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WOUND CARE AND HYPERBARIC MEDICINE

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

MAGAZINE PRODUCTION
Lorraine Fico-White
lorraine@bestpub.com
Diana Robinson
diana@bestpub.com

ADVERTISING/SPONSORSHIPS
info@bestpub.com

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In This Issue…

In this issue of *WCHM* we continue to invite leading experts in the fields of hyperbaric medicine, diving, and wound care to share their valuable and insightful expertise.

Our Clinic in Focus section features the Edward Hospital Wound Healing and Hyperbaric Center located in Naperville, Illinois. If you are a part of an exceptional hyperbaric or wound care center, contact us to be our next featured clinic.

Dr. Doug Ebersole contributes two articles in our Dive Medicine section. His first article addresses coronary artery disease with diving. With more than 3 million certified divers in the United States (and an increasing number being middle-aged or older), the subject matter is timely.

Continuing to keep in mind the health and safety of the scuba diver, Dr. Ebersole, in his second article, discusses the relationship of the patent foramen ovale (PFO) to decompression sickness (DCS) and provides an understanding of the link between PFO and DCS and various treatment options.

We welcome back Dr. Heather Hettrick with Part 2 of her Wound Geography and Tissue Types series as her journey continues. The video course is also available for 1.0 nursing CE credits. Find all the details and links in the article.

We also welcome back hyperbaric safety director Darren Mazza as he discusses basic training processes for the entire staff in a hyperbaric safety facility.

Join us in delivering the highest-quality publication in the industry as we focus on advancing the knowledge and practice of wound care, diving, and hyperbaric medicine. Share *WCHM* magazine with colleagues and clients. Add your clinic to our Map of Wound Care and HBO Centers (*www.bestpub.com*).

Please send us your comments, articles, industry information, press releases, and updates. We look forward to hearing from you!

Lorraine Fico-White
Managing Editor, *WCHM* Magazine

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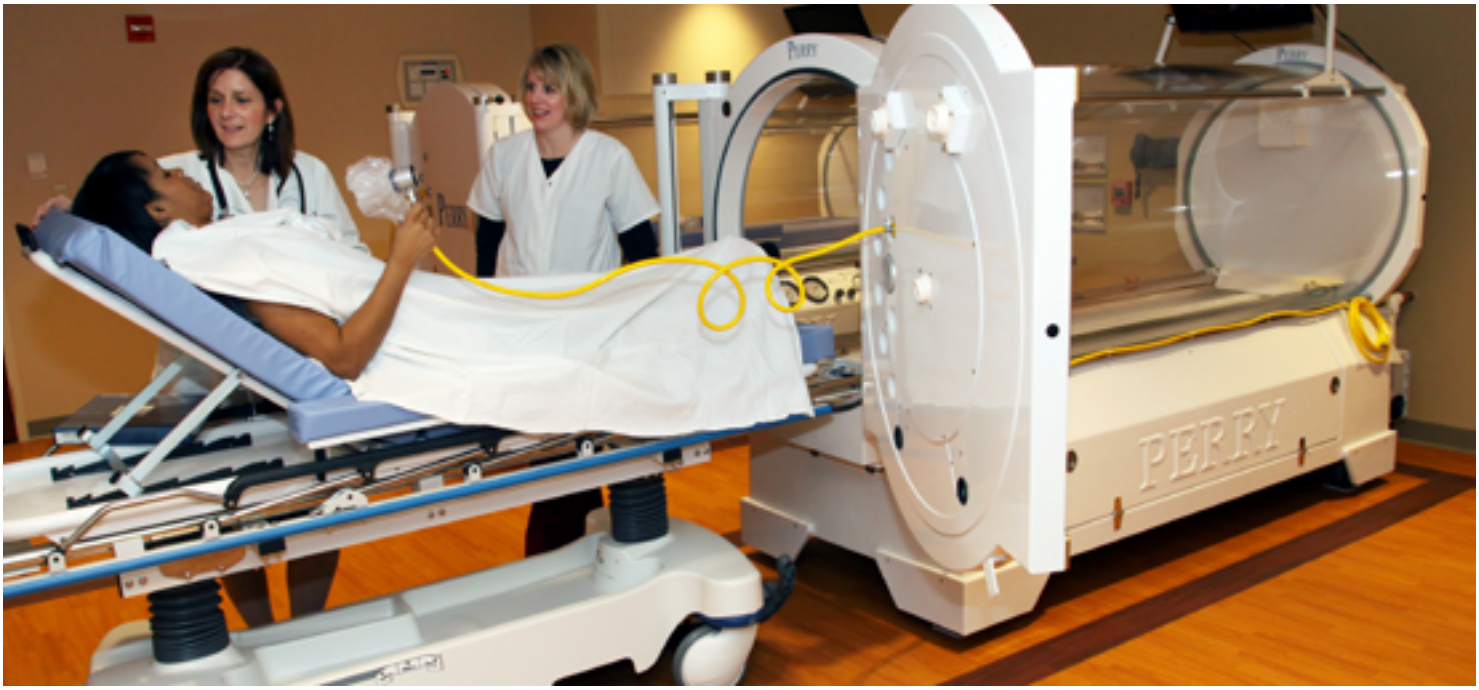
WOUND CARE AND HYPERBARIC MEDICINE



CLINIC IN FOCUS

Edward Hospital Wound Healing and Hyperbaric Center

Continuing our series of interviews featuring outstanding hyperbaric and wound care centers around the world, we spotlight in this issue the Edward Hospital Wound Healing and Hyperbaric Center at Edward Hospital in Naperville, Illinois.

How has seeking UHMS accreditation affected your clinic?

It has demonstrated our passion to provide the highest quality care and outcomes. It united the clinic to work for a common goal and to continue to treat our patients with best-practice medicine. Having hyperbaric oxygen therapy in the wound clinic itself allows all the staff to coordinate care for the patients. It provides streamlined and efficient care.

What are the most common indications treated at your clinic?

Wounds and ulcerations of various etiologies are treated at the Edward Hospital Wound Healing and Hyperbaric Center. Wound care indications such as diabetic foot ulcers, venous leg ulcers, peripheral arterial ulcers, and pressure ulcers are the main four, with diabetic foot ulcers as the most common. Evidenced-based practice guidelines are utilized to

CLINIC DETAILS

Clinic Name: Edward Hospital Wound Healing and Hyperbaric Center

Location: Edward Hospital, Naperville, Illinois

Website: www.edward.org

Phone: 630-527-3002

How long in business: 8 years

How many chambers: 3

Chamber types: Perry Sigma 36 and Perry Sigma 40

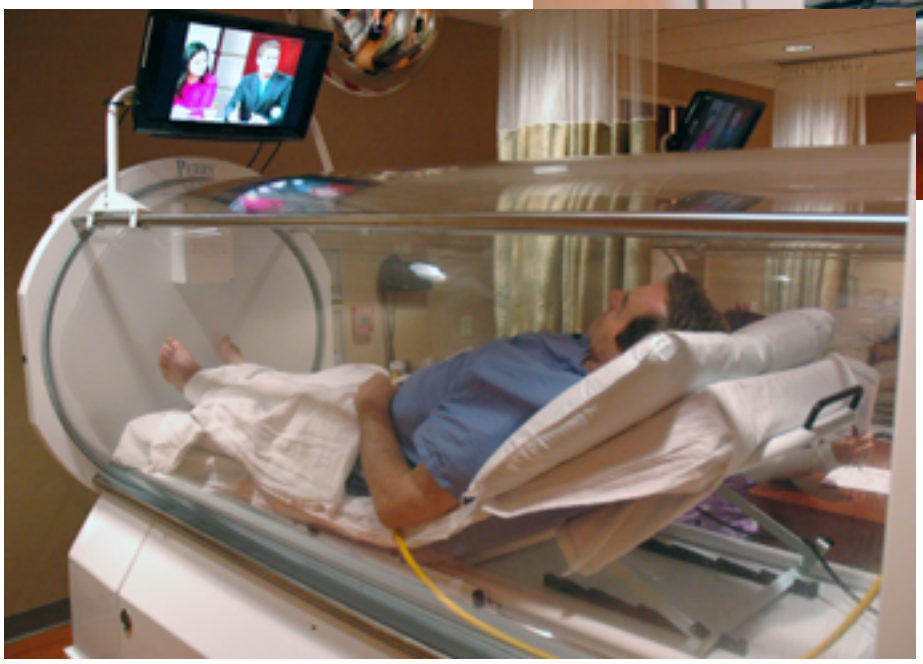
How many physicians/nurses/CHTs: 5 physicians, 2 APNs, 5 RNs, 4 CHTs

Medical director: Dr. Najjar (wound care), Dr. Villanueva (hyperbaric)

Date of UHMS accreditation: October 2014

develop comprehensive treatment plans to assist with the healing of the patient.

The most common indications treated in the hyperbaric oxygen (HBO) therapy program include diabetic foot ulcers, compromised flap/graft, osteomyelitis, and soft tissue radio necrosis. After appropriate patients go through a screening process and it has been determined that HBO therapy may assist with healing, treatment is started in conjunction with standard wound care. Treatments consist of two hours a day, five days a week, for an average of thirty treatments.



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

A 40-year-old female with a history of neurofibromatosis injured her left lateral leg in March 2014 after it was hit by a car door. She was treated for a staph and fungal infection. In August 2014, the wound was surgically debrided and started on negative pressure wound therapy. Due to complications of infection and comorbidities, the wound deteriorated, and the threat of loss of limb was pending. With collaboration of a vascular surgeon and infectious disease and wound care team, the patient was followed very diligently. She was seen in the wound care center three times a week for close visualization of wound. Infection was managed, and she improved to the point of accepting a biosynthetic skin graft. Currently she is 99% healed and should have total resolution of the wound within the next few visits.

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

Collaboration between all the staff is the main reason for our success. Everyone works together on the patients' care. Physicians, RNs, APNs, medical assistants, and hyperbaric techs all give their input. Everyone's treatment is individualized based on best-practice guidelines

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

Communicating with the community's physicians is an important marketing tool. Wound care and hyperbaric medicine are specialized services to help patients heal. Reports are sent to primary physicians to update them on their patient's progress, and referrals to other specialties (diabetes education, physical therapy, weight-loss clinics) can be coordinated. Also, it is helpful to communicate the benefits of hyperbaric medicine to oral surgeons and cancer physicians who may not be aware of some treatment options that hyperbaric medicine can provide.

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

All of our hyperbaric techs are CHT. We also have two nurses who are CHRN and four nurses who are certified in wound and ostomy. All the staff has taken a forty-hour hyperbaric training course — even the director of the program has taken it. It shows how invested the staff and hospital are to providing the best care and outcomes for their patients. ■

Scuba Diving and Coronary Artery Disease

By Douglas Ebersole, MD, FACC, FACP

It is estimated that there are about 3 million certified scuba divers in the United States. A large number of these individuals are middle-aged or older and at risk for coronary artery disease. Cardiovascular disease is the third most common cause of death while diving and remains the principal cause of death in the general population. The development of symptoms of angina, pulmonary edema, or sudden cardiac death underwater carries with it a much higher mortality than would the same event on land. This article will review the workloads related to scuba diving, ways to assess risk in those with or at risk of developing coronary artery disease, and make recommendations to make scuba diving safer.

Scope of the Problem

In 2008, Dr. Petar Denoble published a paper showing the annual death rate for scuba divers was 16.4 per 100,000 persons.[1] This was similar to the rate of 13 jogger deaths per 100,000 participants each year[2] or the risk of driving where motor-vehicle accidents result in 16 deaths per 100,000 persons per year.[3] Thus, while the likelihood of dying when scuba diving is quite small, understanding how and why these deaths occur is imperative.

Unfortunately, the ultimate cause of death while scuba diving is drowning. This does not give us great insight into what led to the drowning. Denoble reported on the causative process of 947 fatalities in an attempt to better define scuba diving fatalities.[4] He divided this into sequential components: trigger, disabling agent, disabling injury, and cause of death. Cardiac events constituted 26% of disabling injuries, and these events were frequently associated with a history of cardiovascular disease and age greater than 40 years. Thus, it looks like underlying cardiovascular disease is a major component in scuba diving deaths.

Workloads Associated with Scuba Diving

It is clear exercise itself is a cardiovascular stress, and the majority of nontraumatic deaths during exercise are cardiac in origin. In most situations, diving is not particularly physically stressful. However, there are times due to current, waves, wind, and other environmental stressors that demands during diving can reach 20 ml/kg/min (6-7 METS). Exercise capacity is reported in terms of estimated metabolic equivalents of task (METs). The MET unit reflects the resting volume of oxygen consumption per minute (VO_2) for a 70-kg, 40-year-old man, with 1 MET equivalent to 3.5 mL/min/kg of body weight.

In the standard Bruce protocol, the starting point (i.e., Stage 1) is 1.7 mph at a 10% grade (5 METs). Stage 2 is 2.5 mph at a 12% grade (7 METs). Stage 3 is 3.4 mph at a 14% grade (9 METs), and Stage 4 is 4.2 mph at 16% grade (12 METs). This protocol includes three-minute periods to allow achievement of a steady state before workload is increased.

Thus, a diver with a steady-state exercise capacity of 6-7 METS can expect to manage most diving contingencies without concern for cardiovascular complications. In most occupational exposures requiring increased physical activity, guidelines recommend maintaining workloads below 50% of maximal oxygen consumption. Based on this relationship, a diver who is expected to minimize safety concerns related to environmental contingencies should have a maximum oxygen consumption of 12-13 METS or about 12 minutes on a standard Bruce protocol exercise test. Divers with peak exercise capacity below that level could expect to dive safely in low-stress conditions such as warm water, minimal currents, and calm seas but could develop cardiovascular limitations under stressful diving conditions.

Who Is at Risk?

For divers older than 35 years, the dominant risk for sudden death is from coronary artery disease. Although the incidence of coronary artery disease death is falling, the rising incidence with age makes this diagnosis the most important consideration when clearing divers who are middle-aged or above.

One strategy to lower the risk of cardiovascular deaths would be to screen all adult participants prior to certification, as most exercise-related cardiac events in adults are due to atherosclerotic cardiovascular disease.

The author diving in West Palm Beach, Florida. (Courtesy of Jim Abernethy)

The Framingham Risk Score is one of a number of scoring systems used to determine an individual's chances of developing cardiovascular disease. A number of these scoring systems are available online.[5,6] Cardiovascular risk scoring systems give an estimate of the probability that a person will develop cardiovascular disease within a specified amount of time, usually ten to thirty years. Because they give an indication of the risk of developing cardiovascular disease, they also indicate who is most likely to benefit from prevention. For this reason, cardiovascular risk scores are used to determine who should be offered preventive medications such as drugs to lower blood pressure and drugs to lower cholesterol levels.

The population risk for divers could be predicted by using tools such as the Framingham Risk Score, and potential participants with a specific score could be identified and excluded. The problem with this approach is that atherosclerotic cardiovascular disease is prevalent among lower-risk subjects. Also, extremely high-risk subjects are only a small part of the total population. Consequently, the largest absolute number of acute events occurs not in the highest-risk subjects but in the moderate- and lower-risk groups. Excluding the highest-risk group likely has little effect on the total number of deaths.

A Framingham Risk Score lower than 10% (less than 1% per year risk) is considered a low score. If a subject is assessed to be at low risk in general, that individual is not likely to have an acute coronary event while diving. On the other hand, high-risk individuals (Framingham score greater than 20%) could be at considerable risk and should have further evaluation to evaluate whether diving will be safe. Intermediate-risk individuals with a Framingham score between 10% and 20% should have further risk stratification to assess their risk for an acute coronary event while diving.

In all individuals, regardless of risk, we should practice primary prevention of coronary artery disease. The recommended performance measures for primary prevention are:

1. Lifestyle/risk-factor screening
2. Dietary-intake counseling
3. Diabetes screening and management
4. Physical-activity counseling
5. Smoking/tobacco cessation
6. Weight management
7. Blood-pressure control
8. Blood-lipid measurement and control
9. Global risk estimation with tools such as Framingham Risk Score
10. Aspirin use in selected individuals

Implementation of these measures requires performance of a careful history and physical examination, laboratory testing for lipids, and formal assessment of cardiovascular risk.

Performing stress testing in selected individuals, such as those with intermediate- or high-risk Framingham scores, is also an approach. In comparison to younger individuals, far less attention has been paid to designing screening programs for older, usually recreational, athletes. Few detailed preparticipation guidelines exist, and there is little reported experience in this age group. Instead, most authorities focus on strategies used in clinical medicine for the early detection of atherosclerotic diseases, as these are the most common cause of death in this age group.

Many divers have returned to diving after either coronary-artery bypass surgery or stenting. Success in return to diving is based on restored exercise capacity without ischemia after revascularization and choosing diving environments that do not produce excess stress on the cardiovascular system.

Since most individuals are asymptomatic, the history is often more helpful in identifying risk factors rather than symptoms. Similarly, there may be few detectable abnormalities at rest or even with exercise, as events are often due to spontaneous rupture of nonobstructive plaque.

The American Heart Association issued recommendations for preparticipation screens in older athletes in 2007.[7] This document recommends that older competitive athletes (over 35 to 40 years old) be "knowledgeable" regarding their personal history of coronary artery disease risk factors and family history of premature coronary artery disease. Further, stress testing should be performed selectively for individuals engaging in vigorous training and competitive sports and who meet the following criteria: men over 40 years or women over 55 years with diabetes mellitus, or at least two risk factors or one severe risk factor other than age. Finally, the document recommends education regarding prodromal cardiac symptoms, such as exertional chest pain.

What about Patients with Established Coronary Artery Disease?

Patients with known coronary disease often have been subject to revascularization either by coronary-artery bypass surgery or by percutaneous coronary intervention, usually with implantation of one or more coronary artery stents. The degree of revascularization can determine safety in diving. With complete revascularization, low-stress diving can be accomplished successfully, but diving in rough seas, fast currents, or cold water could be risky. Many divers have returned to diving after either coronary-artery bypass surgery or stenting. Success in return to diving is based on restored exercise capacity without ischemia after revascularization and choosing diving environments that do not produce excess stress on the cardiovascular system.

Patients with significant reduction in left ventricular systolic function (LVEF under 35%) are at risk for exacerbation of congestive heart failure while diving. Water immersion itself results in approximately 700 cc of fluid shift into the central circulation. This could provoke congestive heart failure in patients with impaired left ventricular systolic function. Additionally, most patients with LVEF under 30-35% will have impaired exercise tolerance when diving as outlined above. For these reasons, patients with significant left-ventricular systolic dysfunction should be advised against scuba diving.

Recommendations

1. All adults should be evaluated for their risk of coronary artery disease prior to scuba diving.
2. Selected individuals with intermediate to high-risk Framingham scores should be referred for additional evaluation, such as treadmill testing prior to scuba diving.
3. All individuals should practice primary prevention strategies to decrease their risk for the development of coronary artery disease.
   a. Smoking cessation
   b. Blood-pressure screening and management
   c. Weight control
   d. Physical-activity counseling
   e. Cholesterol screening and management
   f. Diabetes mellitus screening and management
4. Patients with coronary artery disease may begin (or return to) diving as long as they have been re-vascularized with no ischemia on treadmill stress testing, have good exercise tolerance (defined as a maximum exercise capacity of 13 METs or an ability to sustain a workload of 6 METs), and have relatively preserved left ventricular systolic function.

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About the Author

DR. DOUGLAS EBERSOLE is a cardiologist specializing in coronary and structural heart interventions at the Watson Clinic LLP in Lakeland, Florida. He is also an avid technical, cave, and rebreather diver and instructor.

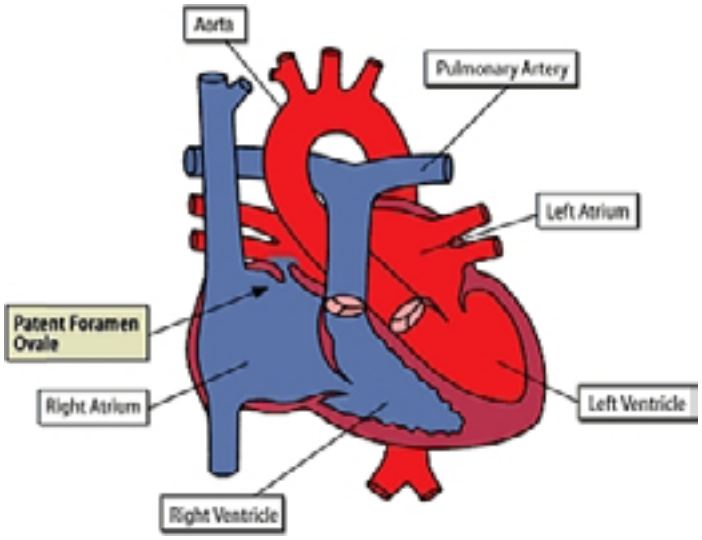
Contact Ebersole at debersole@watsonclinic.com.

Patent Foramen Ovale and Decompression Sickness

By Douglas Ebersole, MD, FACC, FACP

Patent foramen ovale (PFO) is a very popular topic in scuba diving as the appreciation of its relationship to decompression sickness (DCS) becomes more widespread in the diving community. Though the incidence of DCS in recreational diving is only about two episodes per 10,000 dives, DCS affects approximately 1,000 divers per year. The presence of a PFO is felt to increase the risk five- to thirteenfold.[1-3] An understanding of the link between PFO and DCS as well as various treatment options is vitally important to the health professionals who treat these patients.

FIGURE 1. Diagram of fetal circulation through the patent foramen ovale



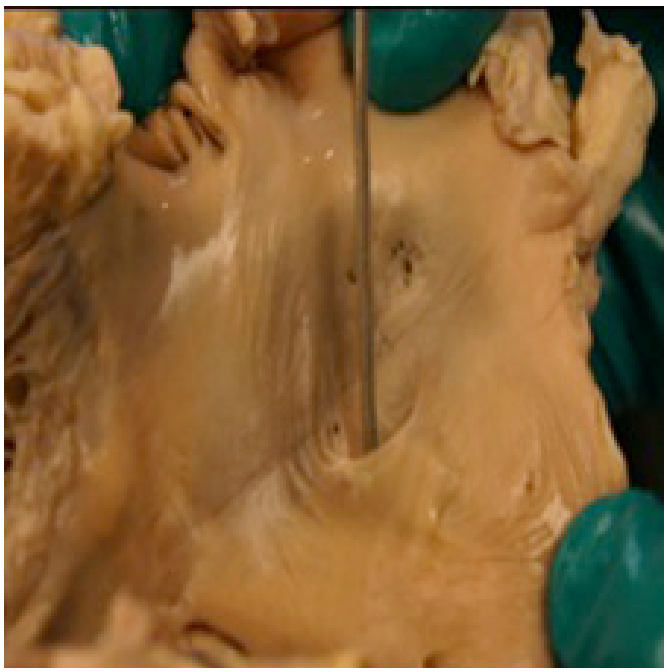
Incidence and Anatomy

The PFO is an integral part of the normal fetal circulation (*Figure 1*). Normally, a portion of the blood from the inferior vena cava passes from the right atrium to the left atrium through the PFO during fetal life, bypassing the lungs. At birth, pulmonary blood flow increases greatly, increasing left atrial pressure. The resulting atrial pressure differences compress the septum primum against the septum secundum, functionally closing the PFO. Anatomic closure of the PFO occurs later in infancy in most people but is incomplete in approximately 25% of the population,[4,5] leaving these individuals at risk for right-to-left shunting (*Figure 2*). PFO diameters are quite variable in size, ranging from 1-19 mm, with the average size being larger in older adults,[4] suggesting PFOs may continue to enlarge during life.

Relationship of PFO to DCS

It was first suggested in 1986 that a cardiac right-to-left shunt may be important for paradoxical gas embolism in scuba divers.[6] Subsequently, the importance of PFO for DCS in divers has been further investigated.[1,7-10] As mentioned earlier, the risk of DCS in sport divers is quite low but is increased by at least fivefold in the presence of a PFO.[1-3] Additionally, the average number of ischemic brain lesions as seen on MRI in experienced divers with PFO has been reported to be twice as high as in divers without PFO.[11] The etiology and clinical significance of these findings are unclear but may represent multiple subclinical paradoxic embolic events across the PFO.

FIGURE 2. Autopsy finding of a patent foramen ovale



Diagnosis

Both transthoracic echo (TTE) and transesophageal echo (TEE) have been used for the diagnosis and assessment of PFO. TEE is the preferred diagnostic test of choice, however, given its better visualization of the atrial septum resulting in greater sensitivity in making the diagnosis.

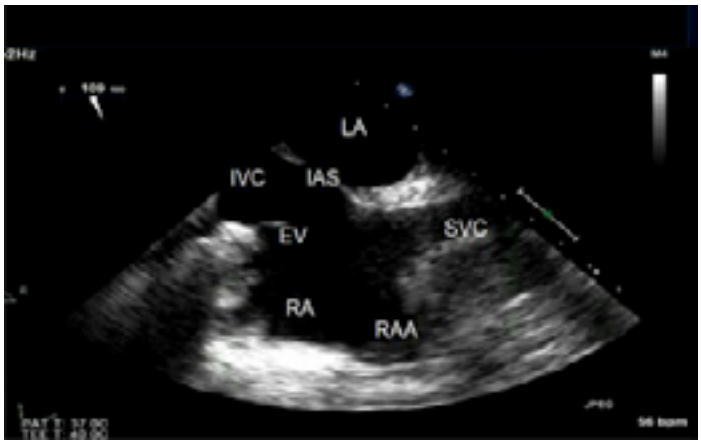
The injection of agitated saline increases the diagnostic sensitivity by enhancing echocardiographic detection of the trivial intermittent right-to-left shunting across a typical PFO. Agitated saline contrast, injected intravenously during Valsalva maneuver with release of straining when contrast is visualized in the right atrium, increases sensitivity. Visualizaton of contrast microbubbles passing from the right to left atrium through the visualized foramen ovale during the release phase is diagnostic of an interatrial communication. In clinical practice, the actual site of right-to-left shunting may not be convincingly visualized or recorded for technical reasons. If a recording convincingly demonstrates microbubbles appearing in the left atrium immediately after arriving in the right atrium, then the presence of a PFO can be presumed. If bubbles appear in the left atrium before or greater than five beats after they appear in the right atrium, then the possibility of anomalous pulmonary arteriovenous connection to the left atrium or pulmonary arteriovenous malformations must be considered.

Contrast injected through an upper-extremity vein may be washed away by contrast-free blood flow from the inferior vena cava directed by the Eustachian valve, creating a false-negative result[12] (*Figure 3*). Injection of contrast via the femoral vein has been proposed to enhance detection by TEE, with the streaming effect of directed inferior vena cava flow to the region of the fossa ovalis and through a patent foramen.[13]

Therapeutic Options

No specific guidelines exist for PFO closure in people who have decompression illness, but the options are to stop scuba diving, decrease the depth and/or time of

FIGURE 3. Bicaval view of a transesophageal echo



LA (left atrium); IAS (interatrial septum); RA (right atrium); RAA (right atrial appendage); EV (Eustachian valve); SVC (superior vena cava); IVC (inferior vena cava)

dives to limit the inert gas load, or undergo percutaneous PFO closure. Some divers decide they have many other interests, and diving is not that important to them. These divers will frequently give up the sport. Other divers who enjoy the sport but dive infrequently often opt for diving "conservatively" to limit their bubble load. This would involve no-decompression diving, limiting depths to less than 100 feet, diving nitrox on air profiles, prolonged (greater than the usually recommended 3- to 5-minute) safety stops at approximately 15 to 20 feet at the end of their dives, and limiting the number of dives per day to 1 or 2.

People who make their living through scuba diving (instructors, divemasters, etc.) and divers who enjoy more aggressive types of diving such as deep wrecks, cave diving, rebreather diving, and mixed-gas diving often elect percutaneous closure of the PFO. This also holds true for divers who have had recurrent "unexpected" DCS events despite diving conservatively as defined above.

Treatment Results

A recent study reported the results of conservative diving practices after an episode of DCS.[14] Eighteen divers in this study had a right-to-left shunt: Nine were small, and nine were large. Mean follow-up was 5.3 years (range 0-11 years). Four of these divers had undergone PFO closure and had no episodes of DCS in follow-up. The absolute risk of suffering DCS before examination for the remaining fourteen divers with right-to-left shunt and no closure was 23.5 DCS events per 10,000 dives for those with a small shunt compared to 71.6 for those with a large shunt. After recommendation for conservative diving practices, the DCS risk at follow-up fell to 6.0 per 10,000 dives in the small-shunt group and 0 in divers with the large shunt. The major limitation to this study is its small sample size, but the results suggest a need for more studies of conservative diving practices for divers with right-to-left shunts.

When DCS has occurred, especially after "undeserved" cases of DCS, divers are often encouraged to seek screening for a shunt, and some diving medical societies classify these divers as ineligible to return to diving.[15] There are also several diving medical specialists who recommend divers with a history of DCS and a positive right-to-left shunt to undergo closure if it turns out to be a PFO, even though there is no clear evidence to indicate that this intervention reduces the risk of DCS or neurologic events.[16-19]

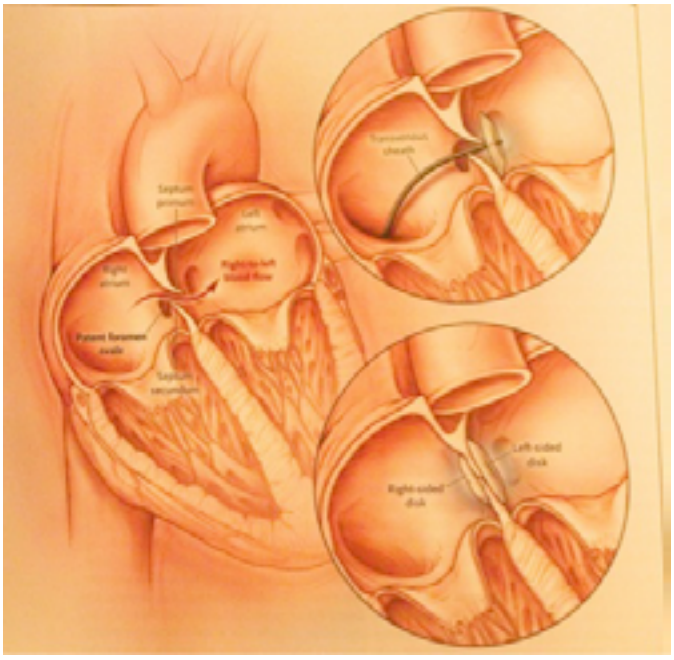
In a 2011 study of 83 scuba divers with a history of DCS and a follow-up of 5.3 years, however, 28 divers had no PFO, 25 had a PFO closure, and 30 continued diving with a PFO without closure.[20] At the beginning of the study, there were no significant differences between the groups in the number of dives, dive profiles, diving depth, or cumulative dives to more than 40 meters of saltwater (msw). After follow-up, while there were no differences between the groups with respect to minor DCS events, the risk for major DCS was significantly higher in the divers with PFO and no closure than in divers with PFO and closure or divers without PFO. Although this offers new evidence that PFO closure reduces the risk for major DCS, the authors do not recommend closure in all divers with a history of DCS but rather recommend further studies to confirm these results.

FIGURE 4. Amplatzer Cribriform Occluder

Percutaneous PFO Closure

The closure procedure for a PFO is relatively painless and is done percutaneously through a femoral vein.

FIGURE 5. Diagram of PFO closure procedure



Imaging during the procedure is done with a combination of fluoroscopy and ultrasound imaging, either TEE or intracardiac echo. The most common device in use in the United States is the Amplatzer Cribriform Occluder (*Figure 4*). This is a wire mesh made out of nickel and a titanium alloy. The device is filled with securely sewn polyester fabric to help close the defect. It is deployed through a small catheter that has been placed across the PFO (*Figure 5*). The procedure takes about an hour, and patients are usually discharged the same day or the following morning.

Ongoing Studies

In 2010, Divers Alert Network (DAN) began a five-year prospective nonrandomized study following divers with a history of DCS and a PFO, whether or not the diver had chosen to close the PFO. At this time 60 of the planned 120 participants are enrolled. Divers interested in participating can find more information at *http://www.diversalertnetwork.org/research/studies/risk_benefit_of_pfo_closure.*

Conclusions and Recommendations

1. **Should all divers be screened for a PFO?**
   No. There is approximately a fivefold increased relative risk of DCS in patients with PFO, but the absolute risk is still quite small.

2. **Should all divers with DCS be screened for a PFO?**
No. PFOs exist in 25% of the population, so one would expect a similar percentage of divers with DCS to have a PFO. Not all scuba dives have the same risk of DCS. The issue with DCS is the inert gas "bubble load," not the PFO. Episodes of DCS in "low-risk" dives, however, especially neurologic events or multiple "undeserved" DCS events, should prompt investigation for PFO.

3. **Should all divers with DCS and PFO have a PFO closure?**
No. Options for divers with PFO and DCS include discontinuing diving, conservative diving practices, or PFO closure. Recommendations should be made on a case-by-case basis based on the DCS event(s), the type of diving performed by the diver involved, and the risks of PFO closure.

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About the Author

DR. DOUGLAS EBERSOLE is a cardiologist specializing in coronary and structural heart interventions at the Watson Clinic LLP in Lakeland, Florida. He is also an avid technical, cave, and rebreather diver and instructor.

Contact Ebersole at debersole@watsonclinic.com.

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Wound Geography and Tissue Types: Part 2

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.

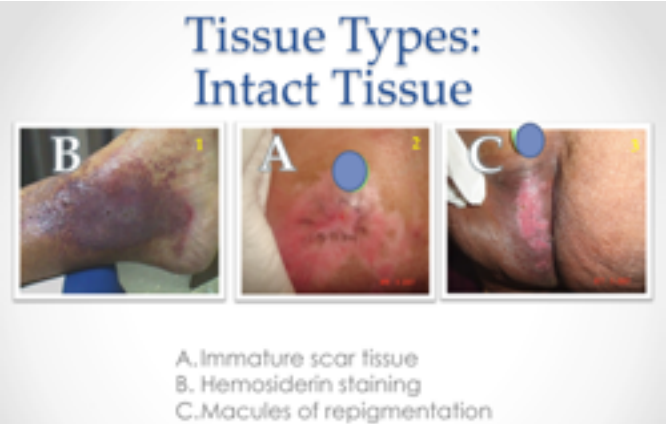
Intact Tissue

Let's begin by looking at intact tissue. Figure 1 shows three different pictures of intact tissue that have some noticeable changes. One picture is representative of immature scar tissue. Another picture is indicative of hemosiderin staining, and a third photo represents macules of repigmentation.

Picture 1 is representative of hemosiderin staining, also known as a "biological tattoo." Venous hypertension leads to regurgitation and backflow of blood. This, combined with insufficient venous valves, leads to a pooling of blood that leaks into the interstitial tissues. Blood outside the vessels begins to break down, and the hemoglobin releases iron, inducing a staining effect on the tissues that presents clinically as hemosiderin staining. This is irreversible but a telltale sign of chronic venous insufficiency.

Picture 2 is representative of immature scar tissue, which tends to present with what we call the three Rs: red, raised, and rigid. This is different than mature scar tissue, which is represented as the three Ps: pale, planar, and pliable. Scar tissue can take several months to two years to become mature.

FIGURE 1.



While scar tissue is actively maturing, there are numerous interventions we can do to help hasten the maturation process. Some interventions include scar mobilization, ultrasound, silicone sheeting, scar massage, pressure or compression, and splinting. It is important to remember that scar modification techniques are effective only while the scar is immature. Once scar tissue is mature, we can no longer modify it or change it.

Scar tissue is never as strong as uninjured tissue. As scar tissue matures, it approaches 80% tensile strength of normal, uninjured tissue but will never regain normal tensile strength. During the maturation process, it is important to protect the area and realize it is always at risk for further breakdown.

Picture 3 is representative of macules of repigmentation. Over time, these macules enlarge and spread, ultimately "resurfacing" the area with near-normal pigmentation. During this process, the area may be slightly hyper- or hypopigmented. Patients with darker skin tones are often concerned about the pink or light color of the tissue, so it is important to educate them about the repigmentation process and reassure them that it will likely normalize over time. However, this process may take several months.

Figure 2 shows two different types of eschar. One is considered stable eschar, and the other is considered unstable eschar. What is the difference?

Stable eschar is noted by intact edges as shown in Picture A. This is a nonfluctuant tissue, which means when you push on it or touch it, it does not yield. Additionally, there is no evidence of drainage or separation at the wound edge. Stable eschar should be monitored as it acts as the body's biological dressing. Aggressive wound care, debridement, and moist wound healing are not warranted when stable eschar is present. At times, the tissue beneath the stable eschar re-epithelializes. If this occurs, the eschar often sloughs off once epithelialization is complete.

FIGURE 2.

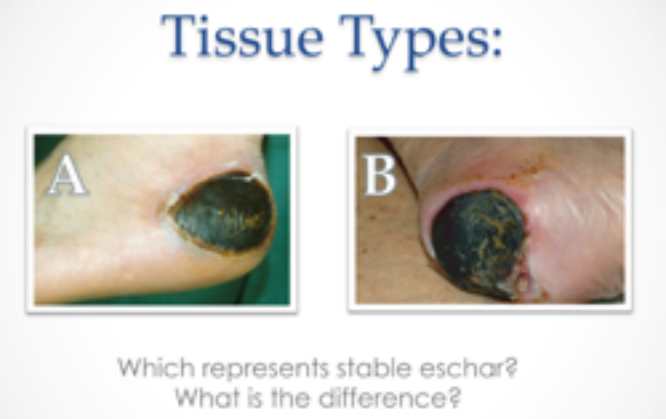
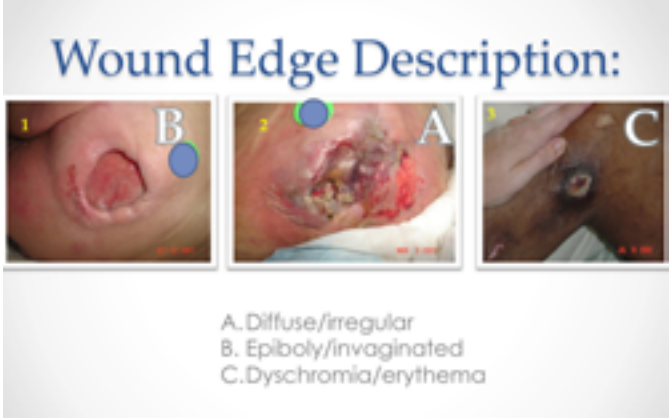


FIGURE 3.



If the eschar begins to demarcate or separate, if the tissue feels boggy or squishy, or if drainage becomes evident as in Picture B, then the eschar is considered unstable and should be managed according to the patient's clinical presentation. An appropriate form of debridement should be selected or performed, and moist wound healing principles should be employed.

Wound Edge

Figure 3 shows pictures of the wound edge. Focus on the area of the wound where the wound base meets the wound margin. One photo is representative of a diffuse or irregular wound edge. Another photo depicts epiboly or invagination, and the third photo presents a wound edge with dyschromia or a form of erythema.

Picture 1 is representative of epiboly or invagination. The epithelial cells were unable to migrate across the wound surface, so in turn, they rolled over the edge, essentially "sealing" the edge. Re-epithelialization cannot occur from a wound edge with epiboly. Sharp debridement of the epiboly, cauterization of the edge with silver nitrate, or roughening the edges must be initiated to help jump-start the wound-resurfacing process.

Picture 2 shows a wound edge that is diffuse or irregular. It is quite difficult to ascertain where the wound edge is located. Wound edges that appear rugged and diffuse are often being subjected to excess friction and shear. Such forces may be iatrogenic or patient-induced if they have cognitive impairments that can disrupt their ability to offload the wounded tissue. Interventions should focus on how to protect the wound from pressure, shear, and friction to allow the wound and wound edge to stabilize.

Picture 3 is indicative of dyschromia. Dyschromia is a darkening of a patient's natural skin tone, and it is how erythema presents on non-Caucasian skin.

Typically erythema is a normal aspect of the inflammatory phase and is confined to the wound margin or perimeter. If it starts to extend beyond the wound edge and well into or beyond the periwound, then we might be dealing with something different, such as an underlying infection.

Picture 3 is a classic presentation of dyschromia in a non-Caucasian patient. It is important to visually look for a darkening of the natural skin tone in lieu of erythema, as it is often very subtle. If needed, you can use a penlight or a flashlight to help you visualize the area.

Periwound Tissue

The next seven photographs focus on the periwound area. The periwound is generally 3-4 cm extending out or away from the wound edge. The wound base, wound edge, and periwound are analogous to an archery target. The center is the wound base, the next area out is the wound edge or margin, followed by the periwound area, which surrounds the bull's-eye. This is often where we need to affix dressings, so it is important to appreciate the quality and integrity of the periwound tissue.

For the following seven pictures, focus on the periwound area, and describe what you see occurring at this location. In Figure 4, is the periwound indurated or hard? Is it macerated or supersaturated? Or is it intact? The answer is the periwound is intact. This is actually a healthy-looking periwound with no overt problems, meaning this periwound is being adequately protected as it is not subject to excessive moisture, nor does it appear denuded or irritated.

Figure 5 has the periwound outlined in black. Is this tissue indurated or hard, macerated or supersaturated, erythematous or all of the above? If you said all of the above, you are correct. Note the shiny firmlike appearance of the periwound. This denotes induration or a hardening of the tissues. If you were

FIGURE 4.

FIGURE 5.

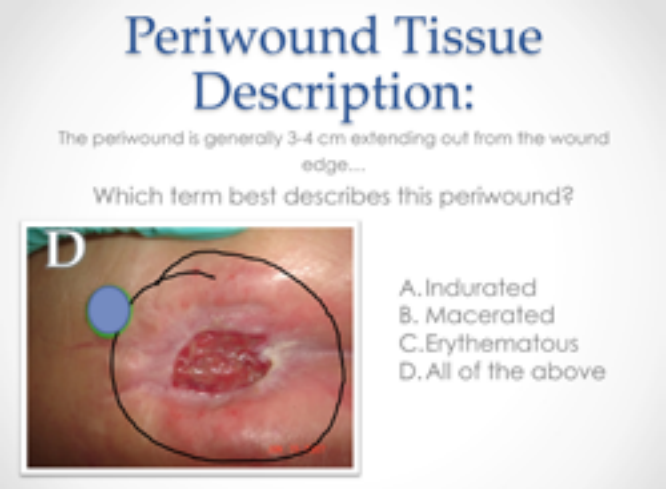
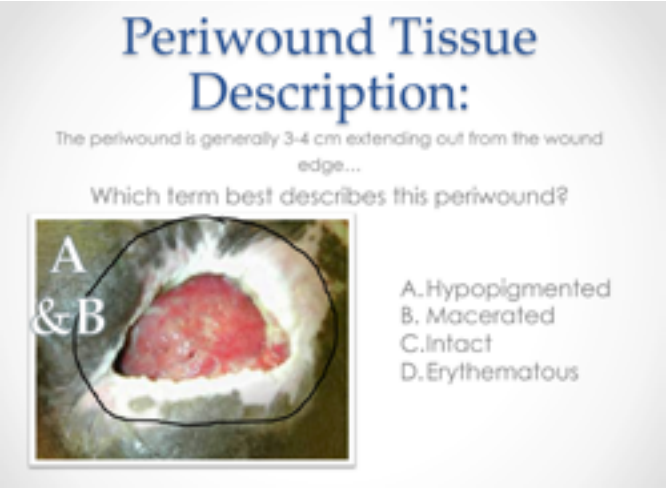


FIGURE 6.



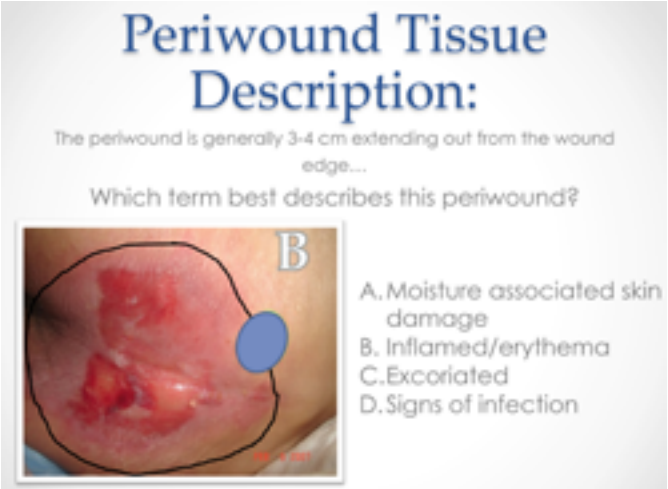
to palpate the tissue, it would feel firm, sometimes even a little waxy in presentation. You can also appreciate some wet-looking tissue or white tissue at the three-o'clock location. This denotes maceration on the right side of the wound. Additionally, the tissue appears red and irritated, indicating a more extensive erythema beyond the wound margin. Collectively, these findings may be indicative of an underlying infection, so further examination would be warranted.

In Figure 6, would you describe this periwound as hypopigmented, macerated, intact, or erythematous? Look closely as there are actually two answers. This periwound is hypopigmented, but it is also somewhat macerated. This can be difficult to see in a picture. it is important to be able to discriminate between these two presentations, however, particularly in patients with darker skin tones. Maceration can make tissue appear white or lighter in color, especially in non-Caucasian patients. This can be managed with more absorptive dressings. For the hypopigmented tissue, it is important to educate and reassure patients about the repigmentation process to adequately manage their expectations.

Figure 7 also outlines the periwound area with a black line. You may have noticed many of these wounds occur at the sacral area. This is a common location for pressure ulcers as 36% of all pressure ulcers occur at the sacrococcygeal area. This is also a common location for moisture-associated skin damage (MASD) due to incontinence. What do you see in this periwound area — MASD, inflammation, or excoriation?

The answer is B. What you're seeing in the periwound area is really an excessive amount of inflammation and erythema. This wound is due to moisture-associated skin damage, which renders the skin-barrier function disrupted. MASD

FIGURE 7.



can predispose an area to further injury and often, pressure ulcers develop concurrently. Note that a pressure ulcer is a different etiology than MASD.

Moisture-Associated Skin Damage Versus Pressure Ulcers

MASD and pressure ulcers are two different etiologies. Pressure ulcers are typically due to shear forces and take on the shape of the pressure-causing agent, whether it's a bone or the patient is laying on something.

MASD is due to moisture where there's presence of excessive moisture on the tissues, and it disrupts the skin-barrier function. The skin has a tipping point as to how much moisture it can be subjected to at any given time. It is not uncommon, especially at the sacral area, to see MASD due to urinary or fecal incontinence. An area with MASD is more likely to develop a pressure ulcer because the skin-barrier function has been disrupted and lacks integrity.

In Figure 8, note the appearance of the periwound area. Take note of the extensive erythema. How would you describe this clinical presentation?

The answer is overt signs of infection. Upon inspection, you can see that the erythema extends beyond the periwound. Linear streaking (lymphangitis) is present and extending away from the periwound. The tissue is red and "angry" in appearance. If you were to palpate the periwound, it may feel hot to touch, and the patient may report higher levels of pain or that their pain level has changed. This is representative of an active infection. Topical and possibly systemic interventions should be implemented to address the infection and associated symptoms.

An area with MASD is more likely to develop a pressure ulcer because the skin-barrier function has been disrupted and lacks integrity.

In Figure 9, which term here best describes the linear marks at the periwound? Look closely at the arrows pointing to the linear marks. Are those marks due to moisture-associated skin damage? Is it inflammation in erythema? Is it excoriated tissue? Or are these skin tears? The answer is excoriation. For thorough documentation, you want to include additional descriptions of how this periwound is presenting. Excoriation is evident by the liner scratch marks. This periwound area, however, is also accompanied with MASD and erythema. It is not uncommon for patients to have an associated pruritus when they're dealing with tissue disruption, especially from moisture. Linear scratch marks, by definition, are considered excoriation. This is different from denudement, which is a diffuse loss of the epidermis.

In the last photograph of the periwound, Figure 10, what do you see? How would you describe this clinical presentation? This is MASD compounded with fungal involvement as indicated by the white, "cheesy" film present on the skin. Take note how this presentation compares to a pressure ulcer. Pressure ulcers typically take on the shape of the pressure-causing agent, whether it be bone or a foreign object. MASD is more diffuse and poorly demarcated. Because MASD disrupts the skin-barrier function, it renders the tissues more susceptible to shear, friction, and pressure

FIGURE 8.

FIGURE 9.

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FIGURE 10.

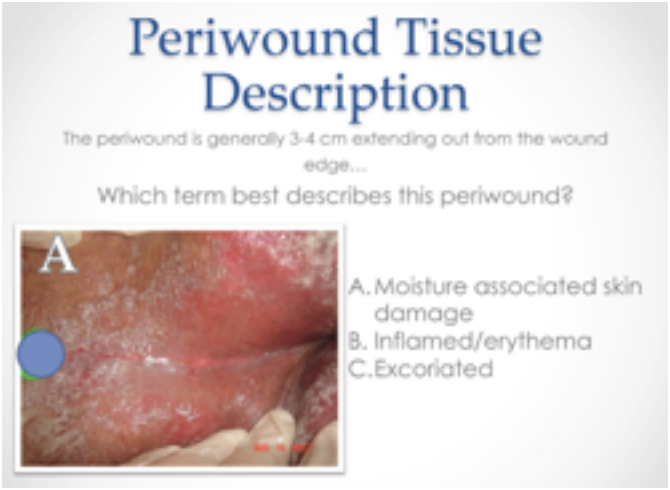

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FIGURE 11.

Photo courtesy of Bragg et al., Pityriasis rubra pilaris, type IV. Dermatol Online J. 2005; 11(4):14.

FIGURE 12.

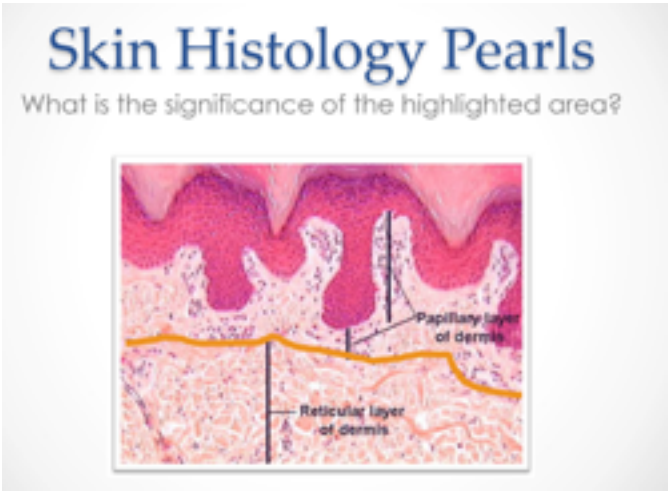

Photo courtesy of https://cyhsanatomy1.wikispaces.com/dermis

forces, and patients may often present with both MASD and a pressure ulcer. Both should be described and documented as separate wound entities.

Now we are going to shift gears and review skin histology. In looking at Figure 11, focus on the purple fingerlike projections. Can you recall the name of these structures and their significance? These are rete ridges or rete pegs, which anchor the dermis to the epidermis to allow the layers to move together as one, mitigating the effects of shear and friction. These structures help to provide skin integrity under stress, strain, and torsion. As we age, our rete ridges reduce in size, adding to age-related skin changes. The skin becomes more fragile and susceptible to minor traumas that can lead to skin tears, senile purpura, and other skin conditions we commonly see in aging skin.

Skin begins to change and age in the third decade of life; however, significant age-related changes become more readily apparent in the sixth decade of life. It is important to be extra-vigilant about skin assessment and skin checks because patients in their 60s and beyond are very prone to minor skin problems that can become major issues, in part due to the reduction in size of the rete ridges. Instead of the epidermis and dermis moving together as one unit, they slip and slide over one another. This can lead to skin tears, friction blisters, purpura, and other trauma from a seemingly minor injury.

In the next histology photo, Figure 12, you will see an area highlighted in orange. I refer to this area as the skin's continental divide. This line represents the two layers of the dermis. The papillary layer is the first layer directly below rete ridges and basement membrane. Trauma into the papillary layer manifests as a partial thickness injury because it is partially through the dermis. Partial thickness injuries do not present with necrotic tissue, and such wounds heal with epithelial resurfacing and no scar tissue. The second layer of the dermis, the reticular layer, is where all the adnexal structures or epidermal appendages reside: hair follicles, sweat glands, sebaceous glands, blood vessels, lymph capillaries, etc. Once permanently damaged, these structures cannot be replaced. Damage into and through the reticular layer results in full thickness wounds. Such wounds heal with granulation tissue formation (raw scar tissue), followed by resurfacing and contraction, creating scar tissue. Unlike partial thickness wounds, full thickness wounds can present with necrotic tissue (slough or eschar).

Clinically, when assessing a wound, it can be difficult to ascertain the level of tissue depth. Typically, partial thickness injuries present with pink or red nongranular and smooth tissue. Full thickness wounds can present with

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beefy red granulation, hyper- or hypogranulation, and even subcutaneous/adipose tissue or deep tissue structures. It is important to note the difference between partial and full thickness wound healing as interventions, and time to full wound closure (resolution) will be different.

Conclusion

Appreciating wound geography, where the wound is located and how the wound is presenting can assist you with differential diagnosis and clinical decision making. This is analogous to a wound scene investigator. Paying attention to the clues at the wound base, wound edge/margin and the periwound area can help direct your plan of care and selection of interventions, improving patient outcomes and quality of life.

Summary

Join us for the CEU video course that complements this article. Details for the video course can be found at *www.WoundEducationPartners.com.* ■

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.

Basic Training

By Darren Mazza, EMT, CHT

Although National Fire Protection Association (NFPA) code 20.3.1.3 explains the role of hyperbaric safety directors and their responsibilities, an individual's job description may vary from one facility to the next. As the safety director for Swedish Edmonds Center for Wound Healing and Hyperbarics in Edmonds, Washington, I have a great deal of responsibility. I take pride that the hospital I work for is committed to supporting me in my role to ensure the safety of both the patients and staff during day-to-day hyperbaric operations. As the safety director, I am responsible for providing training to all staff in our facility, as it pertains to hyperbaric operations, monthly staff emergency training, or just hyperbaric orientation for all new employees hired in our facility.

It's absolutely crucial to provide hyperbaric therapy department orientation to every new employee from the secretary to the wound-care provider. This orientation time is what I call "basic training." Everyone receives one-hour orientation/education pertaining to the operation and safety guidelines of the hyperbaric department. In the event an emergency evacuation is needed and the certified hyperbaric technologist (CHT) needs assistance, everyone receives both basic chamber door operation and gurney operation training and is signed off after orientation time.

Staff training never ends. All staff in our facility participates in monthly hyperbaric emergency procedure training on one of the following topics:

- Otic barotrauma
- Sinus barotrauma
- Anxiety
- Nausea/vomiting/diarrhea
- Hypoglycemia/hyperglycemia patient
- Unresponsive patient oxygen toxicity/seizure
- Pneumothorax
- Gas embolism
- Loss of oxygen pressure/contaminated air
- Power loss
- Severe weather
- Fire in the chamber room
- Fire in the building
- Fire in the chamber

These emergencies and the emergency-response procedures are in plain sight, laminated, and attached to the railings of each chamber.

Every month, I select an emergency topic, and the staff is taught the appropriate emergency response for the specified emergency according to the clinic's hyperbaric emergency procedures policies.

Example: Unresponsive patient

CHT immediate actions:

- Note time and depth.
- Notify MD.
- Abort treatment.
- Start ascent to surface @ 5 psi/min.
- Check vital signs.
- Start basic lifesaving (BLS) protocols as appropriate.
- Check blood glucose.
- Call for assistance as appropriate.

All staff members are in-serviced on three critical components:

1. Type of emergency
2. The CHT appropriate response
3. The appropriate response of all first responders to the chamber room

Constant and adequate communication between the CHT and wound-care staff is a must at all times. Every shift, I communicate my needs for staff assistance for patient change-outs/ prepping to the charge nurse in wound care so she can coordinate with the backup CHT who works in wound care primarily through the day. I then provide a hand

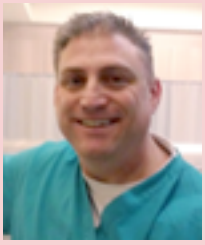
radio to the charge nurse, hyperbaric nurse, and backup CHT so I can maintain constant direct communication with everyone in the event that I may need them for any reason, especially an emergency.

Another responsibility of mine as a safety director is to educate the medical community on both the benefits and risks of hyperbaric therapy. Occasionally I have the opportunity to present to medical providers and other medical professionals. I enjoy this because this enables me to be part of the hospital outreach program, educating the medical community on the evidence-based science and benefits hyperbaric therapy and the conditions we treat in our facility.

Without a doubt, a hyperbaric program with a good safety program in place is also a successful one. The more we educate the staff, the better the outcome in the event of an emergency. One important message for other wound care and hyperbaric facilities is to offer every clinical and nonclinical staff member a UHMS-approved forty-hour introductory to hyperbaric medicine course. I have found that this not only improves hyperbaric safety awareness among staff but also greatly improves staff knowledge in the science and benefits of hyperbaric therapy.

Final note: When it comes to hyperbaric safety, establishing a "basic training" process for the entire staff in your facility will not only better prepare the staff for an emergency but also will improve the day-to-day operation of the hyperbaric program. The entire wound-care staff is more inclined to be vigilant in coordinating care with the CHT as they will have a better sense of time constraints in the hyperbaric environment. ■

About the Author



DARREN MAZZA is the CHT and safety director at the Center for Wound Healing and Hyperbarics at Swedish/Edmonds, located in the greater Seattle area. He has twenty years of experience in health care, which includes eight years as an EMT in the greater Sacramento region. Mazza also worked as a preceptor trauma tech in a Sacramento hospital for several years. After leaving California and moving to Idaho in 2005, his hyperbaric career began after becoming the department head of an outpatient wound care and hyperbaric center.

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