritis among travelers, including passengers on cruise ships.

Uncomplicated TD does not involve fever or bloody stool and is generally a self-limited disease. While self-limiting, TD is common, debilitating when present, and can potentially ruin a diving trip. In particular, TD is often associated with dehydration, which puts divers at risk for decompression illness (DCI).

Travelers can prepare for the eventuality of TD by bringing antibiotics, preventative medications, and antidiarrheals (*Table 1*). Ciprofloxacin reduces the duration of symptoms, although increasing resistance to this medication has been reported. In locations with high ciprofloxacin resistance, the nonabsorbed antibiotic rifaximin is an alternative.[26] Travelers should be aware that resistance to rifaximin in *E. coli* also has been reported recently.[27] Azithromycin is safe for treatment of TD in pregnant women and children. Besides these prescription medications, over-the-counter bismuth preparations such as Pepto Bismol® have been shown to help prevent and reduce symptoms of TD.[28] Probiotics, such as lactobacilli, also have proven effectiveness and should be considered for prevention of TD.[28,29] If symptoms appear, an antimotility agent such as loperamide is reported to be safe for TD and may prove essential when a bathroom is unavailable.[30] Loperamide and Lomotil® should not be used if fever accompanies diarrhea. Antimotility agents also are not recommended for children. Oral hydration with solutions such as Pedialyte® is helpful for bacterial TD and is the main treatment for epidemic norovirus gastroenteritis.

TABLE 1. Medications for Travelers' Diarrhea

MEDICATION	INDICATION	COMMENT
Trimethoprim/ sulfamethoxazole	Treatment of TD	Antibiotic resistance is common. Not recommended for prevention.
Ciprofloxacin	Treatment of TD	Antibiotic resistance is increasing. Not recommended for prevention.
Rifaximin	Treatment and possible prevention of TD	Antibiotic resistance is emerging. May be used for prevention. Expensive.
Bismuth subsalicylate	Treatment and prevention of TD	Over the counter. Recommended.
Probiotics	Prevention of TD	May also be used for treatment.
Oral fluid replacement	Treatment of TD	Pedialyte® or equivalent. Ideal for viral diarrhea.

Dengue Fever and Chikungunya Infection

Mosquito-borne viral diseases are common and increasing in frequency in many popular diving destinations, especially the Caribbean.[23] Dengue fever is caused by an arbovirus carried by the mosquito *Aedes aegypti*, resulting in between 50-100 million cases per year. Sufferers develop a fever and an erythematous macular rash that soon becomes confluent. The common name for dengue disease is "breakbone fever," which, as the name suggests, involves myalgias and arthralgias as well as headache. This classic presentation is seen mostly in adults; children can have a mild flu-like or asymptomatic presentation. More rarely, dengue infection can progress to dengue hemorrhagic fever, resulting in thrombocytopenia, vasculitis, capillary leak syndrome, and shock.[24] Unfortunately, no vaccine or specific treatment is available; fluid therapy and supportive care are mainstays of treatment.

Another increasing problem at many diving destinations is chikungunya virus, carried by the same mosquito that transmits dengue.[25] Chikungunya transmission was unknown in the New World until 2013. Since then, chikungunya infections have reached epidemic proportions in most Caribbean islands.[23] In the United States, most cases are seen in returned travelers, although sporadic cases of endemic transmission have been reported.

Solar Injury FAQs

1. **What is sunburn?**
   Sunburn erythema is a cutaneous inflammatory response accompanied by DNA damage and cytotoxicity. Microscopy may reveal edema, vasodilation, and endothelial cell swelling. "Sunburn cells" have enlarged nuclei and vacuolated cytoplasm, appearing several hours after exposure.[32] Histamine is released, and mast cells degranulate, contributing to the warmth, pain, and discomfort of sunburn.

2. **Besides sunscreens, what can mitigate sunburn?**
   Antioxidants given before, but not after, exposure can decrease erythema, but their effect on DNA damage is unknown. A majority of randomized trials have concluded that topical NSAIDs, antihistamines, and oral steroids are ineffective, shortening the duration of sunburn.[33]

3. **What is the MED?**
   MED stands for mean erythema dose and is the smallest amount of UVR, generally UVB, that can cause discernable dermal erythema. MED is often measured in time. For example, MED may be 20 minutes of exposure in a fair-skinned person. DNA absorption of UVB correlates with extent of erythema.

4. **What is a tan?**
   Immediate pigment darkening occurs from melanin precursors that are preformed, generally in darker-skinned people. Delayed pigment darkening occurs from UVB and UVA (mostly UVA) when melanocytes proliferate and melanin synthesis occurs.

5. **Is the age of solar exposure important in developing skin cancer?**
   Yes. Australians who arrived from Britain younger than 18 have high rates of basal cell carcinomas. If they arrived older than 18, immigrants to sunny Australia had skin cancer rates similar to their British counterparts.[34] Intermittent exposure to sun, fair skin, and red hair are risk factors for skin cancer.

6. **Does high cumulative lifetime exposure to UVR cause melanomas?**
   Increased occupational exposure over a lifetime decreases the risk of melanoma, the most deadly form of skin cancer. Lower latitude and intermittent high doses of UVR, especially at young ages, correlate with melanoma risk. Melanoma is associated with increased number of nevi; nevi numbers increase with childhood UVR. Whether regular sunscreens reduce melanoma and other skin cancers is uncertain and remains controversial. Sunscreens have not conclusively been shown to reduce the risk of melanoma or other cancers.[35,36]

Originally from Africa, chikungunya is a risk for travelers to that continent, as well as to the Indian Ocean and Western Pacific. In addition to sharing the same vector as dengue, chikungunya causes a similar clinical syndrome to dengue fever. A high fever and myalgias are common. Incapacitating joint pains are often prominent in chikungunya disease. Joint pains may last for weeks or months after the initial infection. Treatment, which involves rest and fluids, is directed at symptoms of the disease. Preventing bites from mosquitoes by using permethrin clothing, DEET-containing repellents, and screened air-conditioned rooms reduces the risk of both dengue and chikungunya.

Solar Injury

Sun exposure causes sunburn, one of the most common conditions experienced by divers. Sunburn is more common in light-skinned individuals, but all divers should take measures to protect their skin and eyes from the sun.

Clearly, solar radiation is not all bad. Some UV exposure is important, because lack of UV inhibits vitamin D production in the skin and can lead to deficiency syndromes such as rickets. Vitamin D and exposure to the sun are linked with a reduced risk of a variety of chronic diseases, including cardiovascular disease, diabetes, and cancer. On the other hand, solar ultraviolet radiation (UVR) causes sunburn and eye injuries that can commonly affect divers and other travelers.

UVR comes in two varieties relevant to human health. UVB measures 290-320nm in wavelength and is responsible for vitamin D production from 7-dehydroxycholesterol via the skin, liver, and kidney. UVB also causes tanning, burning, and some skin cancers. UVA has longer wavelengths (320-400nm) and contributes to photoaging, tanning, burning, and cancers. UVA is also responsible for the phototoxicity of certain pharmaceuticals.

The amount of UVA and UVB that reaches the skin is affected by time of day, season, and clouds. The majority (65%) of UVR falls between 10 a.m. and 2 p.m., so efforts to protect skin from the sun should focus on those hours. Season is also important. In northern latitudes, June has 100 times more UVR than December. Latitude makes a difference in UVR exposure, with higher intensity in tropical locations.[31] As one moves away from the equator, UVR decreases 3% with every degree of latitude. Additionally, surface features such as water and sand reflect UVR. Areas such as the chin, lips, and nose may require protection from reflected light. Albedo is the reflection of solar radiation from white objects, especially snow, reflecting as much as 85% of visible light and UVR. Compared with snow, water reflects less UVR. Clouds attenuate UV radiation by 20-80%,

Sunscreens

1. **What is the meaning of SPF?**
   SPF stands for skin protection factor. SPF is calculated by the ratio of MED (mean erythema dose) — measured in time — of skin with sunscreen divided by the MED of unprotected skin.

$$SPF = \frac{\text{MED protected skin}}{\text{MED unprotected skin}}$$

2. **What number SPF should I look for? Is higher better?**
   SPF 15 blocks 93% of UVB. So, in theory, it should be fine. However, in practice the SPF may be much lower than advertised because of sweating, abrasion, inadequate application, etc. So a higher SPF gives a bigger margin of error. Several real-world and clinical studies suggest that using higher SPF (at least SPF 30) gives increased protection — at least from sunburn.

3. **Does the DEET in insect repellant affect my sunscreen?**
   Yes, DEET reduces the efficacy of sunscreen by about 30%. This is another argument for higher SPF sunscreens. In endemic areas, there is a trade-off between mosquito protection (e.g., chikungunya risk) and sunburn protection.

4. **How do sunscreens work?**
   Sunscreens have two mechanisms of action. Physical blockers are generally metals (e.g., zinc or titanium) that reflect and scatter UV radiation away from the skin. These physical agents often appear visible (e.g., white, yellow), but newer micronized formulas are transparent on skin but still reflect/scatter UVR. Physical agents are effective on both UVA and UVB.

   The second way that sunscreens work is via chemical agents that absorb solar radiation. These are the most common active ingredients in sunscreens. They are often degraded by the process of absorbing energy and are often used in combination to improve their stability.[40]

5. **Don't some sunscreens cause rashes, even allergies?**
   One of the first sunscreens developed was PABA. It is no longer widely used because it sensitizes the skin in about 1 in 20 people. Sensitized people will develop photo eruptions and photo allergy. Despite the discontinuation of PABA, sunscreens are still the No. 1 cause of photoallergic reactions.[41]

6. **My sunscreen says it is water resistant. What does that mean?**
   This is an FDA-regulated claim. It means that the sunscreen should retain its SPF rating after 40 minutes of exposure to water or 80 minutes of water exposure as indicated on the label.

generally 40%, which may not be enough to protect from sunburn.

Protection from the sun involves protective eyewear and the use of sunscreens (*Table 2*). However, in warm climates sunscreen effectiveness is compromised by sweating, toweling off, and frequent entry and exit from the water. In those settings, protective clothing and hats are recommended while on land or topside. Sun protection from clothing is variable, depending on the kind of fabric and weave. Synthetics are generally better UV blockers than cotton. On the other hand, denim SPF equivalent is greater than 200, while a T-shirt may be as low as SPF 4. Certain brands (e.g., Solumbra®) make clothing specifically designed to protect from solar UV. These clothes are labeled with UPF, which is similar to SPF; higher UPF indicates better protection from sunburn.

TABLE 2. Sun Protection

SUN BLOCKER	EXAMPLE	COMMENT
Sunscreen physical blockers	Titanium or zinc	Often opaque, but microsphere preparation does not appear white. Block UVA and UVB. Can harm corals
Sunscreen chemical filters	Oxybenzone benzophenones	Block UVA or UVB. Photosensitivity common. Harmful for corals.
Stretch fabric shirt/suit	Rash guard, Lycra or spandex	Generally 50+ UPF. Provides sting protections Safe for corals
Wetsuit	Many brands	50+ UPF. Thermal protection. Provides sting protection. Safe for corals.
Hat	Wide-brimmed preferred over baseball cap for UV protection.	Topside only (although surfing versions exist)
Sunglasses	Look for sunglasses that advertise UV protection.	Protect from UV keratitis. Unknown effectiveness for cataracts and pterygia.

For divers, lycra suits and wetsuits provide thermal protection (from cold) as well as sun protection. Lycra and wetsuits also offer excellent protection against stings from venomous marine organisms such as jellyfish. Relying on wetsuits/clothing for sun protection has another important benefit relating to marine conservation. Divers should be aware that their activities, including the use of sunscreen, adversely affect coral health.[37] Sunscreens have been shown to cause mortality, promote viral infections, and induce

bleaching in corals.[38] For these reasons, fabrics are preferred to sunscreens in sensitive coral reef areas and where marine stings are anticipated.

For most people, sunglasses are useful for protection against ocular overexposure of UVB. Photokeratitis of the cornea is characterized by corneal edema, corneal surface defects, and blurred vision. Although regular eyeglasses provide some UV protection, most sunglasses block more than 99% of UVR. Sunglasses may provide protection from cataracts and premature aging of the eye, although these benefits remain controversial.[39]

FIGURE 4. Clothing sun protection

Lycra rash guards and wetsuits provide excellent sun protection as well as protection from stinging marine organisms. Unlike sunscreens, fabrics are nontoxic to corals.

Heat Stress

The sun is a source of heat as well as UV radiation. Humans deal with hot environments by shedding heat in four ways: radiation, conduction, convection, and evaporation. Humans rely largely on evaporative cooling with sweating, a strategy that does not work as well in hot and humid environments or when clothing or wetsuits prevent evaporation. Underwater, direct contact with water usually results in heat loss by conduction. Rarely, very warm water causes net heat gain. Heat gain from ambient water is a stress that the human body is poorly equipped to cope with.

Of the two most important heat syndromes, heat exhaustion is serious but less life-threatening than heat stroke. Heat exhaustion is characterized by dehydration induced by sweating and increased demands on the cardiovascular system. Evaporative fluid losses generate a high workload for the heart, especially because cardiac output can increase to 20 liters per minute during acute heat stress. Heat exhaustion is usually reversible by replacing fluid losses and removing the person from the hot environment.

Heatstroke, the most severe form of heat illness, is defined as mental status changes accompanied by a core temperature greater than 40°C. Exertional heat stroke is a shock state caused by thermal injury to critical organs, including the gut. Cells die because of direct thermal damage, apoptosis, inflammation, and systemic coagulation.[42] Thermal injury causes damage to intestinal epithelial cells, reduces tight junction integrity, and allows bacteria to translocate from the bowel into the blood.[43] These result in systemic inflammation resulting from endotoxemia, endogenous pyrogens, and elevated levels of the cytokines IL-1 and TNFα. The presence of bacterial products in the blood and elevated pro-inflammatory cytokines in heat stroke highlights its similarity to septic shock, which is a clinical mimic of this condition.

Heatstroke requires emergent treatment including active cooling measures. Ice-water immersion is both fast and effective and is the best immediate method of cooling for heatstroke.[44] However, this technique introduces difficulties in monitoring and resuscitating patients. Other treatments include evaporative cooling using cool mist and fans, with the caveat that high ambient humidity limits its effectiveness. Ice packs can be placed on the groin and axillae, and cooled IV saline can be given if available. Antipyretics such as acetaminophen and ibuprofen are not useful in heatstroke.

In addition to cooling the patient, heatstroke victims are uniformly dehydrated and can present in shock. Hypovolemia should be treated aggressively with fluids. Rhabdomyolysis that is a common complication of heat stroke also benefits from volume resuscitation while arranging evacuation to hospital.

Risk to Divers: Heart Disease and Other Drivers

Although this review has focused on common travel risks for divers (jet lag and sunburn) and high-profile travel-related diseases (chikungunya virus), it is worth noting that the most likely causes of mortality for travelers are heart disease and trauma.[45] In other words, what can kill you on a dive trip is the same as what can kill you at home. Cardiovascular disease accounted for more than 50% of travel deaths in a large North American sample, whereas travel-related infectious disease caused less than 1%.[45] Trauma accounted for 27% of deaths among travelers from the United States and Canada.[45,46] By contrast, the risk of DCI was 0.03% per

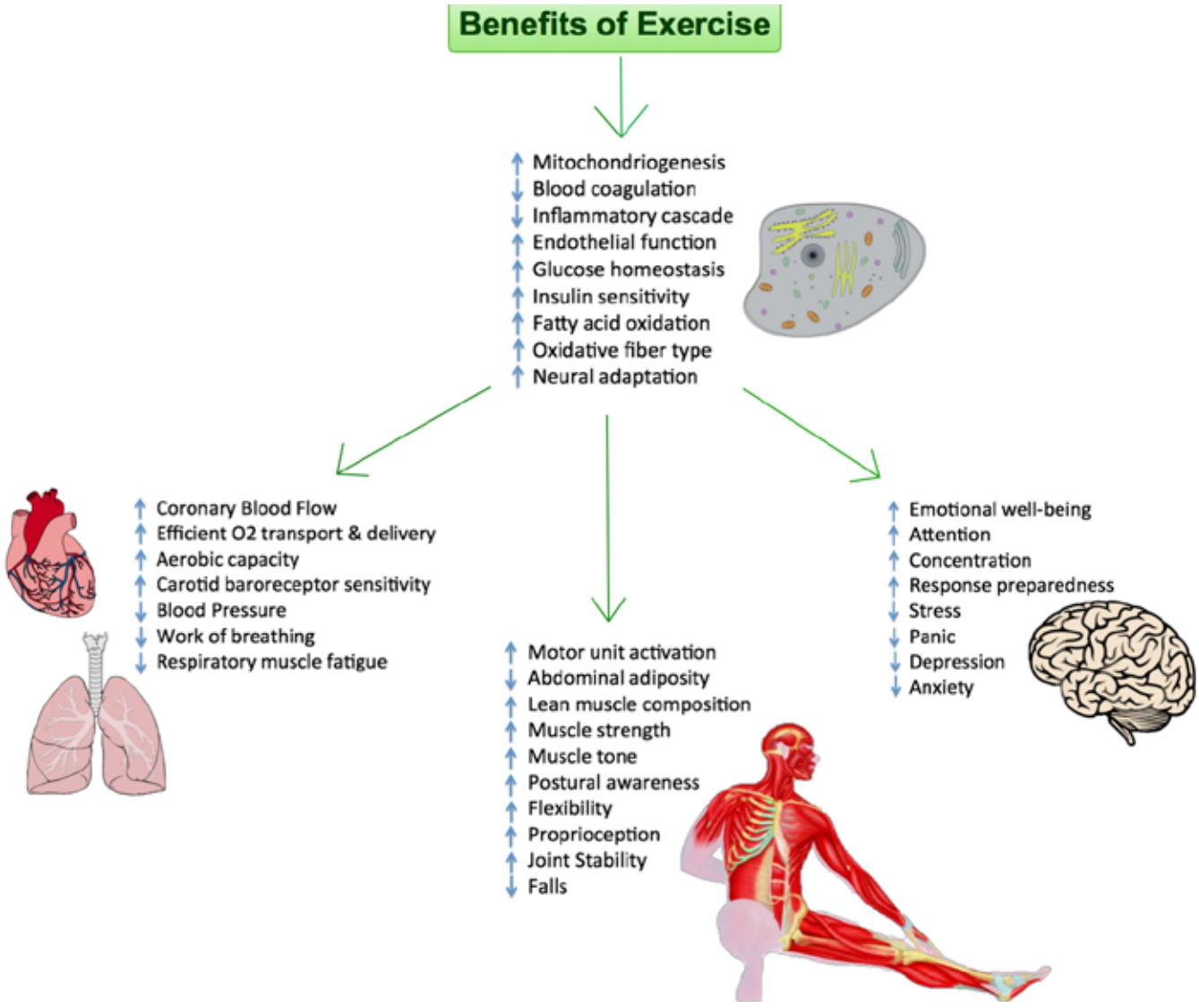
dive in a series of recreational divers reported by Divers Alert Network.[47] DCI accounts for a negligible source of travel mortality, mostly from air embolism.[47] Many deaths that occur during diving result from drowning, and about 20% of those can be attributed to in-water cardiovascular events.[48]

The other mortality risk for travelers is trauma. A special kind of trauma occurs when divers collide with boats. Being struck by boat propellers causes devastating injuries and carries a mortality rate of 15-23%.[49] Although serious, boat accidents are relatively rare. By contrast, traffic accidents are a major cause of trauma mortality. In the developing world, a recent increase in the number of cars on the road combined with poor road infrastructure has resulted in an epidemic of traffic deaths.[50] Compounding this problem, road and automobile safety features are poor, and access to emergency health care is often subpar in developing countries as compared with developed nations.[50] These problems make automobile and road trips the most dangerous part of travel to a dive destination in the developing world — certainly a greater risk than DCI. Bottom line: To stay safe on a dive trip, attend to cardiac risk factors, stop smoking, exercise, and wear your set belt!

Ways to Cope with Stress: Physical Fitness, Breathing and Conditioning

Traditionally, exercise during and immediately after diving has been disfavored because of a concern that it increased nitrogen uptake and decompression stress.[51] Contradicting this concern, the majority of research during the past 15 years shows a protective effect of regular and acute bouts of exercise for DCI.[52-55] In a counterexample, Madden

FIGURE 5. Benefits of exercise



Exercise has benefits on multiple organ systems, including the immune system, cardiovascular system, muscles, and brain.

and colleagues showed that vigorous exercise after diving can result in the arterialization of bubbles, resulting in subclinical air embolism, likely through intrapulmonary shunts.[56] On balance, the benefits of regular exercise likely outweigh the reported risks. Exercise is recommended for physical fitness and general health and has benefits for mental health and cognitive functioning. Figure 5 summarizes these benefits to divers and travelers.

The physical and psychological stresses of travel are commonly experienced by divers, particularly during overseas travel. These stresses are an expected and normal response to being in an unfamiliar and unpredictable environment. Divers can prepare by increasing physical fitness through exercise, which has been associated with reduced levels of stress hormones,[57] improved sleep,[58] enhanced recovery from jet lag,[12] and reduced anxiety.[59]

Exercise and regular physical activity have a variety of benefits for divers. Exercise training is linked with increased vagal tone, improved heart-rate recovery after exercise, and a reduction in cardiovascular events.[57] In addition, exercise combined with breath control (e.g., yoga, qigong, and tai chi) have been shown to reduce baseline parameters of stress and reduce anxiety.[59] These mind-body practices, involving movements of the upper chest, shoulders, and accessory muscles of breathing, also improve recovery from the stress response.[60] Performed regularly, they may have the added benefit of reducing the work of breathing at depth.[61]

During travel, there are many factors that divers cannot predict or control, but one exception is their own ventilation. Breathing techniques can minimize predive stress and anxiety while also improving ventilatory efficiency. These techniques also augment exercise tolerance and fin-swimming capacity throughout the dive.[61-63] Practice with controlled breathing and respiratory muscle training has beneficial neuroendocrine effects. These include activating stretch receptors and vagal activation in the peripheral and central nervous system that inhibit sympathetic nervous activity.[64] These practices, as part of a physical exercise program in the months prior to travel and diving, are a useful strategy to improve flexibility and fitness for diving and can help divers get the most out of their dive travel experience.[52] ■

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Hyperbaric Oxygen Treatment for Stroke Patients

By Philip B. James, Emeritus Professor of Medicine

Stroke is an ill-defined lay term that has found its way into general use in medicine and derives from the old English word "stricken." It relates to the abrupt onset of neurological symptoms, usually a "hemiplegia" with half of the body, most commonly the left side, developing weakness or paralysis. Speech and the muscles of the face may be affected, causing drooping of the mouth. Such symptoms are easily recognized, but, unfortunately, strokes may also affect cognitive function and cause loss of memory and even personality changes often only apparent to near relatives.

Strokes are the equivalent in the brain of a heart attack; indeed they have often been referred to as "brain attacks," and both are due to the lack of oxygen caused by a reduction of blood flow. As heart attacks may be abruptly fatal, the need for emergency treatment is obvious; nevertheless, patients admitted to the hospital with a stroke are now more likely to die than those who suffer a heart attack. Clearly, if outcome is to be improved, the same urgency is needed in stroke treatment. Strokes are the now the scourge of the Western world: In England and Wales, with a combined population of about 58 million, about 53,000 patients die from a stroke each year, with more than 450,000 surviving with severe disability. The estimated cost of their long-term care is more than £9 billion a year.

Strokes mainly affect older people and are associated with the disease of the lining of arteries known as atherosclerosis, which results from a high-sugar and high-fat diet. The damage reduces the diameter of the vessel and attracts platelets — essential components of clotting — causing thrombosis. Debris from affected areas may dislodge and flow down the artery to lodge and reduce or block blood flow. The mechanism is known as thromboembolism. Unfortunately this disease process is now affecting people in middle age. The Stroke Association in the UK has recently reported that the number of strokes occurring in men between the ages of 40 and 54 has increased by almost 50% in less than 15 years.

Strokes may also occur when material breaks off damaged heart valves and when clots form in the heart. This is most commonly associated with stagnation of blood in the left atrium in patients suffering from the irregularity of contraction known as atrial fibrillation. Fibrocartilagenous emboli from spinal disc degeneration may enter the circulation and cause stroke at any age and is well-recognized in veterinary medicine. Younger stroke patients must also be investigated for the antiphospholipid syndrome known as Hughes syndrome because it can be treated with aspirin. Stroke can also be associated with a persisting hole in the heart, which allows the trapping normally undertaken by the lungs to be bypassed; these holes can now be closed without open-heart surgery. Even a baby still in the womb may suffer a stroke leading to a cerebral palsy evident after birth, although most strokes in newborns are the result of a birth injury.

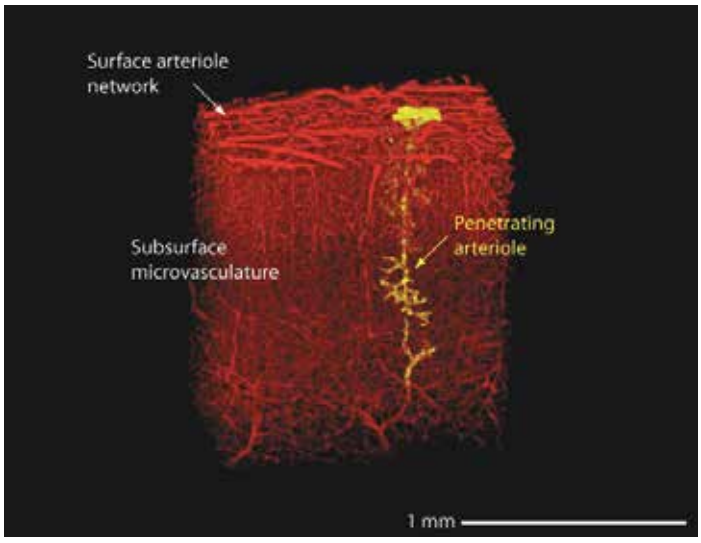
The symptoms typical of a stroke are not always associated with partial or complete blockage of a major blood vessel in the brain; symptoms indistinguishable from stroke may affect younger patients eventually labeled as having multiple sclerosis — only the age of the patient and a history of other symptoms allow it to be distinguished from a stroke. In this case the reduction of oxygen delivery is due to tissue swelling; the increased water content limits the transfer of oxygen from blood into the tissues.

The only drugs approved for the treatment of stroke are the thrombolytics, popularly known as "clot busters," which aim to restore blood flow in thromboembolic stroke. The need to restore flow is to improve oxygen delivery, and it would seem obvious that giving patients more oxygen to breathe should be helpful. However, the only measurement made in clinical practice is of the so-called "saturation" of the pigment hemoglobin with oxygen, and most doctors will

allege that when the value is 100% there is no value gained from supplementing air with more oxygen. However, this reflects a primary failure in medical education because it is only the oxygen dissolved in plasma that is responsible for the transport of oxygen from blood into cells. The amount dissolved can be safely increased from a typical value of 100 mmHg breathing air at sea level to more than 2000 mmHg breathing pure oxygen in a simple pressure chamber. Also, not in medical consciousness is the fact that oxygen controls both cardiac output and blood flow.

The collection of 3 trillion cells said to comprise the body requires a vast hydraulic system of blood vessels to stay alive, and the brain itself contributes about 100 billion. Despite being only 2% of body mass, blood flow through the brain requires 20% of the cardiac output, and the brain is responsible for 20% of the blood oxygen consumed. The most important function of the cardiovascular system is to deliver oxygen and, because of the unique vulnerability of the brain to a lack of oxygen, the provision of blood vessels is very generous; astonishingly, a cubic millimeter of the cortex of the brain of a mouse contains a thousand capillaries (*Figure 1*). In practice this means that brain cells can survive if a capillary is blocked by circulating debris, an event that is probably more common than we recognize judged from the "unidentified bright objects" commonly seen on MRIs.

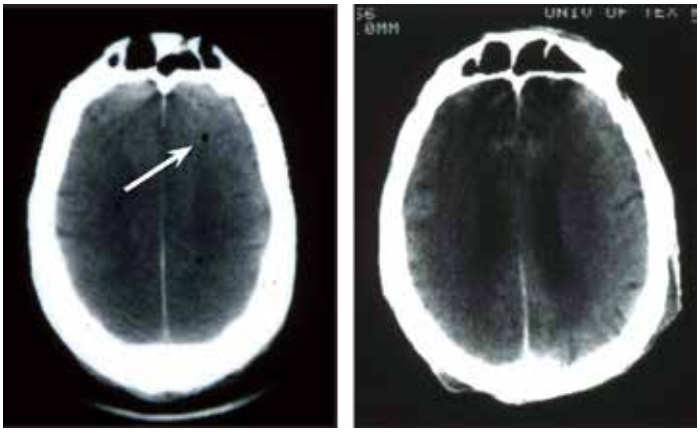
FIGURE 1



The capillary network in the gray matter of a mouse brain (Courtesy of Drs. Tsai and Blinder)

Air embolism in divers is one cause of stroke where the use of hyperbaric oxygen treatment is well-established and, as treatment is normally prompt, it is usually very effective. The recommended pressure is 2.8 atmospheres absolute (ATA). Air embolism is often overlooked clinically but may complicate many invasive medical procedures, especially open-heart surgery. Air embolism of the middle cerebral artery (arrowed) occurred in the case of the patient whose CT image is shown in Figure 2. It followed an attempt to insert a central venous line. Because of a delay in the use of hyperbaric treatment, the right brain hemisphere (right image) was extensively damaged.

FIGURE 2



CT images: air embolism of the right middle cerebral artery

Clearly the compression of gas bubbles reduces their size, redistributes them in the circulation, and assists in their reabsorption. The lack of oxygen and the associated inflammation is also reduced by hyperbaric oxygen treatment.

There can be no question that procedures like the US Navy Treatment Table 6 (TT6), which use oxygen at 2.8 ATA, have been largely successful in treating acute air embolism and also neurological decompression sickness, although there is an important problem associated with such a high oxygen pressure that is rarely discussed — it may cause deterioration of neurological symptoms. This is almost certainly due to the vessel constriction produced limiting blood flow "upstream" of the damaged tissue, which is responsible for convulsions at 2.8 ATA. The pressures used, especially for chronic neurological disease, are controversial, reflecting the lack of understanding of the physiology involved. Although the oxygen delivered is usually 100% and the treatment pressure is set accurately, it does not indicate the oxygen level present in the arterial blood, which determines the response to treatment. This is mainly because of the mismatch between the areas of the lung engaged in ventilation and those through which blood is flowing. This is known as the "ventilation/perfusion ratio," but, unfortunately, it cannot be measured during treatment.

The actual level of oxygen a patient breathes also depends upon the delivery system; for example, masks are notoriously

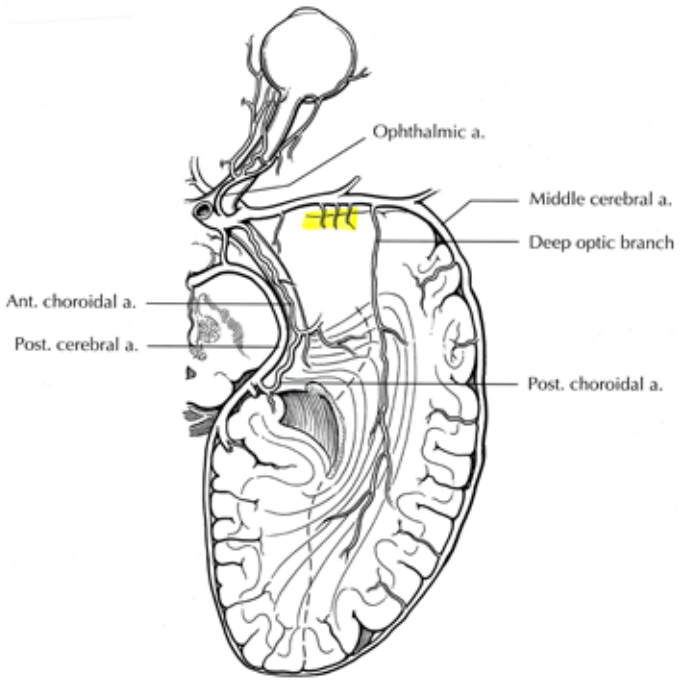
prone to leakage. An indication of the variation in blood oxygen level comes from measurements made in a New York University study in 1983. Using a well-designed full-face mask at an accurate chamber pressure of 2 ATA, the lowest blood oxygen level measured was 1.11 ATA and the highest was 1.59 ATA — almost 45% higher. Clearly, the chamber pressure determines the maximum level possible breathing 100% oxygen, but not the minimum level.

There are also variations in the transfer of oxygen into damaged tissue because of the inevitable involvement of the internal microcirculation. The patient's response can be seen by looking in the eye; in some patients there is a very marked reduction of the diameter of the arteries of the retina; in other patients the reduction is minimal. Once an oxygen deficiency has been corrected, the oxygen level should be lowered so that the constriction of the blood vessels does not limit the transport of other agents in blood vital for recovery. This approach is used in TT6; the sessions of oxygen breathing at 2.8 ATA are interrupted by breathing chamber air because of the risk of a convulsion, and after three or four cycles of oxygen breathing at 2.8 ATA, the oxygen pressure is reduced to 2 ATA. Divers are usually young and fit and their condition is acute. In trials of hyperbaric oxygen treatment in stroke, the delay to treatment has often been many hours, and by this time use of high oxygen pressure is inappropriate. The treatment of patients with multiple sclerosis and mild traumatic brain injury over the past 40 years has confirmed beyond doubt the effectiveness of oxygen pressures lower than those used for divers. In general, the longer treatment is delayed, the lower the oxygen pressure that should be used. Military doctors recommend treatment at a pressure of 2 ATA for the subtle residua post treatment that may be detectable only on psychometric testing. Importantly, much animal evidence indicates that a pressure of just 2 ATA is as effective in resolving acute air embolism, and, as in other forms of stroke, the window of opportunity is just a few hours.

The symptoms described as a stroke may result from rupture or, more commonly, a partial or complete obstruction of an artery. The arteries in the brain have thinner walls than those in other parts of the body, and rupture may occur if blood pressure rises, leading to hemorrhage, and pressures as high as 250 mmHg can be generated by straining. Arteries may be obstructed by an embolus or by a thrombosis that forms on an area of damage to the artery lining associated with the common disease atherosclerosis. In both conditions, hyperbaric oxygen treatment may be valuable. Animal studies support the use of oxygen in brain hemorrhage, and the author has personal experience of a very positive outcome in a diver who suffered an intracranial hemorrhage from a ruptured berry aneurysm after completing a shallow dive. Because it was not possible to eliminate gas embolism due to pulmonary barotrauma as the cause of his symptoms, he was treated on TT6 and regained consciousness breathing oxygen at a pressure of 2.8 ATA. However, he lost consciousness as pressure was reduced and, after assessment at a local hospital, was transported by helicopter to a regional neurosurgical unit. The surgeon was astonished at the diver's rapid recovery post surgery given the severity of the hemorrhage seen on CT imaging. The downregulation of neutrophil activity and inflammatory genes by oxygen provides a sound scientific basis for hyperbaric oxygen treatment in subarachnoid hemorrhage.

The blockage of blood flow in an artery of the brain only causes a major problem when other blood vessels cannot compensate for the reduction in blood flow: Flow must be rapidly restored to allow the transport of critical oxygen to resume. Most of the arteries of the brain connect to other arteries — they anastomose — which can minimize damage from a blockage. Unfortunately, vessels in the middle of the brain, known as the lenticulostriatal arteries, do not have such connections, which leave the areas of the brain they supply, including those known as the internal capsules in the middle of each hemisphere, vulnerable. They are known as "end arteries," and the optic nerves and areas of the spinal cord also have end arteries. The lenticulostriatal arteries in one hemisphere are highlighted in Figure 3. Damage to the fibers passing through these areas is the most common cause of stroke.

FIGURE 3



Brain arteries: Lenticulostriatal arteries highlighted are not anastomosed. (Courtesy of Dr. James Toole)

Clot-busting drugs, the only helpful treatment, must be used within an hour of the onset of symptoms to be properly effective; after four hours, they are not beneficial and increase the risk of bleeding. Their use is effectively restricted to just 5% of stroke patients because of this complication. Because of this time constraint, the treatment of stroke will never be entirely satisfactory; about a third of patients with stroke die, and, sad though it is for the loved ones, this limits the human effort expended and the costs to society. A third of patients will eventually make a reasonable recovery and regain their independence, but this leaves the remaining third as survivors in need of constant care, often in an institution.

The human and financial costs are truly enormous and, with the population aging, are getting ever greater. If the treatment of stroke could be improved with fewer patients suffering prolonged disability, much suffering could be avoided and enormous savings made. Attempts to develop drugs for stroke by three major drug companies — Pfizer, Glaxo Smith Kline, and Astra Zeneca — have been abandoned after billions of dollars in investment have not yielded results. As it is universally recognized that a stroke is caused by lack of oxygen, the question arises: Can giving oxygen under hyperbaric conditions with clot-busting drugs improve the immediate treatment of stroke patients?

Oxygen delivery obviously depends on blood flow, and when the heart stops, the arrest of blood flow to the brain could be termed a "global" stroke. It is commonly stated that unless the heart is restarted, the brain dies in four minutes. This is not the case; detailed postmortem studies published in a research letter in *The Lancet* in 1998 titled "Recovery of Axonal Transport in Dead Neurons" have shown that brain cells remain viable for many hours in culture provided that oxygen and glucose are supplied. The damage occurs when blood flow returns after the heart is restarted; this is called reperfusion injury. A heart removed from a donor must be used within four hours; otherwise it deteriorates, leading to the death of the recipient. Heart transplant research in the 1970s has shown that the damage to the heart occurred when blood flow was reestablished due to the release of oxygen free radicals. For many years biochemists pondered the obscure ways in which free radicals may be involved, but the answer was provided by research that followed a world event — a little girl named Jessica fell down a well in Midland, Texas. Her blackened leg was saved by hyperbaric oxygen treatment. Controversy followed — it was suggested in a letter published in *JAMA*, despite the leg being saved, that a high level of oxygen in a pressure would have worsened her condition because of free radicals. In fact, giving more oxygen prevents the formation of oxygen free radicals, and it is an intriguing story that involves white blood cells called neutrophils.

Following the controversy, Dr. William Zamboni received funding to study hyperbaric oxygen treatment in limb salvage. Using a ligature to stop blood flow in an experimental model, he watched events under a microscope when flow was released after four hours. White blood cells returning with the blood flow began to stick to the walls of the veins, eventually blocking flow and leading to the death of the muscle. However, a large dose of oxygen delivered under hyperbaric conditions stopped the white blood cells from sticking, and the muscle survived. Dr. Zamboni reported that he could reimplant severed human limbs successfully up to 12 hours post injury. The fall of the oxygen level attracts neutrophils to the affected area, and, if the lack of oxygen is not relieved, they release free radicals, which damage the walls of blood vessels as they are programmed to do in infection. This undoubtedly happens as a stroke develops and may lead to the most serious complication, the bleeding into the tissues of the brain, known as a hemorrhagic stroke. The red blood cells are broken down, and the iron released from their hemoglobin generates the most toxic of free radicals, the hydroxyl radical, causing the death of cells in their vicinity.

It is critical to progress for it to be recognized that these events occur entirely within the confines of blood vessels, and they apply to the restoration of blood flow in any organ — including the brain. Hyperbaric oxygenation is the key to preventing this problem because it stops neutrophil aggregation and, hence, their release of free radicals in the tissues. The case for using high levels of oxygen in acute stroke together with thrombolysis is supported by sound science, including a large number of animal models of stroke, most using rats, which, with a brain weight of only 2 grams, represent a "worst case" situation.

The question then remains: Is there benefit to stroke patients beyond the acute stage with long-term disability? Here it is necessary to discuss a concept first proposed by neurologists in the 1980s that has since been demonstrated by SPECT imaging. Judged by the certain yardstick of the pathology that results from stroke, some brain tissue dies and slowly is replaced by scar tissue. However, a much bigger volume of tissue is affected and remains for years "not dead but sleeping." It is termed the ischemic penumbra. The brain cells survive because the brain has two circulations: one of blood, and the other of the tissue liquid known as cerebrospinal fluid (CSF). The CSF can assist in maintaining low levels of oxygen, ensuring that neurons, while not having sufficient energy available to function, can survive. However, for new capillaries to grow and establish an adequate blood

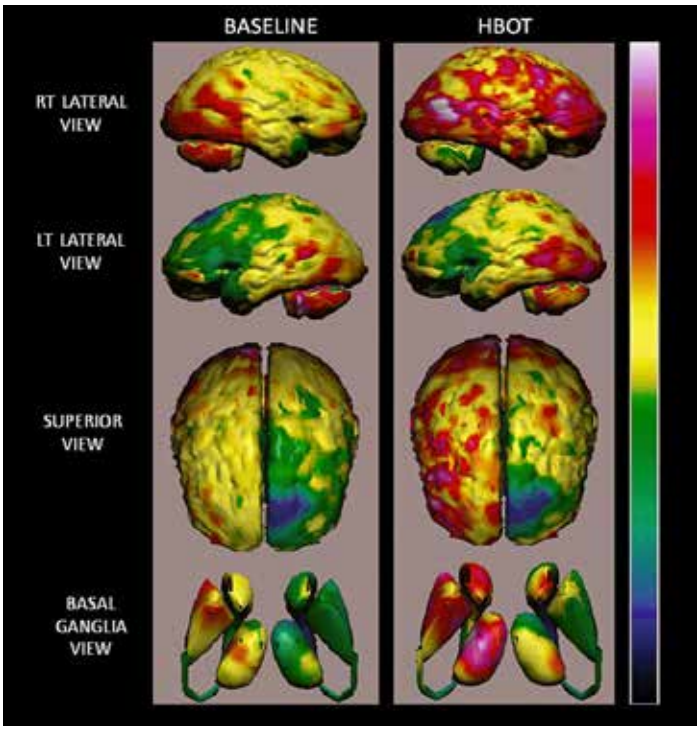
flow for function requires additional energy and a greater availability of oxygen. It is now well-established that the adult human brain contains stem cells capable of rebuilding the brain by forming new cells. Also, bone marrow stem cells can migrate into the brain, as they can into the heart, but, as with drug delivery, their migration is hampered when blood flow is reduced and the water content of the tissues increases due to swelling. Long-standing tissue swelling can be reduced by hyperbaric oxygen treatment, allowing in stem cells to rebuild damaged areas, which makes a substantial difference to both the extent of permanent damage and the speed of recovery.

Several studies of hyperbaric oxygen treatment in acute stroke patients in the 1960s and '70s showed that patients often improved while in the chamber breathing oxygen. In most cases, the patients had only a single treatment, and, not surprisingly, the improvements usually disappeared shortly after the session was completed. There was very limited understanding of the biology of oxygen at the time, and no studies were undertaken using a course of oxygen treatment to see if the improvement would persist. There were, however, notable exceptions to this approach. Dr. Edgar End in Milwaukee County Hospital, who rarely published his work, started treating stroke patients in the 1940s, and he had seen sustained improvement from courses of treatment. In 1980, he collaborated with Dr. Richard Neubauer to publish the results from the treatment of 122 patients in the journal *Stroke* using what were regarded at the time as low oxygen pressures — that is, from 1.5 to 2 ATA. This was a critical move away from the much higher pressures used in diving and many benefits were found.

Drs. Neubauer, Gottlieb, and Kagan described the benefit of a course of hyperbaric oxygen treatment for a 60-year-old female patient in a letter titled "Enhancing 'idling' neurons," published in *The Lancet* in 1990. The patient had suffered a stroke 14 years previously but regained the ability to live independently, and her improvements correlated with remarkable changes evident on SPECT imaging undertaken before and after treatment. The images identified still-viable brain tissue in the area surrounding the zone of tissue death. It is telling that the letter in *The Lancet* did not generate any correspondence, but the concept has been confirmed by further imaging studies. In 2007, an issue of the journal *Neurological Research* was dedicated to detailed papers on the use of oxygen treatment in neurological conditions, including stroke.

With drugs failing to show benefit, the need to focus efforts on oxygen, the only proven neuroprotective agent, is all too apparent, and a comprehensive study has recently been completed in the Assaf Harofeh Hyperbaric Centre near Tel Aviv, Israel. SPECT imaging was used before and after treatment. Figure 4 shows the 3D brain images of a 72-year-old man who still had right-sided weakness 34 months after a stroke. He was unable to hold either his right arm or leg against gravity and had little movement of the fingers of his right hand. He could only communicate with single words, being unable to complete sentences. A course of 40 sessions of hyperbaric oxygen treatment was undertaken using 100% oxygen by mask at 2 ATA five days a week. The session time in the chamber was a total of 90 minutes.

FIGURE 4



3D SPECT images of a 72-year-old man shows improved brain blood flow post 40 HBOT sessions. (Courtesy of Dr. Shai Efrati et al Assaf Harofeh Medical Centre Israel)

After the course of treatment, he was able to hold both his arm and leg against gravity and to move his fingers. His speech was significantly improved with the ability to complete sentences. The baseline 3D SPECT image of the brain prior to oxygen treatment shows a wide area of penumbral brain tissue (green regions) around the stroke area (blue region) involving part of the left motor cortex, which correlates with the impairment of his right leg and hand function. The left cortex of the frontal lobes, Broca's area, which is responsible for speech, also shows improved blood flow, as do the areas in the midbrain known as the basal ganglia. To put his treatment into perspective, it must be remembered that the sessions took a total of 60 hours — that is, less than a week of his life. The use of SPECT imaging provides spectacular evidence (pun intended) of the

importance of oxygen in restoring viable brain tissue. Given the cost of institutional care, these results desperately need to be translated into practice, as many patients will be able to retain their independence. The care in the community model developed for multiple sclerosis patients provided in 65 centers in the U.K. can provide an inexpensive way of adding life to the years that medical science has made possible.

The known science relating to the fundamental role of oxygen in healing stands in stark contrast to the lack of knowledge of its actions in the medical community. The unique role of oxygen in controlling the circulation — the output of the heart and blood flow — and the regulation of our key genes must be taught in our medical schools. ■

References

All references in this article can be found in the following publication:

James PB. *Oxygen and the Brain: The Journey of Our Lifetime*. North Palm Beach, FL: Best Publishing Company, 2014.

About the Author

PHILIP B. JAMES, MB, CHB, DIH, PHD, FFOM, qualified in medicine from Liverpool Medical School in 1966 and after a fellowship in surgical research studied industrial medicine in Dundee, Scotland. After Royal Navy training in 1973, he specialized in diving medicine, combining an academic post in the University of Dundee with consultancies to many international diving contractors. In 1983, he received the Craig Hoffman Award from the Undersea and Hyperbaric Medical Society for diver paramedic training and contributions to diving safety, including a minimum oxygen content in helium, high oxygen partial pressures in divers' emergency supplies, and the use of helium/oxygen mixtures in recompression treatment. In 1983, he published evidence for subacute fat embolism as a cause of multiple sclerosis in *The Lancet*, comparing the pathology to decompression sickness and endorsing the use of hyperbaric oxygen treatment.

Five multiple sclerosis patients treated by Dr. James in 1981 founded a community hyperbaric facility in Dundee, and there are now 65 charity centers operating in the UK and the Republic of Ireland, providing low-cost hyperbaric oxygen treatment for neurological conditions. The centers were deregulated by an Act of Parliament in 2008. Dr. James retired in the same year, but he continues as honorary adviser to the charity, as a consultant to the offshore oil and gas industry, and a passionate advocate for using oxygen in treating disorders of the brain.

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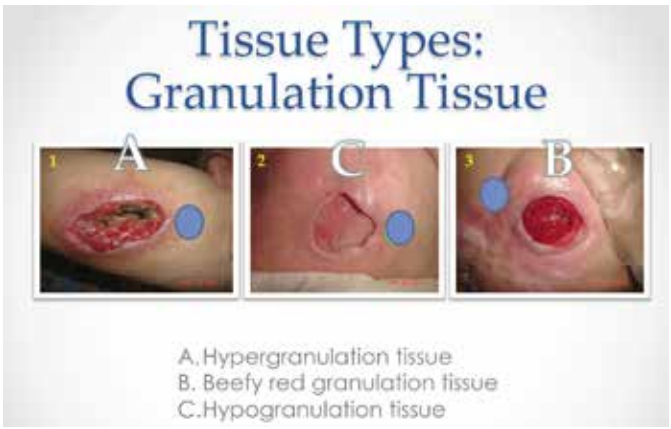
Wound Geography and Tissue Types: Part 1

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

This article (and the accompanying video course available at *www.woundeducationpartners.com/woundgeography*) will introduce you to pictures and ask you to identify the correct answer as information is shared.

Hyper- or Hypogranulation Tissue

FIGURE 1



These pictures represent different types of granulation tissue. Which picture represents hypergranulation tissue, beefy red granulation tissue, or hypogranulation tissue?

Hypergranulation tissue (A) is shown in Picture 1. This is noted by the overproliferation of granulation tissue, which is almost cauliflower-like in appearance. In veterinary medicine, it is called proud flesh. This tissue is an abnormal proliferation of granulation tissue, believed to be due to excess moisture or possibly even an underlying infection. This wound will not close until this hypergranulation tissue is managed.

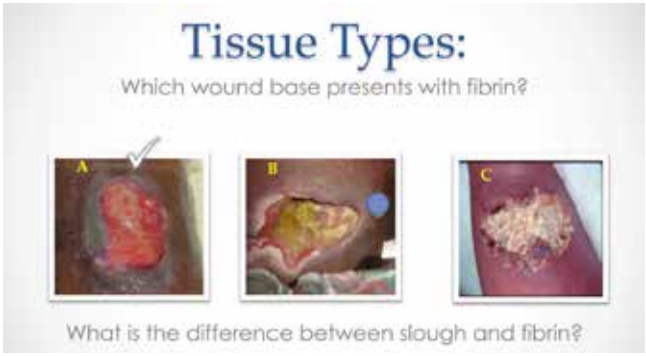
Picture 2 represents hypogranulation tissue (C). The absence of granulation tissue could be due to a lack of oxygen or poor profusion of the tissues, trauma to the area, or an underlying infection. As you can see, there is a frank absence of beefy granulation tissue at the wound base and is therefore considered hypogranulation tissue.

Picture 3 is representative of beefy red granulation tissue (B). This is the type of tissue we strive to achieve during full-thickness wound healing when we are doing good, comprehensive wound management. The tissue is healthy, red, viable, well-perfused, and it's not a hyper- or hypoproliferative. This wound is on a good trajectory to resolve, if all else remains equal.

Note with hyper- and hypogranulation tissue that both of these are abnormal pathologies associated with granulation tissue production. It is important to address underlying facts contributing to these forms of granulation tissue for wound healing to occur. Depending upon the type of tissue (or tissues) present, we can direct our interventions according to wound bed preparation principles.

Difference Between Slough and Fibrin

FIGURE 2



Which picture represents a tissue commonly mistaken or described as slough? What is the difference between slough and fibrin? Which wound base presents with fibrin?

The correct answer is A. Fibrin is technically not slough but a "glue-like" protein present in the body. It is the scaffolding for granulation tissue and a component of the extracellular matrix. Fibrin can appear light yellow in color, and it tends to be firmly adhered to the base of the wound.

Viable, moist fibrin is not necrotic tissue. However, if the wound bed is not managed properly through moist wound healing, then the tissue may convert and become nonviable. When looking at these pictures, try to discern the difference between fibrin and slough. Slough tends to be stringy and

sometimes a thick yellow tissue. Fibrin tends to be thin and tightly bound to the wound base. Fibrin is scaffolding for granulation tissue and does not need to be debrided.

Many people use the terms fibrin and slough interchangeably, and there is a lack of consensus about the true differences between these two tissues. New theories are emerging that state that slough and possibly fibrin are byproducts of biofilm. Until more evidence exists, just be descriptive, and note the characteristic differences between these tissues.

Different Types of Eschar

FIGURE 3

These pictures represent different clinical presentations of eschar. With these pictures, identify and describe the different types of eschar for proper documentation. Eschar can present in different ways, depending upon its moisture content. Even though these photos depict different forms of eschar, they are all representative of nonviable tissues.

Picture A should be documented as dry, hard, intact leathery eschar. It has the texture and consistency of beef jerky. Hard, dry eschar that is unstable should be removed to facilitate wound healing.

Picture B should be described as soft, lysing, tan eschar. It is important to objectively describe what you see. This eschar is already starting to soften and break down, possibly due to autolytic debridement and/or moist wound healing. If you visualize the wound like a clock, with the top of the picture being 12:00 and the bottom of the picture being 6:00, note there is significant drainage at 6:00.

Picture C is representative of dry gangrene. This commonly occurs in critical limb ischemia due to lack of perfusion. When it presents dry and stable, it is best to protect and monitor the area, keeping it dry and free from trauma.

You may note the yellow discoloration on the nails and the dorsum of the foot, which is due to the application of Betadine to keep the tissue dry. It is imperative not to start aggressive moist wound healing with dry gangrene as it will open up a Pandora's box. Follow the directions of your medical director or the attending physician while protecting and monitoring the dry gangrene. This is often self-limiting, and auto-amputation or surgical intervention may be required.

Realize with any type of eschar, the tissue is devitalized and ultimately needs to be removed. The appropriate form of debridement should be based upon the tissue types present and the overall clinical presentation of the patient.

Summary

Part II of this series will appear in the next issue of *WCHM* magazine. We will discuss other tissue types, the wound edge, periwound tissue, moisture associated skin damage versus pressure ulcers, and more. ■

About the Author

HEATHER HETTRICK, PT, PHD, CWS, CLT, CLWT, is an associate professor in the physical therapy program at Nova Southeastern University in Ft. Lauderdale, Florida. As a physical therapist, her expertise is in integumentary dysfunction with clinical specialties in wound, burn and lymphedema management. Her certifications include Certified Wound Specialist by the American Board of Wound Management, Certified Lymphedema Therapist by the Academy of Lymphatic Studies, and dual international certifications as a Certified Lymphedema and Wound Therapist through the International Lymphedema Wound Training Institute.

Dr. Hettrick's work experience includes assistant professor and director of clinical education at the University of New Mexico; vice president of Academic Affairs and Education for Gordian Medical, Inc. (dba American Medical Technologies); clinical assistant professor in the department of physical therapy at New York University; adjunct professor at Drexel University; program coordinator for Burn Rehabilitation Research at the William Randolph Hearst Burn Center at NY Presbyterian Hospita; and a master clinician at the Hospital for Joint Diseases at the Diabetic Foot and Ankle Center.

A past president of the American Board of Wound Management, Dr. Hettrick is currently on the executive committee of the Association for the Advancement of Wound Care. She is program director at Hospital St. Croix in Leogane, Haiti, where she oversees and manages a lymphatic filariasis clinic. She is actively involved in numerous professional organizations, conducts research, and publishes, presents and teaches, nationally and internationally, on integumentary related issues.

Experiences with Gentian Violet as a Wound Dressing Agent

By MB Strauss, CK Jones, SS Miller, and A Daniller

ABSTRACT

Introduction: More than 2,000 products are available for wound dressings, with prices ranging from a few cents to several thousand dollars per application. If one dressing agent is not successful, is a more costly one likely to be effective? This is not necessarily so. We have found that gentian violet (GV) can be an effective, inexpensive option for certain wounds when other agents have not been successful.

Methods: After mitigating deformities, deep infections (bursa, scar, and bone) and ischemia-hypoxia, a small percentage of lower-extremity wounds failed to improve with interventions that ranged from negative pressure wound therapy to active ingredient-impregnated ointments. Serendipitously, we began using GV as a wound dressing agent for certain well-defined wound types that were not improving with other agents.

Results: GV was utilized for the following three nonhealing wound types: 1) small, superficial wounds usually with underlying bony deformities; 2) superficial wounds when moisture control and skin maceration are problems; and 3) ischemic wounds associated with peripheral artery disease and/or vasculitis. Zinc oxide, another inexpensive agent, is often used in conjunction with GV and is applied around the wound margins for its drying effects. Nearly all of the wounds refractory to other dressing agents have either healed or improved enough to become easily managed and allow restoration of functional lower-extremity activities when GV was used as the wound dressing agent. In a couple of situations, other interventions became necessary when new problems such as pathological fracture or unrelated wounds at other sites developed.

Conclusions: We used GV in three subsets of superficial wounds, which had been refractory to other interventions. The clinical improvements we observed using GV now justify using this agent on selected wounds at earlier stages in patients' wound courses. The ease of application and the economies of GV have made this agent an increasingly used intervention in our armamentarium of wound care products.

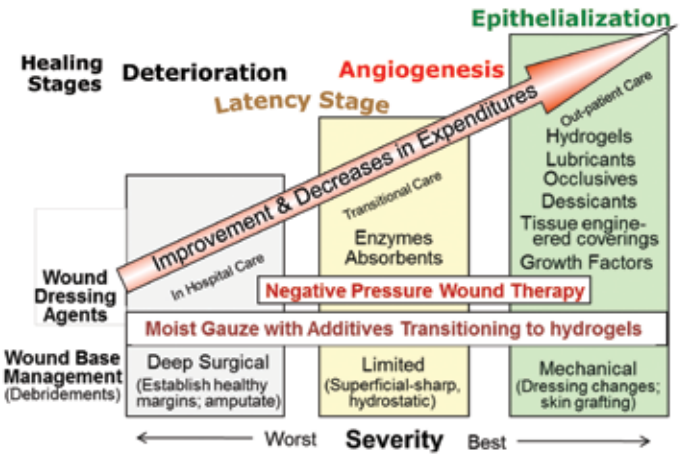
Introduction

In managing difficult-to-heal wounds, the following five strategies must be addressed:[1]

1. Management of the wound base
2. Protection and stabilization of the wound environment
3. Optimal medical management, especially with respect to diabetes management, cardiac activity, and renal function
4. Selection of the wound dressing agent
5. Wound perfusion-oxygenation

Of the five strategies, selection of the wound dressing agent is the most challenging. This is because there are more than 2,000 choices. Often different vendors have products that for all practical purposes have the same mechanisms of action, challenging wounds typically pass through different stages of healing — each stage with a preferred dressing agent. Finally, there are a variety of reasons why wound healing is not progressing. There is an enormous range of charges

FIGURE 1. The continuum of healing in the challenging wound and the changes in wound dressing agents as the wound improves



Legend: As the wound transitions through the different stages of healing, wound dressing agent requirements change in the direction of becoming less complicated to use and less costly. The use of gentian violet fits in this less complicated and less costly category.

for wound dressing agents. They can vary from a few cents to many thousands of dollars for each application (*Table 1*). Caregivers' time for dressing applications must also be factored into the cost of wound management. Consequently, costly agents that require infrequent dressing changes can be more cost-effective than less-expensive agents. When dressings are not complex, willing family members can do or supplement the dressing changes, which is also a cost-effective technique.

With respect to healing of the difficult wound, we have observed four clearly defined stages.[2] These include an initial deterioration stage, which is typically manifested by slough, dehiscence, and/or infection of the wound. The second stage is a resting stage in which wound deterioration is no longer occurring, but improvement is not yet taking place. We label this the latent stage of the healing. Angiogenesis is the predominant finding in the third stage in the healing of the difficult wound. Finally, wound healing culminates with closure and epithelialization.

Several corollaries must be appreciated with the stages of wound healing. First, each stage may require a different dressing agent (*Figure 1*). Second, there is great variation in the times required for each stage to evolve, which may vary from several days to several months. Third, in reality the wound heals in a continuum of responses such that elements of two or more stages may be observed in the healing wound at any one time. We label the wound healing stage based on the predominant finding of the wound base.

Reasons difficult-to-heal wounds fail to improve include ischemia-hypoxia; unresolved deep infection of bone, bursa, or reactive cicatrix formation; underlying deformity; uncontrolled bioburden; malnutrition; matrix metalloproteinases; transudates into the wound base from inadequate edema control with wound margin maceration and inflammation; requirement for steroids or other anti-inflammatory agents; and inadequate compliance with wound care (*Table 2*). Hence, the wound care provider must be aware of all these reasons and base the wound dressing agent selection on what is most important and effective for the particular stage of the healing wound.

If one dressing agent is not successful, is a more costly one likely to be effective (*Table 1*)? This is not necessarily so. After thousands of patients' experiences, we observed that gentian violet (GV) in special circumstances is an effective alternative when other wound dressing agents have not been effective, exceed caregivers' capabilities, and/or become prohibitively expensive. However, this agent should be used only for the appropriately selected wound as described in our methods selection.

This paper describes some of our observations with using GV for three types of difficult-to-heal wounds, the situations in which it has been ineffective, and conditions for which it is a logical choice for the wound dressing agent.

Methods

The information using GV in this paper is observational. It reflects our use in wounds in which healing seemed to be stalled with other agents (*Figures 2 and 3*). GV is an aniline, cationic dye that is recognized by the World Health

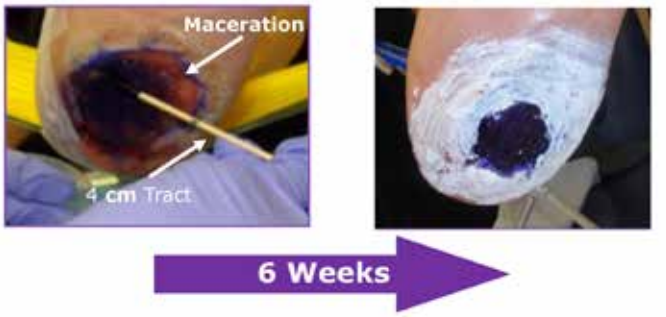
TABLE 1. Examples of wound dressing agents and their costs*

Agent (Examples)	Mechanisms	Estimated Procurement Costs**	Comments
Gentian Violet	Chemical induced anti-bacterial, fungal & helminthic properties; Possibly hinders mitochondrial activity & microbes cell growth;	$3 for 30 cc bottle	Does not appear to interfere with granulation tissue & epithelialization
Antibacterial ointments	Cyclic peptides which disrupt both gram+ & gram- bacteria by interfering with cell wall & peptidoglycan synthesis	$5-$6 for 1 oz. tube	Multiple choices; some such as mupiricin are 5 to 10 times more expensive
Coverings & films (Xeroform®)	Provides a moist environment; some with antibacterial and/or deodorizing effects Mesh types allow exudate egress	$10-$30 for small size wound	Pieces trimmed for additional dressing changes from same package
Hydrofiber (Aquacel-Ag®)	Broad antimicrobial activity due to silver; absorptive properties from fibers	~$70 for box of 10 4 x 4 sq inch pads	As above; typically trimmed to conform to wound
Absorbent (Iodosorb®)	Iodine has antimicrobial activity; starch component absorbs fluids; keeps wound environment moist	~$40 to $65 for 40 gm tube	Dressing changes every 1 to 3 days depending on wound exudation
Honey (Medihoney®)	Maintains moist environment; osmotic effects kill bacteria & reduce edema, promotes healing; induces wound contraction	~$20 for 1.5 oz. tube	Healing effects include angiogenesis, granulation tissue formation, contraction & collagen synthesis
Enzymatic (Santyl®)	Collagenase dissolves protein material in wound base & secondarily stimulates granulation tissue	~$120 for 4 oz. tube	Should be applied daily to the thickness of a nickel on the wound base
Growth factor (Regranex®)	Bioengineered platelet derived growth factor to stimulate many aspects of wound healing	~$600 for 1 oz. tube	Premarket approval based on reduction of wound surface area studies
Negative pressure wound therapy	Micro and macro negative pressure stresses to promote wound healing; removal of secretions	~$100 per day to rent equipment	Absorbent sponge changes twice a week about $60 per set-up
Bioengineered wound coverings	Coverings for healthy based wounds to promote angiogenesis and keratinization. Multiple OR applications are often required	$80 (Oasis®) to $2000 (Dermagraft®) per sheet	In clinic applications possible; others: Niox®,Epifix®, Graphix®, Appligraft®, etc.
Operating room applied (Alloderm®, Integra®, Graftjacket®)	Xenograft derived, some bilayered such with silicon & biodegradable collagen	As above, but $8000-$10000 for operating room	In clinic applications possible; others: Niox®,Epifix®, Graphix®, Appligraft®, etc. Multiple OR applic often required

Notes: *Costs do not include caregiver expenses
**Many of the agents can be used for multiple applications

Organization as a wound dressing agent.[3] It has drying as well as antimicrobial properties, which includes antifungal, antibacterial, and anthelmintic effects.[4]

FIGURE 2. Heel wound size reduction and elimination of maceration



Legend: Improvement (reduction in size & elimination of maceration) in a chronic (2 years), stable heel wound after 6 weeks of daily Gentian Violet applications. The 4 cm tract closed. This occurred after a variety of wound dressing agents including bioengineered skin had been used.

The wound base laid directly on the periostium over the calcaneus with total absence of the soft tissue heel pad. The patient refused surgery to debulk the plantar surface of the calcaneus.

Note the use of zinc oxide (white rim in right photo) used for moisture control of this highly transudative wound.

According to our patients, the most challenging part of using GV was locating a pharmacy that carried the product. Some reported going to two or three pharmacies before they found one that carried GV on the shelf. If special ordered, the price was typically more than mentioned above.

A common use of GV is for managing thrush infections of the mucus membranes, but it is also used for superficial wounds, especially in countries south of the United States border. Before initiating a randomized control trial with our institutional review board, we show in this paper situations in which we have used GV for superficial wounds that are not improving with other agents.

GV is named for color of the petals of the gentian flower. However, the product is not made from the flower. It is a triarylmethane dye. Other names used for GV include crystal violet and methyl violet. It has largely been superseded by other wound- and infection-controlling agents, but our observations may help resurrect it as a remarkably useful wound dressing agent.

TABLE 2. Reasons wounds fail to improve

Reason	Explanation / Mechanism	Interventions
Ischemia-hypoxia*	Lack of oxygen for metabolism and infection managing needs	Revascularization, medical management, hyperbaric oxygen
Unresolved deep infection*	Osteomyelitis, infected bursa and/or infected cicatrix	Debridement, antibiotics
Underlying Deformity*	Mechanical problems, mal perforans ulcers	Surgical interventions
Uncontrolled bioburden	Ischemic, non-viable tissue in wound base	Debridement, wound dressing agents, and antibiotics
Malnutrition	Inadequate substrates for healing, inability to generate a response to infection	Supplements, feeding tubes, hyperalimentation
Matrix metalloproteins (MMPs)	Protein derivatives that interfere / inactivate wound healing responses	Use of MMP inhibitors
Wound transudation	Inadequate moisture control often complicated by hypo-albumenia (malnutrition); skin maceration and infection from the moisture	Edema control, moisture barriers and absorbent agents
Steroids / Anti-inflammatory agents	Interfere with wound healing and infection control responses; for mitigation of collagen vascular diseases	Taper doses, vitamin C
Inadequate compliance	Lack of comprehension, motivation, adequate care and/or insight	Education, assistance in care, frequent re-checks

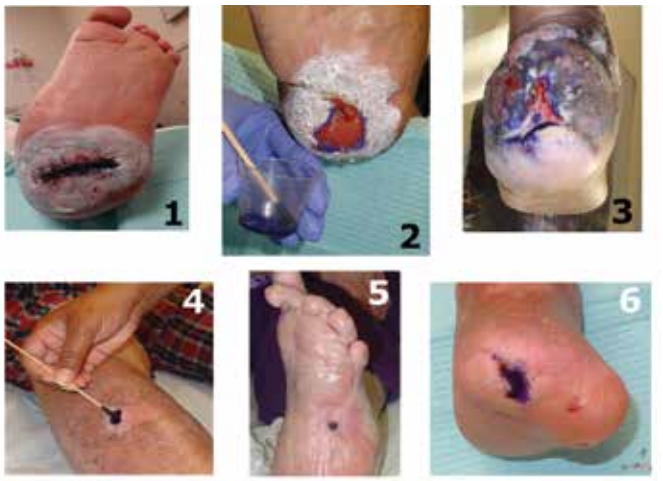
Note: *Over 90% of our patients hospitalized with diabetic foot ulcers had one or more elements of ischemia-hypoxia, deep infection and/or deformity in their hospitalization requiring wounds. We label these three wound healing confounders the "Troublesome Triad."

After mitigating deformities, deep infections (bursa, scar, and bone), and ischemia-hypoxia, a small percentage of lower-extremity wounds fail to improve with customary interventions such as negative pressure wound therapy, bioengineered dressings, silver impregnated ointments, etc. (*Table 1*). For the superficial, chronic stable wound, we began using GV as a wound dressing agent. As our

The utilization of GB in our Wound Healing Center was serendipitous. When first proposed by one of our coauthors (AD), we were skeptical that he was using a "dark ages" remedy for his patients. He decided to use this agent on a refractory heel wound after a variety of wound dressing agents were ineffective in a patient with diabetes mellitus, end-stage renal disease, Charcot arthropathy, peripheral neuropathy and peripheral artery disease. Not only did it simplify wound care, but moisture control (with the use of zinc oxide around the periphery) was markedly improved, and marginal epithelialization of the wound was observed during succeeding clinic visits.

observations increased, we began using GV in a variety of superficial wounds in which other agents had been used, but progress was not occurring. This has evolved to the point of becoming comfortable with recommending GV for specific wounds and defining its indications and contraindications. Thus far, we have used GV as the wound dressing agent for more than 25 patients with difficult-to-heal wounds.

FIGURE 3. "Stalled" wounds in which gentian violet simplified wound care, reduced size of wound, or achieved healing



Legend: GV applications with reasons why it was used: 1) Moisture control, 2) Soft tissue atrophy/absence plus moisture control, 3) Chronic-stable, 4) Ease of wound care, 5) Chronic-stable and 6) Soft tissue atrophy/absence .

Wounds 4, 5 and 6 have healed; GV continued in others because of slowly decreasing wound size and user "friendliness" after using a variety of other wound dressing agents.

Observations/Results

Our observations have demonstrated three situations in which GV has been effective (*Figures 2 and 3*). First, we have used it in a subset of wounds that we label as "chronic-stable," in which the wounds were neither improving nor worsening but were not so severe that they did not appreciably interfere with the patients' activities and lifestyles.[5] These wounds were small — that is, less than 3 cm in diameter — superficial and had vascular bases. Invariably, a variety of other products from films to ointments to bioengineered dressing had been used before switching to GV as the wound dressing agent. Many of the patients in this group had underlying bony prominences, but surgical removal of them was problematic because of concerns about healing or making the plantar surface unstable for weight bearing (i.e., "the cure could be worse than the disease"). The patients in this subset liked using GV for its simplicity of application and economies. In this group, we observed gradual reduction in the surface areas of the wound due to epithelialization of its margins, but only a few healed completely because of the underlying deformity.

A second subtype of nonhealing wound for which GV was an effective wound dressing agent was in wounds in which fluid leakage through the wound base was a prominent feature. This occurred in patients with fluid retention from lymphedema, venous stasis disease, or combinations of these. The surrounding skin margins, because of the fluid leakage, were invariably macerated and inflamed. Many of the patients had courses of antibiotics because it was difficult to ascertain whether the inflammation was from cellulitis or irritation from the maceration. All patients in this group had previously used dressing agents to absorb secretions. Often the dressings became saturated with moisture, smelly, and colonized with bacteria between dressing changes, even if done twice a day. For this group, a one-two-three-four protocol was used. The moist, macerated skin at the wound margins was pared down to healthy-appearing epithelium. Next, the film or fibrous membrane was cureted to a bleeding base. GV was then coated on the wound base. Finally, zinc oxide, an effective moisture barrier and controlling agent, was applied to the previously macerated, debrided skin margins. Like GV, a tube of zinc oxide is inexpensive, costing only a couple of dollars. As in the first subtype, the patients appreciated the ease of application but also were pleased that moisture and odor control were managed so well with this technique.

A third subtype of chronic nonhealing wound for which we have used GV is for the patient with atrophy and/or absence of soft tissue under the ulceration complicated by advanced peripheral artery disease and/or ulcers secondary to vasculitis. This group is differentiated from the first group because of absence of underlying, possibly surgically correctable, bony deformities. GV was used in this group after healing progress was not observed with other wound dressing agents and was preferred by the patient and their caregivers because of the "friendliness" of the GV dressing technique.

Discussion

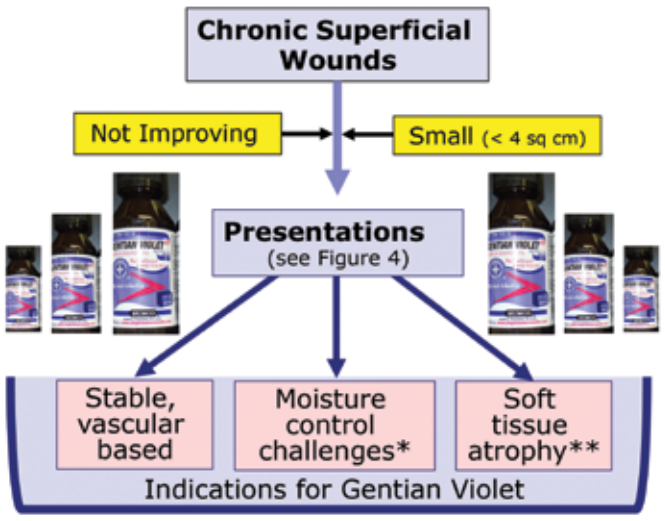
GV is not the answer for every wound, but there are reports of its use in a variety of wound types.[6-8] We have not used it in wounds with bases wider than 4 cm in diameter. Although we are unaware of toxicity when GV is used locally on wound bases, we feel other techniques, especially surgical interventions such as debridements, deformity correction, and grafting are needed for larger wounds. GV was not found to be effective in radiotherapy-induced moist skin desquamation.[9] We also observed lack of effectiveness in chronic, refractory venous stasis ulcers in which underlying cicatrix needed to be excised before successful skin coverage could be achieved. In addition, we do not advocate using GV over exposed bone or tendon. We use an algorithm approach for making decisions about using GV for our patients with chronic wounds (*Figure 5*).

FIGURE 4. Gentian violet bottles and methods of application

Legend: GV is available in one and two ounce bottles; note the Spanish labels in the one-ounce bottles.

The cotton tip of a cotton-tipped applicator is wetted with GV and then "painted" into the open wound—almost like being an artist. For punctate wounds, the wooden end of the applicator is used for coating the wound base with GV.

FIGURE 5. Our algorithm for using gentian violet



Legend: When the above criteria are met, we recommend using GV for the three subsets of chronic stable wounds.

Notes: *Usually used in conjunction with zinc oxide as a moisture barrier and to prevent maceration of the skin.
**Typically wound bases rest on bone and is frequently observed in patients with hidebound skin, vasculitis/collagen vascular disease and/or aging.

A minor complaint expressed by some patients using GV is that it "stains everything." Almost anything GV comes in contact with is stained a dark blue, including dressing coverings, skin, and clothes. That is advantageous as well as problematic. We have observed that the GV stains the skin adjacent to the wound and persists until the stained skin desquamates. However, the stain does not persist on granulation tissue. Consequently, with each dressing change, removal of the gauze covering is in effect achieving an autolytic debridement of the wound base. The skin-staining problem is mitigated by educating the care provider to apply a light coat of GV only over the wound base with a cotton-tipped applicator (*Figure 4*). With this technique, a 30 cc bottle of GV can be used for multiple applications. When GV is applied lightly, a single gauze layer is usually sufficient for preventing the dye from staining clothing. The donning of gloves, while not required, will prevent care providers from getting GV stains on their fingers.

We try to instill in the care provider — whether it is the patient, a family member, or a nursing service applying the GV — to think in terms of being an artist. The cotton-tipped applicator (CTA) is the brush, and the wound base is the easel. In addition, because of GV's staining spreadability, only the distal tip of the cotton needs to be moistened with GV. When the wound is very small, we advise using the wooden end of the CTA to precisely apply the GV to the wound base. If zinc oxide is used around the periphery of the wound, it too can be applied with a CTA. This artist analogy almost makes the GV application a "fun" activity for the care provider.

Since the stain persists on tissues, we question how frequent the GV applications need to be done. While we presently recommend applying GV once a day to the wound base, preliminary observations with every other day and even weekly applications seem to show its effectiveness. However, if moisture control for the wound base is needed, daily applications are advised to prevent moisture accumulation in the dressing from irritating the wound and surrounding tissues.

GV has been incorporated in foam dressings and in combinations with methylene blue with positive reports of its effectiveness.[9-12] We have had experiences with Hydrofera Blue® but have not used it for the indications we use for selecting GV (*Figure 5*).

The use of GV for wounds lends itself to randomized control trials (RCTs). Our decision to use GV was based on observations that healing was stalled with other agents. Our observations, while supportive of the effectiveness of GV, could be substantiated with RCTs that assess each of the three permutations (underlying deformity, moisture control, and ischemic-hypoxic wound types) described in the observations/results section above. Not only could its effectiveness be confirmed, but studies could show its cost-benefits. If the decision is made to "live" with a chronic-

stable wound, it is unlikely any other wound dressing agent could be less expensive.

Conclusions

GV had been used as a "last resort" intervention in many of our patients' wounds that had been refractory to other interventions. The clinical improvements we have observed using GV now justify using this agent on superficial and mildly cavitary wounds at earlier and earlier stages in the patients' wound courses. The ease of application and the economies of GV have made this agent an increasingly important intervention in our armamentarium of wound care products. ■

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About the Author

DR. MICHAEL STRAUSS attended Stanford University, where he majored in biology, and Oregon Health and Sciences University in Portland, Oregon, where he graduated from medical school in 1965. He completed his internship at Los Angeles County/University of Southern California Medical Center in Los Angeles followed by a one-year general surgery residency at Mount Sinai Hospital in New York City before entering the U.S. Navy. He completed his orthopedic surgery residency at the Navy Medical Center in San Diego.

In 1977, he joined the staff at Long Beach Memorial Medical Center (LBMMC) in Long Beach, California, and served as associate director of the hyperbaric medicine department until 1992, when he became medical director. He helped establish the hospital's Wound Healing Center and significantly contributed to its distinction along with hyperbaric medicine as a LBMMC Center of Excellence.

Dr. Strauss is a clinical professor of orthopedic surgery at the University of California Irvine as well as the orthopedic consultant for the PAVE (Preservation-Amputation Veterans Everywhere) Clinic at the Veterans Affairs Health Care Medical Center in Long Beach, California. He serves on the editorial board and the Hyperbaric Oxygen Therapy Committee of the Undersea and Hyperbaric Medical Society.

Classroom or Online CME Courses?

Which Is More Effective?

By WCHM Editors

The ultimate outcome of continuing medical education (CME), whether in person or online, is increased knowledge of current skills and practices, which should translate to better patient outcomes through the application of the information learned. But which CME activities are most effective for achieving these goals?

We asked two CME accreditation experts, the Undersea and Hyperbaric Medical Society (UHMS) and the Wilderness Medical Society (WMS), for their insight on whether to go live or online for CME credits. Their feedback is below.

Company name:
Undersea and Hyperbaric Medical Society

Types of CME options your company provides (live, online, or both):
Live, online, journal

Types of credits your company provides:
Physician CME: AMA PRA Category 1 Credit(s)™, Nursing/RRT contact hours, NBDHMT Category A/Category B

1. In your opinion, what general components make a CME course effective?

I believe an effective course is one in which the objectives are met and the participants have gained some form of knowledge, competence, and/or performance that will change the way he/she practices that will have an overall impact on better patient outcomes.

2. In your opinion, what are the benefits of traditional, live, classroom-based CME?

The live classroom allows a face-to-face relationship with the faculty and peers who share a common interest for various reasons. Almost as important in a live classroom setting are the breaks outside of the classroom where you can network with other professionals and potentially gain some information on other specialties, devices, or products and make lasting relationships with other professionals. Live didactic lecture allows the participant to have a direct relationship with the faculty member and to be able to ask any questions or make comments for immediate discussion or response.

3. In your opinion, what are the benefits of taking a CME module online?

Online CME modules allows participants to save costs of travel, lodging, meals, and, most importantly, time away from their practices. Many participants pay for all of their expenses out of pocket. Online CME modules allow them to work at their own pace, in their own environment, and provides a cost-effective platform to do so. It also allows them to be selective in choosing the most appropriate educational activity to meet their needs and learning style rather than attending a program because it fits their schedule. Online CME modules are practical in today's fast-paced world, and we are almost always connected to access online education while waiting for a flight, in between meetings, or on a lunch break.

4. In your opinion, which type of CME better translates to an increase in better patient outcomes and physician skill and knowledge?

I believe it is not so much the "type of CME" that will increase better patient outcomes and physician skill and knowledge, but, more importantly, it's the content of the CME course. Do you have expert faculty who are top-notch in the field? Does the faculty use current references and resources since the medical field is ever-changing and procedures/devices can become out-of-date quickly? Is the content relevant to the participant's practice? Are the faculty relatable and able keep the

participant's attention? Specifically to the type of CME, every participant learns differently. Many learners are visual learners, so a hands-on session may benefit them the most. Others may not focus well for a full day of live didactic lecture, so an online platform may allow them to learn at their own pace. I feel the most effective way to improve patient outcomes and physician skill and knowledge is to include as many different platforms as possible within reason to tailor to all or most of the participants' learning needs.

5. In your opinion, can CME effectiveness be comparatively measured? How is this determined?

Yes, CME effectiveness can be comparatively measured. Depending on what you want to measure and to what degree you want it measured, there are different methods. Some may use an audience-response system that measures the impact of the activity in real time. This may allow the faculty to alter their presentation if the participants are not picking up on certain topics or did not spend enough time with a certain area, reflecting from the responses. The UHMS asks participants questions at the completion of the course specifically to determine how the educational activity had an impact on them, if and how it will change the way he/she practices, what barriers are present that might hinder change, if the activity met his/her needs, what changes he/she would like make to the program in planning future activities, etc.

* * * * *

Company name:

Wilderness Medical Society

Types of CME options your company provides (live, online, or both):

Both, including video and article review

Types of credits your company provides:

ACCME approved only

1. In your opinion, what general components make a CME course effective?

The planning process, gap analysis, up-to-date content, and good evaluation process are the most important aspects of an effective CMS course.

2. In your opinion, what are the benefits of traditional, live, classroom-based CME?

Benefits include the learner and teacher interaction — the ability to ask questions and have them answered in real time. A second benefit is hands-on skill development and testing.

3. In your opinion, what are the benefits of taking a CME module online?

You can obtain more in-depth material over several sessions with a CME module online.

4. In your opinion, which type of CME better translates to an increase in better patient outcomes and physician skill and knowledge?

This question is impossible to answer well. It has to be a mix of skills and knowledge base.

5. In your opinion, can CME effectiveness be comparatively measured? How is this determined?

I'm not sure this is possible. The variables that would need to be controlled are staggering. The testing procedures would be hard to develop. Standardized testing for all age groups has not worked well as far as predicting high education success. Skills, on the other hand, are better adapted to comparative testing.

* * * * *

Whatever your CME preference is, we hope this article has helped provide insight and perspective on the benefits of online CME versus live courses. When deciding which option is best for you and/or your clinic staff, you may want to take in to consideration lost revenues caused by time away from the office (in the case of live courses) and travel expenses. In contrast, there are considerable benefits to taking advantage of the networking opportunities that come only with attending a live event. We invite you to share your thoughts and experiences on the issue with our community of wound care and hyperbaric professionals on our LinkedIn and Facebook pages or email us at *info@bestpub.com*. ■

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Development of a Wound Care and Hyperbaric Medicine Center

By Rudy C. Pruneda

Excerpted from Chapter 4: Development of a Wound Care and Hyperbaric Medicine Center in Hyperbaric Facility Safety: A Practical Guide *by Rudy C. Pruneda. It is reprinted with permission of the publisher, Best Publishing Company.*

CENTER ORGANIZATION

The need for a center that specializes in healing diabetic, venous stasis, and other chronic wounds is self-evident. In addition, many physicians and podiatrists do not possess all the tools or protocols that can successfully heal recalcitrant ulcers. The challenge of chronic ulcers is complex and involves multifactorial problems. A complete assessment by physicians and nurses, laboratories, and other diagnostic tests is required to confirm clinical judgment, and the treatment plan may call for surgical intervention, nutritional assistance, off-loading of affected limbs, compression therapy, antibiotics, etc. The center should contain the appropriate diagnostic and therapeutic equipment to perform the majority of wound care inside the premises, thus allowing patients to confine their visits to one area.

Role of Wound Care Specialists

This type of treatment is best undertaken in a center that incorporates various physician specialties (orthopedic, plastic, and vascular surgeons; infectious disease specialists; podiatrists; endocrinologists) and nurses and technicians trained in wound care and hyperbaric oxygen. Treatment protocols are designed with the understanding that a chronic wound should be converted to an acute wound via a surgical debridement, when appropriate. If infected, the wound should also be treated with antibiotics until no visible signs of infection are present. The wound environment is kept moist to facilitate fibroblast formation; therefore wound dressings are applied that allow the proper environment to exist. Edema is removed from wounds with compression wraps, stockings, and pumps. Protocols are agreed upon by the wound care team, and forms are designed to record visit activities, wound measurements, and any other diagnostic or therapeutic activities. In certain cases, hyperbaric oxygen therapy is recommended.

Role of Hyperbaric Oxygen in Wound Healing and Infection Control

Studies by Pai and Hunt[6] revealed oxygen plays a critical role in wound healing. Their studies revealed that increasing the oxygen levels improved the healing of wounds. Knighton[7] showed that oxygen acts as an antibiotic, which allows white blood cells to form oxygen radicals that destroy bacterial cell walls. Other researchers have been able to show that oxygen and certain antibiotics act synergistically to destroy bacteria.[8,9] Sheffield[10] demonstrated that wounds heal better when hyperbaric oxygen is used.

Identifying Team Members

Most communities will have certain physicians and podiatrists who are known as the "wound care experts," and their fellow doctors will usually refer difficult-to-heal wounds to these individuals. These doctors will usually welcome the opportunity to join a center that is organized to support his/her efforts. Reimbursement for supplies is usually negligible in a physician's office, plus the doctor has to pay staff to support his medical and billing functions. The wound center can make staff and supplies available to the doctor's patients. As an outpatient center of the hospital, the supplies are covered, and the staff belongs to the contractor of the wound care service or the hospital; therefore the doctor is spared these expenses. In addition, the wound care center provides forms that allow the doctor to keep track of his professional activities and billing.

It is important to establish a multidisciplinary team of doctors, which can support wound healing activities. Initially, infected wounds contain necrotic tissue or wound fluids that inhibit healing and must be removed.[13] Availability of a general surgeon who understands the importance of debridement to fresh, bleeding tissue or cancellous bone is optimal. In other cases, revascularization may be needed to support blood flow to the lower leg. A vascular surgeon is ideal in these instances. Other times, a large shallow wound may be better covered with a muscle flap or skin graft, thus a plastic surgeon is needed.

In diabetics, it is vital that blood sugars are kept under control to prevent the harmful effects of compromised white blood cells[14] and formation of deposits on blood vessels that constrict blood flow.[15] Internal medicine, endocrinology, and diabetic education nursing specialties can help develop treatment plans that include medication and diet modification. Diabetics also suffer from infections more severely than normal patients.[16] Many times, a simple cut may lead to gangrene or osteomyelitis. Infectious disease specialists can often help guide the administration of IV antibiotics and subsequent oral antibiotics.

Nurses with specialty training in wound care are often difficult to recruit. Enterostomal nurses have the background and experience in wounds; unfortunately these nurses are usually only found working in large or teaching university hospitals. As a general rule, inexperienced RNs and LVNs can be trained at hyperbaric medicine courses recognized by the Undersea and Hyperbaric Medical Society (UHMS). More information can be obtained online at *www.UHMS.org*. Certain basic wound care courses provide excellent information on the physiology of wounds, treatment protocols, and current innovations in wound supplies.

Trained hyperbaric technicians are also very difficult to recruit, so individuals with EMT backgrounds or previous military hyperbaric training can be hired and trained in-house. These individuals help with wound care, monitor the hyperbaric treatments, and perform maintenance on the hyperbaric chambers.

Location of Wound Center

Ideally, the wound center should be located on the hospital premises. This allows better coordination of patient transportation, diagnostic testing, dietician support, and services such as physical therapy, surgery, and laboratory support. Access to the hospital parking lot is important, especially with wheelchair access.

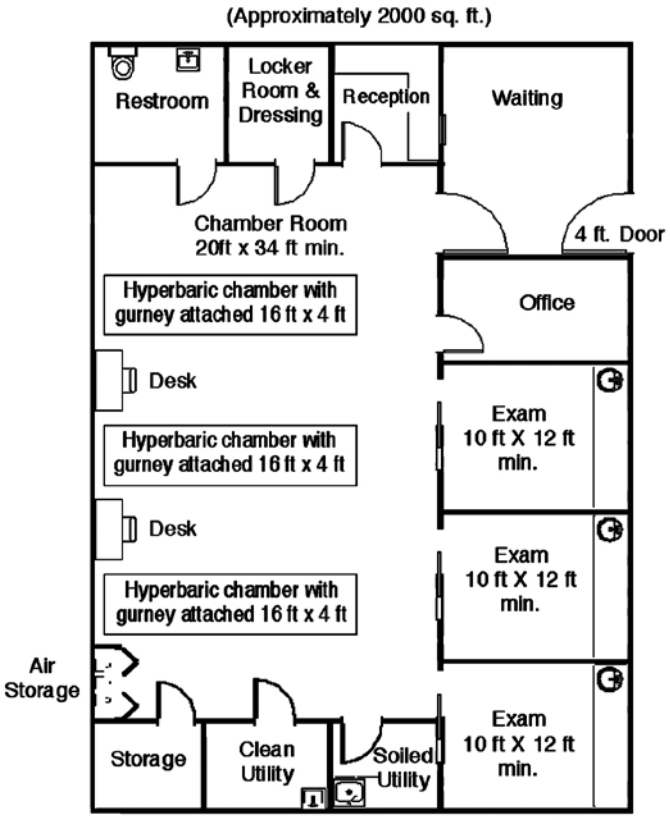
Many times hospital space is at a premium or occupied with other services, and the wound center must be located in a professional building adjacent to the hospital. In these cases, transportation becomes an important consideration. Covered hallways to the hospital should be considered to help minimize exposure of patients to inclement weather. Wheelchair and stretcher access, with doors having a minimum space clearance, must be considered when developing the program.

LOGISTICAL CONSIDERATIONS

Flow of Patients in the Center

Consideration should be given to establishing a traffic flow that does not result in bottlenecks throughout the day. There will usually be three types of patients in the center: new patients for evaluation, patients for wound care only, and patients for wound care and hyperbaric oxygen therapy. Space should be established so that patient flow is in one direction — from entrance to exit, thus minimizing patients crowding hallways and/or waiting for evaluation or wound care procedures. Figure 1 gives an example of a wound center that allows good patient flow.

FIGURE 1. Wound center with good patient flow



Room Design

Each state may have requirements that must be followed when building a wound care/hyperbaric medicine center. For example, in Texas certain requirements are outlined for wound centers that have hyperbaric facilities.[17] The following are considered the minimum requirements for a hyperbaric suite:

Patient waiting area: The area should be out of traffic flow, under staff control, and contain enough seating capacity for patients and their relatives throughout the day. When the waiting area is for both inpatients and outpatients at the same time, separate areas shall be provided with privacy between both areas. Patient waiting areas are not required for two or fewer individual hyperbaric chambers.

Control desk and reception area: A control desk and reception area shall be provided to greet patients, fill

out registration information, and serve as traffic control center throughout the day.

Holding area: This area should accommodate inpatients on stretchers or beds and be out of the traffic flow. This area may be omitted for two or fewer hyperbaric chamber facilities.

Patient toilet rooms: Toilet rooms shall be provided with hand-washing fixtures, which have hands-free operable controls with direct access from the hyperbaric suite.

Patient dressing rooms: Dressing rooms for outpatients should include a seat or bench, mirror, and provisions for hanging patients' clothing and for securing valuables. At least one patient dressing room should accommodate wheelchair patients.

Staff facilities: Toilets with hand-washing fixtures with hands-free operable controls may be outside the suite but convenient for staff use.

Consultation room: An appropriate room for private consultations with wound care physicians shall be provided for outpatients.

Storage space: A clean storage space shall be provided for clean supplies and linens. Hand-washing fixtures shall be provided with hands-free operable controls. When a separate storage room is provided, it may be shared with another department.

Soiled holding room: A soiled holding room shall be provided with waste receptacles and soiled linen receptacles.

Hand washing: A lavatory equipped for hand washing with hands-free operable controls shall be located in the room where the hyperbaric chambers are located.

Housekeeping room: The housekeeping room shall contain a floor receptor or service sink, storage space for housekeeping supplies and equipment, and be nearby.

The hyperbaric area should be large enough to allow two to four monoplace chambers or one multiplace chamber, plus room for stretchers and a console area where a technician or nurse can monitor the treatments and communicate with patients and/or staff inside the chamber. Space between monoplace chambers is as follows: chamber and sidewall, five feet; between chambers, six feet; and between the chamber headboard and wall, three feet. A minimum passage space of four feet shall exist at the head of the chamber. Typically, a monoplace operation may require between 2,000-2,500 sq. ft. for all operations, while a multiplace chamber operation may require 3,000-4,000 sq. ft.

Timetable for Establishing a Wound Center

In general, the timetable for opening a wound center with monoplace chambers may vary from 90 days to 180 days, depending on approval by state agencies, architect plans, administration approvals, etc. A multiplace chamber installation may take up to one year before patients can be treated. The following steps must be accomplished to help expedite final construction and patient treatments:

- Construction
- Architectural drawings completed
- Approval by the state agency to begin construction
- Approval of a contractor to begin construction
- Completion of construction and approval by local fire marshals for occupancy
- Final inspection by state agency to begin patient treatments

"The hyperbaric area should be large enough to allow two to four monoplace chambers or one multiplace chamber, plus room for stretchers and a console area where a technician or nurse can monitor the treatments and communicate with patients and/or staff inside the chamber."

Hyperbaric Chambers

Ordering chamber(s): Normally chamber manufacturers need six to eight weeks for construction once the order is placed. Schedule chambers to arrive approximately the same time as final state inspection. A multiplace chamber may require six months to a year for construction. Many times, construction is done around a multiplace chamber, so coordination of this facility is much more involved.

Connect chambers and perform safety checks to ensure chambers are operational. Conduct training of medical personnel to ensure safe chamber operation.

Equipment

- Wound care chairs
- Wound care lamps
- Transcutaneous oxygen monitors
- Surgical supplies for debridement procedures

- Digital camera for wound photos
- Computer to record wound care activities and schedule patients

Supplies

- Curlex rolls
- Saline
- Specialty dressings
- Gels
- Tape
- Gauze
- Scissors
- Compression wraps

Marketing

Once the decision is made to run a wound program, education of the hospital staff should begin. Nurses on the floors may provide good referrals on wound care patients. Educational efforts may be in the form of literature on wound care, newsletters, videos, or scheduling wound care specialists to give talks on the subject. If continuing medical education (CME) units are included, physicians and nurses from outside the hospital will be encouraged to attend. Home health agencies are a good target group, since they care for the majority of patients outside the hospital.

The general public should also be educated and made aware of this service in the community. The most common forms of advertisement include television, radio, and print ads. These ads may be aimed at certain populations, i.e., diabetics or patients with venous stasis disease. Scheduling of ads should be aimed at starting immediately before the wound center is ready to treat patients and should run until the wound center is well established.

Summary

Establishment of a successful wound care/hyperbaric medicine is based on several factors: (1) a patient population that needs the service; (2) a team of physicians, nurses, and technicians committed to the cause of wound healing; and (3) a hospital that is willing to commit the resources necessary to provide the service to the community and surrounding areas. Requirements for construction of the wound center/hyperbaric medicine facility will vary depending on local, county, state, and NFPA 99 requirements. Marketing the program will require identifying the medical community to educate as well as the public, which ultimately benefits from the service in community. The wound center can certainly impact the quality of life for patients who have previously found these wounds debilitating and costly to the health care system. ■

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