Case report: Treatment of mild traumatic brain injury with hyperbaric oxygen

COLONEL JAMES K. WRIGHT1, EDDIE ZANT2, KEVIN GROOM3, ROBERT E. SCHLEGEL4, KIRBY GILLILAND4

1720th Special Tactics Group, Hurlburt Field, Florida, USA; 2Hyperbaric Medicine Inc., Fort Walton Beach, Florida, USA; 3The Anchor Clinic, Destin, Florida, USA; 4Center for the Study of Human Operator Performance, The University of Oklahoma, Norman, Oklahoma, USA

ABSTRACT
Two United States Air Force Airmen were injured in a roadside improvised explosive device (IED) blast in Iraq in January 2008. Both airmen suffered concussive injuries and developed irritability, sleep disturbances, headaches, memory difficulties and cognitive difficulties as symptoms of mild traumatic brain injury (mTBI). Six months after injury, repeat Automated Neuropsychological Assessment Metrics (ANAM) testing showed deterioration, when compared to pre-injury baseline ANAM assessment, in all measured areas (simple reaction time, procedural reaction time, code substitution learning, code substitution delayed, mathematical processing, and matching to sample).

The airmen were treated with hyperbaric oxygen in treatments of 100% oxygen for one hour at 1.5 atmospheres absolute, resulting in rapid improvement of headaches and sleep disturbances, improvement in all symptoms and resolution of most symptoms. Repeat ANAM testing after completion of the hyperbaric treatments — nine months after initial injury — showed improvement in all areas, with most measures improving to pre-injury baseline levels. The airmen received no other treatment besides medical monitoring. Repeat neuropsychologic testing confirmed the improvement. We conclude that the improvement in symptoms and ANAM performance is most likely attributable to HBO treatment.

INTRODUCTION
Traumatic brain injury has been called one of the signature injuries of Operations Enduring Freedom and Iraqi Freedom. The RAND Report documented a 19% self-reported incidence of probable TBI among returning service members, with 320,000 probable TBI cases. Most of these cases (80%) are considered mild traumatic brain injury, or mTBI (1).

On a per-case basis, one-year costs for mTBI were estimated at $27,259 to $32,759 in 2007 (2). The lifetime costs of even mild TBI impairment in young service members can be deemed incalculable (3).

Mild TBI is usually characterized by a concussive event that causes a brief period of unconsciousness (lasting less than 30 minutes) or a period of confusion or amnesia lasting less than 24 hours. The Department of Defense has developed criteria for the diagnosis of mTBI, which must include one of the following:

1) any period of loss of or a decreased level of consciousness lasting less than 30 minutes;
2) any loss of memory for events immediately before or after the injury lasting less than 24 hours after the event;
3) any alteration in mental state at the time of the injury such as confusion, disorientation, or slowed thinking lasting less than 24 hours after the event;
4) transient neurological deficits (e.g., weakness, loss of balance, change in vision, praxis, paresis or plegia, sensory loss, aphasia); and
5) normal intracranial imaging.

Findings may be transitory, and late sequelae that are not explainable by other means may qualify an individual for the diagnosis of mTBI. Patients with more than one of these findings may be assigned a higher level of TBI (4).
Since the symptoms of mTBI may develop gradually, are often subtle, and can be confused with other illness such as post traumatic stress disorder, mTBI may be unrecognized and undiagnosed (5). A concussive injury causes diffuse axonal injury, structural neuronal damage and diffuse neuronal dysfunction (6).

The symptoms of mTBI are variable and may include headache, irritability, impulsivity, anger, cognitive impairment, memory difficulty, loss of executive function, and vestibular and sleep disturbances (7). Electroencephalogram and sleep studies are usually normal. Most individuals with mTBI recover in three to 12 months, especially those who are young (8). However, some victims do not recover, or recover slowly; they are at risk for future injury and deterioration of brain function (9).

Mild TBI usually resolves without treatment within months, although approximately 20% of patients with mild TBI continue to have lingering symptoms for one year or longer after injury (1,10). Poor scores on neuropsychological testing months after injury have been correlated with poorer outcomes and unresolved symptoms (11).

Patients with several post-concussive symptoms are unlikely to improve after one year, in spite of traditional therapy (12). Treatment of mild TBI has included rest and observation, education, cognitive rehabilitation and phamacotherapy (13).

Pharmacologic treatment may be required for control of disabling symptoms of headache, irritability, depression, and anger (14). Because of the efficacy of hyperbaric oxygen (HBO) in treating brain dysfunction from decompression sickness and carbon monoxide injuries, as well as anecdotal reports of its efficacy in treating concussive injuries, we felt HBO might prove of use in treating two airmen injured in a blast.

CASE REPORT

In January 2008 Airman B, a 23-year-old male vehicle operator, was a convoy lead vehicle commander sitting in the passenger seat of an M915 14-ton truck. Airman C, a 22-year-old male vehicle operator, was driving the vehicle that was attacked with an improvised explosive device (IED). The detonation occurred on the passenger side of the vehicle, nearer to where Airman B was sitting. The vehicle was damaged, and Airmen B and C sustained concussive injuries with a sense of being dazed for several minutes. There was no known direct blow to the head for either occupant or loss of consciousness, although both occupants had tinnitus. Airman B, who was approximately 3 feet closer to the blast, suffered immediately from a severe headache. Airman C continued to drive the damaged vehicle for several minutes and had no immediate symptoms other than being slightly dazed; however, he developed a mild headache some hours later.

Later in the day, Airmen B and C reported to the medical clinic, where no additional injuries were found. They were given acetaminophen for their headaches and placed on light duty. Two weeks later their symptoms had largely resolved, and they were returned to full duty.

Three weeks post-injury both airmen noted the return of headaches, with difficulty sleeping. Airman B expressed his headache severity as 5-6 and Airman C as 4-5 (on a scale of 1-10, with 10 being the most severe pain imaginable) with headaches occurring daily and lasting for several hours. Both individuals had difficulty falling and remaining asleep, and they reported sleep duration of three to six hours per night. Additionally both individuals felt they were quick to anger and stayed angry from trivial provocations for several hours. Lack of attention to detail, forgetfulness, and fatigue were also reported by both airmen. These latter symptoms began insidiously about three weeks after injury, progressed for about two months and remained constant for the next four months, until treatment with HBO was administered.

Upon arrival at their home base, the airmen presented to the clinic complaining of headaches, fatigue, lapses in memory, irritability and sleep disturbances. Neurological exams were normal, although the airmen appeared tired. Computerized tomography of the brain, EEGs and sleep studies were normal.

On initial deployment both airmen had received the Automated Neuropsychological Assessment Metrics test (ANAM) on 11 November
2007, two months prior to injury. This test was repeated on 21 July 2008, six months after injury. The repeat ANAM testing showed marked declines from the pre-injury baseline in several areas of measurement (Figures 1A and 1B, Page 394).

Airman B presented a statistically significant change in Simple Reaction Time and Matching to Sample tests, with declines in all other areas. Detailed neuropsychological testing of Airman B at six months post-injury and prior to HBO therapy revealed a diffuse or scattered pattern of deficits. Although his IQ score was within the average range, his neuropsychological functioning on a summary measure (Repeatable Battery for the Assessment of Neuropsychological Status – RBANS Form A) (15) was at just the 7th percentile.

Moreover, Airman B showed marked attention dysfunction for both auditory and visual material; cognitive processing speed was slowed and subjectively observed in casual conversation with the patient. He showed difficulty in repeating sentences and digit sequences as well as learning digit sequences over repeated trials.

Airman B also demonstrated problems in both verbal learning and visual memory. His reading speed was slowed, fingertip-tapping speed was slowed in both hands, and clerical speed for coding tasks was mildly impaired. He showed difficulty for rhythm perception and visual-motor integration for copying geometric designs. His reaction time was slowed on a computerized measure of attention. Reading level for sight words remained at the college level, but written arithmetic was at just the sixth-grade level.

Airman B also demonstrated statistically significant and drastic changes in both Simple Reaction Time modules (at the beginning and end of the battery), along with declines in all other areas except Mathematical Processing. Detailed neuropsychological testing of Airman C at the same time — prior to HBO therapy — was largely within normal limits notwithstanding problems for inconsistent attention and upper-right extremity dysfunction for grip strength and somatomotor integration. His RBANS (Form A) total score was at the 50th percentile, average range.

Initially, treatment of the headaches with ibuprofen and butalbital-aspirin-caffeine capsules (Fiorinal®) was tried, but these drugs were ineffective in relieving the pain. The airmen were placed on limited duty and daytime work only.

As the airmen had experienced at least one of the symptoms of mTBI after the blast (confusion, alteration of mental state) and their symptoms had no other reasonable explanation, they were given the diagnosis of mTBI in accordance with the Department of Defense criteria (4).

Because the two airmen had shown no improvement in their symptoms for seven months and were having difficulty performing their occupations, it was decided to begin hyperbaric oxygen treatment. Treatment with HBO was begun eight months post-initial injury. The treatment protocol was 100% oxygen for one hour at 1.5 atmospheres absolute. Treatments were given five days per week.

Clinical improvement was rapid. Airman C reported that his headaches vanished by the fifth treatment and did not return, and that he was able to sleep seven to eight hours per night uninterrupted. Airman B reported that his headaches weakened to 3-4 on a pain scale of 1-10, lasted only one to two hours instead of the previous eight to 10 hours, and that he was able to sleep eight to nine hours per night uninterrupted.

Both airmen reported that they felt more mentally alert and were less prone to forgetting, although they still did not feel “normal.” At the completion of the 40-treatment protocol, Airman C felt that his symptoms had ostensibly resolved, and Airman B felt that he was much improved, notwithstanding some lingering irritability and forgetfulness.

Repeat ANAM testing showed improvement in essentially all areas for both airmen. Airman C’s ANAM scores returned to pre-injury baseline levels, and Airman B’s ANAM scores returned to pre-injury levels, with no statistically significant differences in any of the tested domains (Figures 1A and 1B, Page 394, and Figures 2A and 2B, Pages 395-396).
areas of cognitive functioning after HBO therapy at 10 months post-injury. His RBANS (Form B) total score was at the 12th percentile. For a patient with mild to moderate TBI, his scores improved faster than would be expected through spontaneous brain healing alone during this time interval. Areas of objective improvement included visuoconstructive abilities, fingertip-tapping speed and verbal learning/memory for word lists. His cognitive abilities status post-HBO treatment was deemed satisfactory to continue his job duties without special monitoring.

Repeat neuropsychological testing of Airman C was generally consistent with his pre-treatment test scores. Areas of subtle improvement such as motor abilities in the dominant right hand, written arithmetic and verbal fluency were observed. His RBANS B total score was at the 47th percentile, which was not a significant change from pre-treatment testing.

**FIGURE 1A — Airman B ANAM Scores**

**FIGURE 1B — Airman C ANAM Scores**

*Throughput scores are presented as the percentile of the comparison group of military members without TBI.*
Airman C was essentially well. Based on these results, it was decided to return Airman C to full duty, while Airman B continued hyperbaric treatment for another 40 treatments following the original treatment protocol.

Repeat ANAM testing on Airman B at the conclusion of the second set of 40 HBO treatments showed improvement in all measures at or exceeding his pre-injury state, except for matching to sample, which was improved markedly from the injury state (Figures 1A and 2A, Page 394 and above).

Airman B reported that he had made continued improvement in cognitive function, felt much more alert and had returned to his pre-injury functional
DISCUSSION

Hyperbaric oxygen treatment has several effects that may be beneficial in treating brain injury. In animal models, HBO has been shown to enhance mitochondrial recovery and to reduce apoptosis in hypoxic nerve cells (16,17). The HBO-induced improvement in mitochondrial function appears to facilitate improved cognitive recovery and reduced hippocampal neuronal cell loss after brain injury (18).

HBO promotes neural stem cell activation and growth (19, 20), and this effect is seen in the hypoxic-damaged brain (21). HBO also alleviates hypoxic-induced myelin damage, up-regulates HIF-1 alpha-enhancing neuronal tolerance to hypoxia, and increases cellular ATP levels and cognitive recovery after concussive injury (22).

Balance beam scores in rats with cerebral contusions were improved after treatment with HBO (23). In a rat model of chronic TBI, HBO improved spatial learning and increased vascular density in the injured hippocampus (24).

Controlled human studies of the efficacy of HBO after brain injury have been few. In a study state. He reported that he was experiencing eight hours of uninterrupted sleep per night, and that his headaches had diminished to about one per week.

He also noted the pain intensity had further decreased to 2-3 on a scale of 1-10, and that the headaches lasted two to three hours versus the original eight to 10 hours’ duration.

**FIGURE 2B — Airman CANAM Scores**
of moderate and severe TBI using the Glasgow Coma Scale and Glasgow Outcome Scale as measures of efficacy, an HBO-treated patient showed improvement over controls (25). HBO has been shown to be clinically effective in mediating the effects of brain injury (26). While the exact mechanism is unknown, HBO is thought to restore neural pathways damaged in TBI with supporting evidence supplied from SPECT brain imaging (27).

ANAM is a library of more than 30 computer-based test modules designed for a wide variety of clinical and research applications and is the direct outgrowth of more than 20 years of computer-based test development across all service branches within the Department of Defense (28). ANAM4™ is a neurocognitive assessment tool that can be used to identify changes in a service member’s cognitive function and mood state as a result of some debilitating event.

The ANAM4™ TBI-MIL test battery used in this case report has been tailored to provide an instrument that is sensitive to cognitive changes that often accompany mTBI. The battery consists of a set of assessment modules that gather data on mood, processing speed (reaction time), working memory, short-term memory, spatial pattern recognition/memory and other cognitive functions. The test is designed for repeated testing and provides reliable measures when used for retesting as a measure of TBI recovery (29).

ANAM is used to establish a cognitive function baseline that can then be used for surveillance post-injury or after suspected injury (30). Although not intended as a diagnostic tool per se, comparative performance on ANAM test modules can be helpful in confirming the diagnosis, as demonstrated in this case report. In cases with known head trauma, computer-based assessments should be supplemented with detailed neuropsychological tests tailored to the patient’s presenting problems and to the specific referral question to be answered.

CONCLUSIONS

Several aspects of these two cases demonstrate the efficacy of HBO for the airmen treated. Although both airmen had stable symptoms of mTBI/post-concussive syndrome that had not improved for seven months, substantive improvement was achieved within 10 days of HBO treatment. The headaches and sleep disturbances improved rapidly, while the irritability, cognitive defects and memory difficulties improved more slowly.

Fortunately, both airmen had taken the ANAM and presented objective demonstration of their deficits from TBI and their improvements after HBO treatment. Both airmen, who were injured by the same blast sitting side by side, had similar symptom complexes of TBI and improved at similar rates after initiation of HBO treatment. Neither airman had any other form of treatment for TBI. It seems unlikely to the authors that any explanation other than the HBO treatments can be offered for their improvements.

REFERENCES


ABSTRACT

Four patients received hyperbaric oxygen therapy (HBOT): Three patients had nonarteritic central retinal artery occlusion (CRAO); a fourth patient had a CRAO in the right eye (OD) and a branch retinal artery occlusion (BRAO) with macular involvement in the left eye (OS) secondary to giant cell arteritis.

The first two patients presented with a one-day history of CRAO, the third patient with a 10-day history of CRAO OD and the fourth patient with a three-week history of CRAO OD and a three-day history of BRAO OS.

The initial visual acuities ranged from light perception to counting fingers at 6 feet. The visual acuity and visual field improved in the first two patients with nonarteritic CRAO. Patients 3 and 4 did not improve. There were no complications. Hyperbaric oxygen treatment may be safe and effective in selected patients with nonacute, nonarteritic CRAO.

INTRODUCTION

Central retinal artery occlusion (CRAO) is an ophthalmologic emergency that frequently results in severe visual loss (1-3). Spontaneous improvement following visual loss is rare; this has led to the development of many proposed treatments including ocular massage, anterior chamber paracentesis, pharmacologic therapy, catheterization of the femoral artery with infusion of thrombolytic agents, etc. (2)

The many causes of this condition are reflected in the varying success of the various treatment modalities. The goal of these therapies is to remove the occlusion, thus increasing retinal oxygenation or maintaining retinal oxygenation until spontaneous recanalization occurs. Recanalization and reperfusion of the central retinal artery generally occur within 72 hours of thrombotic occlusion (2). Hyperoxia can restore retinal oxygenation after arterial occlusion, and favorable results using hyperbaric oxygen therapy (HBOT) for acute retinal artery occlusions have been reported (4-6).

Hayreh (7, 8, 9) has posited that there can be no effective treatment for nonacute CRAO cases with durations longer than 240 minutes based upon his study of old, atherosclerotic and hypertensive rhesus monkeys. Yet, Duker and Brown (10) reported spontaneous reperfusion and visual acuity improvement from light perception to 20/30 in a 70-year-old man with a 96-hour history of a combined CRAO and lateral posterior ciliary artery obstruction.

Hayreh’s acute clamping of the CRA in a rhesus monkey model may not be analogous to the human clinical situation. The human retina may be more resilient than previously thought. Duker and Brown (10) state that “the reversal of the arterial obstruction or increased retinal oxygenation through some other means during this period could theoretically lead to an improvement in vision in these specific cases.” I have previously reported (11) treating three patients with CRAO of six-, seven- and eight-day durations with HBOT at 1.5 ATA. 1.5 ATA was empirically chosen for several reasons. The retina is a neural tissue and Holbach, et al. (12) reported that 1.5 ATA had a “favorable effect” on injured brain tissue as compared to 2.0 ATA. Animal and human studies have demonstrated that hyperoxia can restore retinal oxygenation (13,14). Visual improvement during treatment with supplemental normobaric oxygen also implies that mild hyperbaric pressure may be sufficient (14,15). Using a
lower ATA would also minimize the risk of complications and shorten the treatment time, making the HBOT more “palatable” to what is generally an elderly population, as well as to sometimes skeptical family members and referring physicians.

The pretreatment visual acuities were hand motion, hand motion and counting fingers (CF) at 3 feet, respectively. Post treatment, the visual acuities had improved to 20/200-1 after three HBOT treatments, 20/60+2 after five HBOT treatments and 20/50 post 10 HBOT treatments, respectively. There was no improvement in the visual field of any treated patient. As the retina in the macular area is thinner than the surrounding area (accounting for the cherry-red spot that represents the choroidal circulation seen through the transparent macula retina and highlighted by the surrounding edematous retina), the metabolic requirements are presumably less than the peripheral retina and more resilient to a decrease in perfusion.

A similar group of four patients with a one- to three-day duration of CRAO that refused HBOT treatment presented with visual acuities of no light perception (nLP), CF at 3 feet, CF at 6 feet and CF at 6 feet, respectively. The first patient's vision improved from nLP to hand motion. Patients 2 through 4 had no improvement in visual acuity or visual field.

HBOT treatment of an additional four patients with a one- to three-week history of CRAO is now reported. The effectiveness of treatment is discussed in relation to the patient’s medical condition.

Each patient underwent ophthalmic examination that included best corrected visual acuity, intraocular pressure, biomicroscopic and dilated fundus examinations, fundus photography, fluorescein angiography and other ancillary testing, as indicated. No adjunctive therapies – i.e., anterior chamber paracentesis, pharmaceuticals, etc. – were used. Patients were screened for the suitability of administering HBOT, and treatments were performed with 100% oxygen at 1.5 ATA for 1 hour.

**Case 1**

A 64-year-old woman with a history of diabetes, hypertension and glaucoma awoke with loss of vision OD. She was examined at 4 p.m. that same day, and the visual acuity was CF at 5 feet OD and 20/30 OS. Dilated fundus examination with fluorescein angiographic confirmation revealed a CRAO OD. Platelet emboli and a cherry-red spot were observed. (At a prior examination five month earlier, the visual acuity was 20/30 OD and 20/25 OS.)

She underwent one HBOT each day for four days, with the first treatment beginning the next morning. When she was next examined – four days later (Day 5 post CRAO) – the visual acuity had improved to 20/200. There was a decrease in the previously observed retinal edema, although the cherry-red spot was still visible. A marked improvement in the arteriolar filling time was observed by fluorescein angiography. The visual field showed no improvement.

The patient underwent six additional HBOT treatments, one per day. On Day 11 post CRAO, though there was no improvement in the central visual acuity, there was a mild improvement in the visual field. The examination remained stable two months later.

**Case 2**

A 91-year-old male with diabetes, cardiac disease and hypertension awoke with a loss of vision OD. He was examined at 11 a.m. the same day; the visual acuity was hand motions OD and 20/30 OS. Dilated fundus examination, confirmed by fluorescein angiography, revealed a CRAO OD. Platelet emboli were noted. The patient underwent one HBOT treatment that afternoon. He subsequently underwent two treatments on Day 2, two on Day 3 and one on Day 4. When he returned three days later (Day 4 post CRAO), the visual acuity OD had improved to 20/400 with eccentric fixation, and fluorescein angiography revealed an improvement in arteriolar filling.

He underwent six additional HBOT treatments over the next eight days. Twelve days later (Day 16 post CRAO), the visual acuity OD had further improved to 20/100, and a marked improvement in the visual field was observed. His medical work-up was positive for a 75% blockage of his right carotid artery, and he was scheduled for carotid endarterectomy.

Encouraged by the visual improvement, he elected to undergo six additional HBOT treatments, two treatments per day. Three days later,
19 days after the initial diagnosis, the visual acuity OD had improved to 20/30, with a concomitant improvement in the patient’s visual field.

**Case 3**
A 55-year-old male experienced a CRAO OD secondary to an embolic plaque from a right carotid occlusion. He underwent carotid endarterectomy four days afterward and was referred for consideration of HBOT six days later (Day 10 post CRAO).

The visual acuity was light perception (LP) OD and 20/400 secondary to amblyopia OS. A relative afferent pupillary defect was present OD. The anterior chamber was shallow, and gonioscopy revealed an occludable angle in each eye. Dilated fundus examination demonstrated a CRAO with retinal edema at the posterior pole and a cherry-red spot OD. There was a very significant delay in arteriolar filling time by fluorescein angiography (greater than 45 seconds). Subretinal fluid with a retinal thickness at the macula of 365 microns was noted by ocular coherence tomography (OCT).

The patient began HBOT at 1.5 ATA the next morning and underwent two treatments per day over the next four days. There was no significant change in the visual acuity on Day 14 after the CRAO. Less retinal edema was observed, and OCT showed there was a decrease in the subretinal fluid. The retinal thickness at the macula was 308 microns.

He underwent an additional seven HBOT treatments at 2.0 ATA, but due to sinus problems, the final two treatments were at 1.5 ATA. Twenty-one days after experiencing the CRAO, the visual acuity remained at LP OD, there was less retinal edema, an improvement in arteriolar filling time by fluorescein angiography was noted, and there was a further decrease in the subretinal fluid by OCT such that the retinal thickness at the macula was 269 microns.

**Case 4**
A 93-year-old woman with hypertension was referred with a three-week history of loss of vision OD and a three-day history of visual loss OS. The visual acuity was light perception OD and CF at 6 feet OS. Dilated fundus examination, confirmed with fluorescein angiography, demonstrated a CRAO OD and an inferior temporal branch retinal artery occlusion with macular involvement OS. Emboli were not observed in either eye. OCT did not demonstrate subretinal fluid.

An erythrocyte sedimentation rate (ESR) was normal for her age and although she denied systemic symptoms suggestive of giant cell arteritis (temporal arteritis), she underwent biopsy of the right and left temporal artery. Both specimens were diagnosed with severe temporal arteritis. The patient was placed on prednisone, 60 mg per day; she also began a course of six HBOT treatments, one per day, at 1.5 ATA for one hour. There was no change in the visual acuity or visual field of either eye following HBOT treatment.

**DISCUSSION**
In Hayreh’s model of elderly, atherosclerotic and hypertensive rhesus monkeys, a CRAO induced by clamping the artery for 240 minutes or longer resulted in massive and irreversible retinal damage (7, 8, 9). Unfortunately, the extrapolation of these facts to humans may have prevented research into treating longer-standing retinal artery occlusions.

Retinal perfusion has many variables, including the varying degrees and acuteness of the reduction in flow and the range, depending upon the patient, of differing perfusion pressures required to avoid retinal damage in different areas of the retina. There are multiple factors affecting the transit time of fluorescein dye: the size and distance of the vessel chosen for injection; the patient’s pulse rate, blood pressure and blood flow to the eye; and the fact that the test is performed on a patient when they are sitting (especially in the elderly) may not be truly representative of the arteriolar transit time to the eye of a supine patient undergoing HBOT.

The CRA may not be totally occluded but partially occluded or obstructed, and there are many variables that would determine the perfusion distal to the obstructed area.

It is reasonable to assume that, depending on the type and degree of obstruction, there will still be flow around the obstruction. This differs from the acute and complete occlusion
in Hayreh’s monkey experiments. Delayed arteriolar filling of fluorescein dye in patients with CRAO is frequently seen, which Hayreh feels is artifactual, a point disputed by others (16). It is also reasonable to assume that the slow obstruction of a vessel may enlist compensatory mechanisms not present in Hayreh’s rhesus monkey model of an acute clamping of the central retinal artery.

There are other factors to consider. HBOT has been shown to reduce intraocular pressure, presumably from a decrease in episcleral venous pressure or a reduction in aqueous formation (17). The luminal size and shape of human cerebral bifurcations change with distending pressure (18). Changes in shape will vary the cross-sectional area, which would lead to a change in acceleration of the flow to the eye.

It is unknown whether the viscoelastic properties of the carotid bifurcation attenuate the variations in flow between systole and diastole. In addition, tethering of the anatomic structures may also act to constrain luminal shape changes.

These variables have not been addressed in the natural history studies that have previously been published, and it is unknown what role they play. As the HBOT treatments were all performed with the patient supine, the perfusion pressure to the eye (in this group of patients with vasculopathy) may be greater than if the patient were sitting. HBOT may not only provide increased oxygenation, but, the reduction in intraocular pressure and an increase in perfusion pressure may help to move an embolus or thrombus to a more distal site and allow the “ischemic penumbra” to sufficiently reperfuse and to become functional.

These factors may all help to explain the temporary visual improvements described in patients while breathing supplemental, normobaric oxygen, as compared to the more long-standing visual improvement of those patients undergoing hyperbaric oxygen therapy.

The presence of subretinal fluid by OCT (Patient 3) may have contributed to the poor visual results. Whether pharmaceutical intervention, *i.e.*, acetazolamide, which may reduce subretinal fluid, would have been beneficial in this instance is unknown. Future studies should include OCT assessment to determine whether the presence of subretinal fluid holds therapeutic implications.

Giant cell arteritis (temporal arteritis) is an important clinical entity whose mechanism of visual loss – and consequently its therapy – should not be grouped with nonarteritic CRAO. Unlike CRAO, temporal arteritis may produce a CRAO and also involve the posterior ciliary artery, the occlusion of which causes an anterior ischemic optic neuropathy and massive visual loss, sometimes in both eyes, as in Case 4. It is imperative that when suspected, the diagnosis and treatment of this condition should be promptly instituted.

Likewise, prompt investigation for the source of embolic phenomena is important to prevent a cerebral stroke from occurring. Treating the CRAO with HBOT should begin after the conditions that may result in serious morbidity and mortality have been excluded.

CRAO occurs in only 0.85 per 100,000 persons per year (2), which has made standardizing treatments difficult in a large trial. Yet, in Europe, the first randomized controlled prospective study comparing two treatment strategies for CRAO is ongoing (19). The EAGLE study was begun in June 2002, and the calculated sample size is 100 patients per subtrial (200 patients total). As of April 2005, 47 patients had been enrolled at 16 centers. The study seeks to compare “conservative therapy,” which they define as isovolemic hemodilution, ocular massage, one eyedrop of timolol 0.5% (*i.e.*, a beta-blocker) and anticoagulation therapy for at least six months versus intra-arterial fibrinolysis. Inclusion criteria include CRAO not older than 20 hours.

Hayreh’s criticisms of the study (20) includes the 20-hour duration (which he feels is too long), the lack of a control group, the absence of specific information regarding patients with giant cell arteritis and, most importantly, that only 15.5% of emboli are platelet-fibrin in nature. The majority of emboli, 84.5%, are composed of cholesterol or calcium and are unlikely to be affected by fibrinolytic therapy (20).

This procedure is not without risk, including death (21), requires the continuous presence of specialized personnel and is expensive.

HBOT may address many of these concerns. The treatment is relatively safe, easily administered, low-cost (by comparison) and may potentially result
in better visual results, as it restores oxygen to the retina, irrespective of the type of embolus producing the occlusion (though this may affect long-term success). It seems apparent that in the above cases of nonarteritic retinal arterial occlusion, the retina may retain functional ability for a longer period of time than previously thought.

References


Letter to the Editor

Air embolism’s new scarlet letter

PETER J. MARIANI and NORMA COONEY
Division of Hyperbaric Medicine, Department of Emergency Medicine, SUNY Upstate Medical University, Syracuse, New York, USA

To the Editor:
On October 1, 2008, federal Centers for Medicare & Medicaid Services (CMS) policy went into effect, eliminating Medicare facility payments for certain hospital-acquired adverse conditions (1, 2). With input from the National Quality Forum and the general public, eight so-called “Never Events” were compiled, adopted and encouraged for additional incorporation into individual states’ Medicaid systems (3). Included among these Never-Event hospital-acquired conditions (HACs) are catheter-associated infection, unintended retained operative foreign body and air embolism.

In selecting its initial eight HACs, CMS judged five attributes to be desirable:
“1. unambiguous;
2. usually preventable;
3. serious;
4. indicative of a safety system problem; and
5. important for public accountability” (4).

Closer examination reveals that the first attribute pertains to a condition being “clearly identified” by a unique ICD-9 diagnostic code (5, 6). This dubious equivalence of non-ambiguity and ICD-9 codeability might not pose concerns regarding the operatively retained foreign body. That condition is both explicitly codeable and with little doubt as to final diagnosis. (Either a hemostat was left in the peritoneum or it was not.) For the case of air embolism, however, such bright line diagnostic certainty is often lacking, and unintended consequences could result.

The diagnosis of arterial gas embolism is predominantly clinical (7). The decision to treat should not hinge on gas confirmation by computed tomography (8-10). For patients at risk, the diagnosis should be high on the differential; and “under suspicious circumstances, air embolism should be assumed present unless proven otherwise” (11). Expeditious hyperbaric treatment can improve clinical outcome, even in absence of radiographically demonstrable air (10).

Under federal reasoning, withholding payment for a forbidden HAC deters against its future occurrence (4). Whether this holds true specifically for iatrogenic air embolism can be put to the test. CMS maintains data linking individual reimbursements to ICD codes, serving as window into disease incidence. For instance, in 2007, CMS noted 57 cases of air embolism among Medicare beneficiaries (5). Notwithstanding its ostensible small size, this figure (as well as perhaps others) could serve as a baseline against which to gauge effects of the new rule.

Unfortunately, not only might confounding variables result in the wrong conclusion as to causation, an unanticipated effect of the primary variable itself could also lead to error. Let’s presume that the reported incidence of air embolism, by whoever’s measurement, decreases after rule implementation, resulting in a declaration of victory by federal rule-makers. Alas, the victory is a mirage, albeit the direct result of the rule, but a mirage nonetheless.
The reality: Due to its new stigma and old inherent diagnostic uncertainty, there are simply fewer diagnoses of air embolism being made for patients who previously would have received the diagnosis. If frank air isn’t henceforth manifest on the CT, then it didn’t happen. There will be ongoing hospital fiscal incentive against diagnosing (and consequently treating) the unsure or questionable case. Patients with small subtle air emboli run the risk of being denied their definitive treatments (10, 12) and could suffer long-term neurologic sequelae as a result.

Is the above argument tantamount to declaring a federal rule to be flawed and potentially harmful to patients due to speculated effects on dishonest providers? No. It is merely an observation that now exists a sizeable factor to tip otherwise balanced medical decision scales towards one direction. This consequence is foreseeable to those having foresight. The scarlet “A” worn by Hester Prynne “had an effect like a spell … enclosing her in a sphere” in announcing her adultery to seventeenth-century Boston (13). In contemporary American hospitals, “A” now stands for air. Having it found where it ought not be is a stigmatized scarlet “Never Event.” Those caring for patients and performing procedures at risk should continue refining preventive strategies. Referring practitioners and consulting hyperbaracists should remain objective when assessing patients for air embolism. Professionals must resist coercion – overt or subtle – against making the diagnosis and must maintain appropriately low thresholds for presumptive treatment with hyperbaric oxygen.

References
1. Medicare Program; Changes to the Hospital Inpatient Prospective Payment Systems and Fiscal Year 2009 Rates; Preventable Hospital-Acquired Conditions (HACs) Including Infections. 73 Federal Register 161 (19 Aug 2008), pp. 48471 - 48490.
2. Medicare Program; Changes to the Hospital Inpatient Prospective Payment Systems and Fiscal Year 2008 Rates; Hospital-Acquired Conditions Including Infections. 72 Federal Register 162 (22 Aug 2007), pp. 47200 - 47218.
5. op cit 1, p 48473.
6. op cit 2, p 47202.
Theoretical and practical solutions as to how to ascend or decompress after scuba diving have been considered since Haldane some 100 years ago, and yet decompression sickness (DCS) still occurs.

The traditional “Haldanian” approach to planning decompression has been to limit supersaturation (the difference between tissue inert gas tension and ambient pressure) according to empirically derived rules that purportedly maximize gradients for inert gas washout and therefore provide a low risk of DCS.

The use of Doppler to monitor the central venous circulation, however, shows this approach still frequently results in bubble formation. Other more recent decompression theories have relied on so-called “bubble models,” which focus on prevention of such bubble formation. To do this, the algorithms limit supersaturation more aggressively and typically result in the imposition of deeper decompression stops. These have been used quite successfully for many years by technical divers for deep diving.

In recent years, the utilization of a deep stop by recreational scuba divers at depths less than 130 fsw (41 m) also has been proposed. This is now appearing in dive computers using “bubble models” such as Varying Permeability or the Reduced Gradient Bubble Model or the Half the Depth Model. Some training agencies have also incorporated deep stops into their training regimens.

However, there is debate as to when to stop, for how long and how often in regard to whether such a deep stop does limit bubble growth or ameliorate the risk of DCS.

This workshop has brought together the most active international individuals with practical human data, animal research and theoretical concepts to help clarify the role of “deep stops” in contemporary recreational scuba and technical diving, and to point out what we know as well as indicate future research needs.

ABSTRACTS

EARLY OBSERVATIONS ON THE EFFECT OF “DEEP” DECOMPRESSION UPON DOPPLER ULTRASONIC BUBBLE SIGNALS FOLLOWING 210/50 AND 170/30 DIVES

Tom Neuman

The problem of whether “deep” decompression stops adds significantly to the safety of a given decompression profile is a difficult one. Ultimately of course, this is a question that must be addressed empirically. However, any studies involving decompression sickness are fraught with a variety of problems. Control groups, blinding and the selection of an appropriate endpoint are some of the difficulties confronting any group attempting to address these issues.

In the middle 1970s the U.S. Navy had specific operational objectives that required a number of dry chamber dives to 210 FSW for 50 minutes on air and to 132 FSW for 30 minutes, using a normoxic nitrogen/oxygen mixture. At that time, we were able to make some unique observations concerning the decompression profiles used for those dives.

The original intent of these experiments was to validate the reliability of Doppler ultrasonic bubble detection methods; however, we were also able to make observations relating to the effect of “deeper” decompression stops upon bubble scores. For these dive profiles there was a significant
reduction in bubble score associated with a short “deeper” stop, independent of overall decompression time. It is, however, premature for these results to be extrapolated across the continuum of diving exposures or across the range of decompression algorithms that currently are used to calculate decompression tables. These results may be solely a consequence of the use of the model that generated the decompression profiles used in these dives.

LANL DEEP STOP DATA BANK AND DUAL PHASE BUBBLE MODEL FOR PROFILE ANALYSIS AND RISK

Bruce Wienke, Timothy R. O’Leary

Linking model and data, we detail the LANL reduced gradient bubble model (rGBM), dynamical principles and correlation with data in the LANL Data Bank. Table, profile and meter risks are obtained from likelihood analysis and quoted for air, nitrox, helitrox no-decompression time limits, repetitive dive tables and selected mixed-gas and repetitive profiles. Applications include the Bennett and Marroni 2.5-minute recreational deep stop, early Duke experiments with helium and deep air switches, NEDU deep stop tests, French Navy deep stop profiles, EXPLORER decompression meter algorithm, NAUI Tables, University of Wisconsin Seafood Diver Tables, comparative NAUI, PADI, Oceanic NDLs and repetitive dives, comparative nitrogen and helium mixed-gas risks, USS Perry deep rebreather (RB) exploration dive, world record open-circuit (OC) dive, and WKPP extreme cave exploration profiles.

The algorithm enjoys extensive and utilitarian application in mixed-gas diving, both in recreational and technical sectors, and forms the bases for released tables and decompression meters used by scientific, commercial and research divers. The LANL Data Bank is described, and the methods used to deduce risk are detailed. Risk functions for dissolved gas and bubbles are summarized. Parameters that can be used to estimate profile risk are tallied. To fit data, a modified Levenberg-Marquardt routine is employed with $L_2$ error norm. Appendices sketch numerical methods, and list reports from field testing for (real) mixed-gas diving. A Monte Carlo sampling scheme for fast numerical analysis of the data is also useful, as coupled variance reduction technique and additional check on the canonical approach to estimating risk. Supercomputing resources are used. This work attempts a (needed) correlation between global mixed-gas diving, specific (bubble) model, and (deep stop) data. The whole issue of deep stops and staging is one of timing, with questions of time and depth at deep stops possibly addressed optimally within consistent model and ranging data frameworks.

TECHNICAL DIVING OVERVIEW

Simon J. Mitchell

There is no universally agreed definition of technical diving, but it is characterized by decompression diving, the utilization of gases other than air, and equipment configurations other than single-cylinder open-circuit scuba in order to visit deeper depths or extend underwater duration or both. The adoption of these techniques, which in the past have been more commonly associated with occupational or military diving, has been driven largely by wreck and cave divers wishing to explore deeper wrecks and deeper and/or longer caves respectively.

The operating depth and duration of single-cylinder scuba air is limited by the small amount of gas that is carried, and the unfavorable characteristics of air as a deep diving gas, including its high density and high nitrogen and oxygen fractions, which at sufficient depth predispose to narcosis and oxygen toxicity respectively. Technical divers utilize complex multiple open-circuit cylinder configurations or rebreather devices to extend duration. Helium is lighter and non-narcotic, and is substituted partly or wholly for nitrogen in deep diving gases. The oxygen fraction is also tailored to the depth being visited. Multiple gas mixes with progressively increasing oxygen fractions are frequently utilized during decompression to optimize inert gas elimination.

Using these methods, recreational technical diving exponents have extended “bounce dive” depths and durations well beyond limits adopted by the more conservative occupational and
military groups. Technical divers have visited depths exceeding 300m and open-ocean wrecks as deep as 200m. Caves as deep as 280m have been dived, and a cave system 90m deep and 11 kilometers long was recently traversed over seven hours of bottom time, followed by 15 hours of decompression. Although these examples represent current boundaries established by cutting-edge exponents, there is a vastly greater number of participants who are “routinely” diving to depths between 60 and 100m.

Although there are no definitive figures, limited data and anecdotes shared by diving physicians and divers seem to indicate that technical diving is hazardous. There is reason to believe that fatality rates are much higher than for scuba air divers, particularly among rebreather users. Moreover, there are numerous episodes of “unexpected” decompression sickness associated with these dives, and this has given impetus to improvement of decompression algorithms for deep “bounce” diving.

Reference

A DECADE OF DEEP STOP TRAINING WITH THE REDUCED GRADIENT BUBBLE MODEL

Timothy O’Leary

Linking a dual phase decompression model to mixed-gas training operations, I will recount and detail protocols and diver training data from 1998 to the present. The diver data and training protocols described will be selected helium based dives and variable mixes with both open-circuit and closed-circuit in the 150 fsw to 300 fsw zones, along with repetitive profiles as used in the technical diver programs. The algorithm and published groupless, no-fuss NAUI mixed-gas decompression tables with repetitive dive protocols have seen extensive application in the technical diving sectors from a wide array of mixed gas instructors and divers in a variety of countries and is currently extending to dive meters in the training field.

A PRACTICAL LOOK AT DECOMPRESSION SURVIVAL ON DIVES DEEPER THAN 100 METER AND USE OF INTUITIVE DECOMPRESSIONS

Tom Mount

This presentation will address the practical decompression procedures used by some “technical divers” and how they evolved. It will describe use of a multimodel approach such as advocated by J.P. Impert and will reflect this to combinations with VPM-RGBM and gradient factors.

The process of decompression may be compared to a giant circle, where practice produces consequence, learning, and management of technique interact, creating the foundation of “intuitive decompression.”

We will also address some modifications to decompression models used by deep trimix divers. The overall process is to use a model but use it in a manner stated by R.W. Hamilton as a “what works-works” approach. Within this, most of us favor deep stops or very slow ascents, which contradicts the thoughts of many researchers and agrees with others. Yet this process is hard to define. We will endeavour to do so.

The paper will also address what the author believes to be the most ideal decompression strategy when using a CCR. This leads us away from the concept of getting off helium. It maintains the same gas for the duration of CCR decompression as the inert gas volume mixed is reduced due to the constant PO2.

At the end of the presentation an understanding of the apparent “what works decompression process” should be evident. Although subjective, as was the “subjective” feeling of nitrox dives vs. air dives long ago, it does seem to provide better post-dive health.
WORLD RECORD CAVE DIVE, DEEP STOPS WITHIN THE WOODVILLE KARST PLAIN PROJECT

Jarrod Jablonski

During the late 1980s a nonprofit exploration group known as the Woodville Karst Plain Project (WKPP) began steadily expanding the length of their deep cave immersions. By the mid 1990s these divers were experiencing bottom times of about six hours at a depth of 300 feet. During this period and in the years to come, the team would focus considerable energy toward safely reducing total decompression time.

This process was primarily a response to the sense that conventional Bühlmann algorithms were not structured with the most favorable arrangement of decompression stop times. Over approximately 7,500 dives spanning nearly 20 years, these divers aggressively experimented with a range of decompression profiles, working to support immersions that have reached nearly 30 hours. The divers utilize a similar decompression time for all dives in excess of six hours’ bottom time, where dives average approximately 280 feet. To date, 10 hours is the longest bottom time obtained, resulting in a decompression of 17 hours.

THE APPLICATION OF ‘DEEP STOPS’ IN ANDI’S TECHNICAL DIVER TRAINING AND EXPEDITIONS

Ed Betts

Introduction

The validity and efficacy of the RGBM is an unresolved topic. To some, it is controversial. After much discussion and investigation, ANDI performed its own trials on two expeditions and recorded all data for our own use. Although no specific case studies and conclusions were offered by others, we decided to conduct our own trials. ANDI has since implemented the use of this methodology by means of the ANDI-Gap software program.

Data collection

The original “field tests” consisted of four series of dives that were performed by groups consisting of 12 males and two females with age variations of 23 to 61. The dive planning was completed using the Gap software in RGBM mode and the individual tables were printed including bailout options. Depths ranged from 24m to 156m, with bottom times at the deepest depths of 20 to 29 minutes. Several dives made at the shallowest depths exceeded 150 minutes of BT. In 2003, ANDI has trained a team of commercial divers using surface-supplied equipment from pre-mixed gas racks. ANDI methodology was used on their project, the Rio-Andirion Bridge, Europe’s largest. Several thousand dives were completed.

Current usage

ANDI has four-plus years of use throughout our network. With training conducted in more than 60 countries by our trainers and instructors in many different languages we require that ANDI-Gap is the only algorithm permitted. We are not scientists, mathematicians or research physiologists. We are engineers, instructors and working divers who are often the test subjects for our own methods. We currently offer students free use of the software and require instructors to purchase it. We have recorded more than 2,500 free downloads of the trial program from our website.

Results of the dives

Regarding the efficacy of the deep stop method we can only say “what works ... works.” I am not the expert here. My position is that decompression is still an art and not yet a science. We are all still learning and especially so at the more extreme exposures. Despite conflicting comments from some colleagues, our experience using this method is as follows.

No incidences of DCI occurred during the expeditions, and all divers reported no sub-clinical symptoms. We have not had one single case ever reported to ANDI of DCI during any training program, nor, any reports of DCI experienced by ANDI-certified divers using this method.
Conclusions
Until contrary data can be offered, it is ANDI’s decision to continue to recommend the use of this diving methodology and training procedure.

DEEP STOPS: AWARENESS AND CURRENT PRACTICE IN THE TECHNICAL DIVING COMMUNITY

Drew Richardson, Karl Shreeves

The practice of making deep stops began with tech diving more than 10 years ago, with Richard Pyle, Ph.D., primarily credited with raising the question and creating awareness. Decompression models such as the Reduced Gradient Bubble Model (RGBM) and others have also come to the fore with recommendations that it is beneficial to begin decompressing deeper than mandated by conventional Haldanian-type models.

Anecdotally, the approach to deep stops in tech diving includes using deep models like RGBM, adding deep stops to conventional Haldanian-type model predictions and not making deep stops, but there have been no data that reveal which practices are present to what extent. The authors initiated a survey of individuals certified as technical and technical trimix divers, instructors and instructor trainers to determine an indication of the present state of deep stop awareness and practice in the technical diving community. This paper discusses the findings from the survey.

Joseph Dituri, Kirk Parsley, Harry T. Whelan

The opinions expressed herein are possessed solely by the authors and do not necessarily reflect those of any organization with which the authors may be affiliated.

Background
Conventional commercial and U.S. Navy deep “bounce” diving is generally limited to 300 feet and requires the support of large surface platforms and a minimum of 13 divers. The breathing media generally used is HeO₂, and the diving apparatus is the MK-21 or Superlight 17 style hard hats.

This method requires recompression chambers, storage racks and equipment in excess of 50,000 lbs. on site.

Methods
Using trimix rebreathers and portable recompression chambers in lieu of the above mentioned method, deeper dives are being achieved with greater safety.

Results
Incorporating new knowledge of decompression tables and algorithms would allow the depth limit for “bounce” dives to be increased to as much as 600 feet. The use of constant partial pressure of oxygen rebreathers and dive computers can increase safety and decrease required decompression time. The incorporation of inflatable chambers and rebreathers would also reduce the required footprint and weight of a team as well as vessel required.

Conclusion
With this proposed – less expensive – system, a team of 12 divers can deploy more rapidly, with 80% less equipment burden, while greatly exceeding the current diving model’s capabilities.

DEEP STOPS AND THEIR EFFICACY IN DECOMPRESSION: U.S. NAVY RESEARCH

Wayne A. Gerth, David J. Doolette, Keith A. Gault

Introduction
Classical decompression algorithms limit hypothetical tissue gas contents and prescribe decompressions that advance rapidly to shallow stops where most of the total stop time (TST) is scheduled. Recent bubble-based algorithms limit calculated bubble profusion and size and prescribe decompressions with TST skewed toward deeper stops. Navy Experimental Diving Unit (NEDU) has completed a controlled comparative study of these approaches.

Methods
Divers wearing swimsuits and T-shirts, breathing surface-supplied air via full face masks, and immersed in 86 °F water in the NEDU Ocean Simulation Facility wetpot were compressed at 60 fsw/minute to 170 fsw. They performed 115-watt cycle ergometer work during
an ensuing 27.2 minutes at bottom and were de-
compressed at 30 fsw/minute with stops prescribed
by one of two schedules, each with 174 min TST.
Schedule 1, with stops at (fsw/minute) 40/9,
30/20, 20/52 and 10/93, was prescribed by the
man-tested, deterministic gas content, VVAL18
Thalmann Algorithm. Schedule 2, with stops at
70/12, 60/17, 50/15, 40/18, 30/23, 20/17 and 10/72,
was the optimum distribution of TST according to
the man-dive calibrated, probabilistic BVM(3)
bubble model.

Decompression sickness (DCS) incidence with
these schedules was compared under the sequential
stopping rules of reject-high if DCS risk > 7% or
reject-low if DCS risk < 3% with 95% confidence.

Results
The trial was terminated after midpoint interim
analysis. Neither schedule was rejected, but DCS
incidence in Schedule 2 (deep stops, 11 DCS/198
dives) was significantly higher than in Schedule
1 (3/192, p=0.030, one-sided Fisher Exact).

On review, one Schedule 2 DCS was excluded,
but the result remained significant (p=0.047). Most
DCS was mild, late onset, Type I, but two Schedule 2
cases involved rapidly progressing CNS
manifestations.

Conclusions
The deep stops schedule had a greater risk of DCS
than the matched conventional schedule. Slower
gas washout or continued gas uptake offset
benefits of reduced bubble growth at deep stops.

DEEP STOPS DURING DECOMPRESSION
FROM 50 TO 100 MSW DIDN’T REDUCE
BUBBLE FORMATION IN MAN

Jean-Eric Blatteau, Michel Hugon, Bernard Gardette

Background
The French Navy uses the MN90 decompression
table for air dives as deep as 60 msw and the MN78
decompression table for trimix dives (60-80 msw).
The resulting incidence of decompression sickness
(DCS) for deep air dives (45-60msw) is one case
per 3,000 dives (with 89% of neurologic DCS). We
hypothesized that introduction of deep stops could
reduce fast tissue bubble formation and neurological
DCS risk in deep air diving. We also expected that
adding deep stops could reduce bubble formation and
decompression stress for trimix diving (80-100msw).

Methods
We incorporated deep stops (DS) into a series of six
experimental ascent profiles (EAPs) developed with
decompression software built on a Haldanian model.
Deep stops for air dives (EAP 1-4) were intro-
duced at about one-half the absolute depth and
about one-third for trimix dives (EAP 5 & 6).
EAPs were tested in the wet compartment of a
hyperbaric chamber. For EAPs 1-5, eight subjects
dove to 50, 60 or 80 msw and ascended according
to the French navy standard tables or an EAP.
Precordial bubbles were monitored with pulsed
Doppler at 30-minute intervals after surfacing.
The signal of bubbles was graded according to the
Spencer scale before being converted into Kissing
Integrated Severity Score (KISS). EAP1: 60 msw /20
minutes, first DS at 27 msw, decompression times
(DT) 59 minutes vs. 48 minutes (MN90); EAP2: 60
msw/20 minutes, first DS at 27 msw, (pure O2
6-0 msw) DT 42 minutes vs. 48 minutes (MN90); EAP3:
repetitive dive to 50 msw/15 minutes with a three-
hour surface interval, first DS at 18 msw, DT 31
minutes vs. 46 minutes (MN90); EAP4: 60 msw /15
minutes, only one DS of two minutes at 25 msw, DT
31 minutes vs. 29 minutes (MN90); EAP5: 80 msw/15
minutes with trimix O218%-He41%-N241% (80-12
msw) and pure O2 (12-0msw), first DS at 24 msw, DT
74 minutes vs. 66 minutes (MN78). For EAP6, 12
subjects dove to 100 msw and ascended only
according to the EAP, which was not compared
to another table. EAP6: 100 msw/15 minutes
with trimix O215%-He45%-N240% (100-30 msw),
nitrox 40% (30-6msw) and pure O2 (6-0 msw),
first DS at 33msw, DT 121 minutes.

Results
We found no significant differences in bubble scores
KISS between standard tables (MN90 or MN78) and
EAPs 1,2,4 or 5. Nevertheless EAP3 produced an
increased level of prolonged bubbling for all eight
divers [mean KISS: 20 (EAP3) vs. 8.6 (MN90),
p=0.03], as well an important tiredness for five divers
that improved with one hour of normobaric O2
breathing. One diver suffered joint pain DCS after
EAP2 while exhibiting Spencer grade 3 bubbles at rest
60 minutes after surfacing. His symptoms improved with hyperbaric oxygen, but MRI showed a bone infarction of humeral diaphysis. EAP6 produced Spencer grade 4 bubbles 60 minutes after surfacing for two divers, without symptoms of DCS; fortunately, bubbling was reduced after 30 minutes of normobaric O₂ breathing.

Conclusion
The utility of deep stops in human decompression has yet to be demonstrated for deep air dives as deep as 60 msw and trimix dives as deep as 100 msw with mixed gas including N₂ ≥ 40%.

**BUBBLE DETECTION AND DCS RELEVANCE**

*Neal Pollock*

Decompression studies traditionally rely upon symptoms of decompression sickness (DCS) as an endpoint. An observation made in the early 1960s that Doppler ultrasound could detect decompression-induced bubbles moving in the bloodstream expanded the possibilities for evaluation. The development of a series of semi-quantitative grading scales followed.

The 0-IV Spencer scale remains the most popular (0 = no bubble signals; I = occasional bubble signal; great majority of cardiac cycles signal-free; II = many but less than half of the cardiac cycles contain bubble signals; III = all cardiac cycles contain bubble signals, but not obscuring signals of cardiac motion; and IV = bubble signals sounding continuously throughout systole and diastole and obscuring normal cardiac signals).

The Kisman-Masurel scale is more sophisticated, with signals separately scored on the frequency, percentage/duration and amplitude of bubble activity before these parameter scores are combined to produce a single 0-IV grade. Kisman-Masurel scores can easily be converted to Spencer grades, but the reverse conversion is not possible.

Ultrasonic monitoring can be used to provide a secondary measure of decompression stress if symptoms are to remain an endpoint. Alternatively, ultrasonic monitoring may be used as a primary endpoint measure of decompression stress if the endpoint of symptoms is not appropriate for ethical or practical reasons. For the latter case, in particular, it is important to consider the limitations of bubble data.

Most critical is that the role that bubbles play in the development of symptomatic DCS is not clear. Part of the problem is that current technology makes it easy to study only intravascular bubbles. We know very little about the development of bubbles in extravascular tissues. Intravascular bubbles are associated with DCS, but DCS can develop in the absence of observed bubbles. Higher intravascular bubble grades (Spencer III or IV) are more strongly correlated with DCS than lower grades, but still at modest levels.

A recent study of 1,726 air dives and 1,508 heliox dives showed extremely poor positive predictive value for Spencer grade III-IV intravascular bubbles. The greatest strength of the bubble data was in the negative predictive value – the absence of DCS symptoms – associated with Spencer grade 0-II bubble scores.

There are additional practical challenges in interpreting ultrasonic data. The marked variance in sampling protocols (inter-measure interval and total sampling duration) may affect the validity of the data. The presence of intravascular bubbles has been reported to peak at 60 minutes post-dive, but this can vary as a function of the dive profile and breathing gas.

Differences in test procedures may also affect the comparability. This can include instrumentation, monitoring site selection, case sampling (rest or rest and various movement cases), and recording/review procedures.

Variability in technician training and experience are also potentially problematic, more so when scoring sessions are not recorded and confirmed.

Finally, self-selection within subject pools can be an issue, notably for more extreme exposures. It is possible that such groups will be disproportionately populated with bubble-resistant individuals, making it difficult to extrapolate the results from such groups to the wider population.

The above points are made not to discredit ultrasonic bubble monitoring but to remind the community that protocols should be carefully thought out and that the results of such monitoring must be critically and conservatively evaluated.
THE OPTIMAL PATH

Richard D. Vann, L.E. Howle, R.G. Dunford, Petar Denoble

The optimal path is the decompression profile that has the lowest possible probability of decompression sickness (DCS) for a given depth, bottom time and ascent time. The optimal path also applies to venous gas emboli (VGE). Understanding optimal paths for VGE will be important if arterIALIZED VGE are proven responsible for cerebral DCS.

Optimal paths were estimated using probabilistic decompression models calibrated to 841 nitrogen-oxygen dive trials that were conducted in 1985 at the U.S. Navy Experimental Diving Unit. Doppler VGE data were also available for these trials. To model VGE probabilistically, we defined a binary variable called “High Bubble Grade (HBG)” with a value of 0 for Spencer Grades of 0-2 and a value of 1 for Grades 3-4.

To validate the model predictions, we estimated the DCS and HBG probabilities for the deep stops trials conducted by the Navy for 30-minute dives to 170 fsw. The DCS model predicted the observed DCS incidences relatively well, but the HBG model was unsatisfactory.

The first decompression stop for the optimal DCS profile was deeper than for the U.S. Navy schedule used from 1957-2008.

THE EFFECT OF DEEPER STOPS ON BUBBLE FORMATION IS DEPENDENT ON LENGTH OF BOTTOM TIME

Christian R. Gutvik, D. Glavas, A. Møllerløkken, Z. Dujic, Alf O. Brubakk

Background
Deep decompression stops compared to more conventional shallower stops have recently been introduced. Most findings and theoretical work on excess gas phase / bubble models suggest an apparent advantage of using deeper stops. However, some reports indicate that the incidence or risk of decompression sickness may actually increase following such procedures.

Materials and Methods
As a part of the validation of the Copernicus decompression model, a series of experimental dives were performed on recreational divers in Split, Croatia. A total of 11 dive procedures with seven to eight divers in each group were tested in water. The protocol included two series of deep/short dives (54 msw / 20 minutes and 45 msw / 16 minutes) and two series of shallow/long dives (24 msw / 70 minutes and 24 msw / 40 minutes). The four dive protocols followed two to three different decompression procedures with both deep and shallow stop regimes. The dives were evaluated using ultrasonic bubble detection.

At UHMS 2007 we presented a hypothesis based on animal experiments and a theoretical concept of stabilized bubble nuclei that the benefit of deep stops primarily applies for long bottom times. The present human data were analyzed to test this hypothesis.

Results
On the 24 msw / 70 minute dive, the experimental deep stop procedure seems to produce fewer bubbles than the Bühlmann shallow stop procedure. On the 45 msw / 16 minute dive, the VPM deep stop procedure gave more bubbles than the experimental shallow stop procedure. Simulation results from the Copernicus model with the implemented nuclei dynamics give the same results.

Conclusions
Although not statistically significant, the results point in the same direction as the previously presented hypothesis and fit well with the Copernicus bubble model. Our suggestion is that deep stops are primarily recommended on longer dives; however, more studies specifically designed to test this are advised.

INTERNATIONAL DAN DEEP STOP RESEARCH FOR RECREATIONAL DIVING

Peter B. Bennett

The predominant signs and symptoms of decompression sickness (DCS) in recreational divers are pain (23.9%), numbness (22.0%) and weakness (7.2%) and are of a neurological nature indicative
of spinal cord involvement rather than joint pain. The incidence of DCS has changed little over the past decades (0.04-0.07%). Review of the history of ascent profiles shows that the 1906 Haldane 2:1 staged ascent was far superior to the Hill linear ascent. Yet today we are still making linear ascents, plus only a shallow stop at three to five meters for three to five minutes.

Since the U.K. and U.S. Navy divers experienced mostly joint pain DCS, they considered the problem was in the joints with their poor blood supply, which saturated or took up gas very slowly. Haldane’s (1906) model of the body had five compartments (or exponentials) representing very full blood supply as in the brain and spinal cord at five minutes, 10 minutes, 20 minutes, with 40 minutes, 80 minutes and 120 minutes (representing poorer blood supply like the joints).

But the recreational diver’s problem is in the fast tissue spinal cord, with 12.5 minutes half time, not the slow joint 120 minutes. It is proposed, therefore, that we now ascend far too rapidly and cause bubbles to form deep.

Working with Italian divers in the Mediterranean and an IDAN team of physicians and scientists, we hypothesized that introduction of a deep stop at half the depth would reduce the deep bubble formation and decompression risk in the spinal cord. A total of 181 dives were made to 25 m (82 fsw) by 22 volunteers with eight different ascent protocols. Ascents of three, 10 or 18 m/minute (10, 33 or 60 fsw/minute) were combined with no stops, or a shallow stop at 6 m (20 fsw), or a deep stop at 15 m (50 fsw) and a shallow stop at 6 m (20 fsw).

Bubbles were detected by Doppler over the heart after reaching the surface. These experiments indeed showed the highest gas loads were in the fast compartments (five and 10 minutes), not the slow. More importantly, the lowest bubble scores were with an ascent rate of 10 m/minute (33 fsw/minute), not three m/minute (10 fsw/minute).

Stops were best for five minutes at 15 m (50 fsw) and 6 m (20 fsw). More recent additional research has shown, in fact, that the best stop time for the deep stop is two and one-half minutes at half the depth. The one-minute stop recommended by some training agencies is too short. We therefore recommend a deep stop at half the depth of two and one-half minutes followed by the customary 6 m (20 fsw) for three to five minutes. While the direct correlation with signs and symptoms of DCS has not yet been made, this still does constitute a definite decrease in decompression stress.

**THE USE OF DEEP STOPS IN RECREATIONAL DIVING:**

**DAN EUROPE AND IDAN – OVERVIEW OF EARLIER STUDIES AND RECENT OBSERVATIONS**

Alessandro Marroni, Frans J. Cronjé

Decompression illness (DCI) affects some 1,500 divers every year. Although DCI is relatively rare, two-thirds of these divers develop neurological manifestations. To study the factors associated with DCI, and to make diving even safer for recreational divers, DAN Europe, in collaboration with International DAN, performed a series of experiments since 1995. This presentation summarizes the highlights of these investigations.

Between 1995 and 1999, DAN Europe conducted an observational study and collected and analyzed 2,105 fully monitored, unrestricted recreational dives. The dives ranged from 5 to 65 meters sea water (MSW) and involved 575 volunteer research divers. The largest number of dives – 33.15% – were made in the 20- to 30-meter depth range. All the divers were Doppler-monitored at fixed intervals post-dive.

The presence of venous gas emboli (VGE) was graded as LBG (low bubble grades – occasional bubbles), HBG (high bubble grades – frequent to continuous bubbles); and HBG+ (very high bubble grades – continuous bubble showers). VGE were detected in 37.4% of the monitored dives; LBG were observed in 25.4%; HBG in 12%; and HBG+ in 2.4% of the dives. Only 15% of the repetitive dives were bubble-free; LBG were detected in 18% of the repetitive dives and HBG/HBG+ were recorded in 67% of the repetitive dives.

Careful analysis of these dives suggested that post-dive high bubble grades were directly related to three key factors (TKF): gas-loading of fast to medium half time (HT) tissue compartments (TC) as per Bühlmann ZH-L8 ADT model; computed venous partial pressure of nitrogen (PvenN₂) in excess of 1,100 mbar; and leading TC nitrogen
Based on these results, a project was started to confirm the validity of the TKFs in controlling bubble grades. Three experimental square dive profiles were selected:

1. a single dive to 20 m for 60 minutes;
2. a single dive to 40 m for 10 minutes; and
3. a series of three repetitive dives to 30 m for 16 minutes with 75-minute surface intervals.

The dives were made according to the original ZH-L8 ADT model and repeated with a modified algorithm designed to stay within the TKF limits. This implied a gradual reduction of the leading TC M-value, inversely proportional to the TC HT (proportional M-value reduction concept – PMrC), extended to include the 80 min HT TC and reaching correction factor 1 for the 160-minute HT TC (i.e., no change).

To achieve these partial pressures and gradients, extra deep stops had to be introduced during the ascent. These drastically reduced post-dive precordial Doppler-detected venous gas emboli (PPDDVGE) in a sample of 14 volunteer divers performing 210 dives and serving as their own controls. The study showed that the pressure gradient (i.e., Delta-P) imposed on the leading TC, irrespective of the rate of ascent, appeared to be the critical factor for bubble production in this series of experimental dives.

Given the experience with the extra deep stops, and in order to establish practical recommendations relevant to typical recreational divers, the next phase of the study considered the effect of adding deep stops of varying durations at half-the-depth of the dive – half depth deep stops (HDDS). These were evaluated during experimental repetitive diving to 25 MSW. The results are presented elsewhere at this workshop.

The final part of the experiment examined the effect of HDDS, in addition to the standard “Safety Stop,” during single and repetitive recreational dives, from 18 to 40 MSW. Eight volunteer divers performed 24 different no-decompression dives between 18 and 40 MSW, with or without HDDS. Six of the profiles involved repetitive dives, designed according to the current USN Diving Tables, with three-hour 30-minute surface intervals. The depth patterns were chosen to reflect the normal habits of most recreational divers (18+18, 21+21, 25+25, 27+21, 30+2, 40+24 MSW respectively).

The introduction of an HDDS generally reduced PPDDVGE, with an overall decrease of high bubble grades compared to the same dives without HDDS. The data suggest that the inclusion of an HDDS on dives between 25 and 30 MSW, with bottom times of 25 minutes or less (i.e., the typical dive profiles performed by recreational divers) reduces decompression stress as measured by PPDDVGE.

The value of HDDS in reducing PPDDVGE was not as evident for shallower (18-21 MSW) and deeper dives (40 MSW), when brought to the limit of the respective no-D bottom time according to USN Dive Tables, and showed conflicting results. Further investigation is now being planned to unravel the apparent ambiguity of HDDS at these depths.

Proceedings for the workshop are available for $50 plus S&H.

To order, go to the UHMS publication website:
UHMS member Dr. Ivan W. Brown Jr., a pioneering heart surgeon, died on Nov. 20 in Lakeland, Fla., of congestive heart failure. He was 94.

A native of Newfane, N.Y., Dr. Brown founded Lakeland Regional Medical Center’s open-heart surgery program 40 years ago. He came to Lakeland in 1968 from Duke University, in Durham, N.C., where he was a professor and researcher. While at Duke, Brown began a blood banking program and invented the Brown Harrison Heat Exchanger, a crucial component of early heart-lung machines.

“He was a very unique individual who had a remarkable breadth of knowledge in many areas beyond medicine,” said cardiologist Dr. Kevin Browne Jr., a colleague. “When I think of Ivan, I always think of the Renaissance man because he was one . . . He had a fantastic mind and a fantastic memory.”

Brown was born July 6, 1915, to Ivan and Agnes Brown. He was an undergraduate at the University of Rochester and at Duke, graduating in 1940 from Duke University Medical School. Soon afterward he served in World War II as a surgeon with the 65th General Hospital unit in England and was reportedly the youngest physician who served with that Duke-affiliated Army medical unit. The 65th was a designated specialty center for neurosurgery, thoracic and plastic surgeries, burns and hand injuries.

In addition to operating on soldiers’ head wounds, removing shrapnel and using metal plates on injured skulls, Brown conducted open-heart surgeries during the war. According to his son, Ivan Brown III, he also removed flak from inside the hearts of wounded soldiers.

Returning to Duke after the war, Brown carried on his lifelong passion for research. He became Duke’s James P. Duke distinguished professor of surgery in 1960. During the Cuban Missile Crisis in 1962, he was a consultant on using blood transfusions to treat radiation sickness.

In 1964, he was an advisor to the national Sea Lab and Man Under the Sea program. Brown joined the executive committee of the National Academy of Sciences in 1965, where he was instrumental in researching hyperbaric medicine. A prolific writer, he contributed articles to numerous medical journals and continued writing and lecturing until his death.

Brown was preceded in death by his wife, Madeleine Davis, and is survived by a brother, Keirn Brown, of Boca Raton; two daughters, Sandy Brown Wyeth of Los Angeles and Diane Fletcher of Newcastle, Australia; his son, Ivan Brown III, Greenville, N.C.; three grandchildren and two great-grandchildren.

Source
Taken from the story by Robin Williams Adams in the Ledger, Lakeland, Fla.

CApT. MAINO dES GrANGES

UHMS member Capt. Maino des Granges, former Officer in Charge of the Navy Experimental Diving Unit, passed away August 19, 2009, in San Diego, Calif. He was 91.

Des Granges headed up NEDU in the Washington Navy Yard during the 1950s.

“He was pretty sharp,” said friend and fellow Navy veteran Charles Bishop. “He was made commanding officer (in 1943) and was one of the youngest at the time.”

During the 1950s, Capt. des Granges was the officer in charge of the Navy Experimental Diving Unit in Washington, D.C.

“He developed the Navy’s first set of diving tables . . . Everybody that goes diving uses the diving tables he developed,” Bishop said, referring to the invention of the widely used handheld wheel computer that served as the prototype of many dive computers used today.

After enlisting in the Navy in 1936, des Granges was selected for appointment to the Naval Academy in 1938. He was among those who graduated early because of World War II: He graduated from the academy in December 1941 and was assigned to submarine patrol in January 1942. Only after completing three war patrols was he able to attend submarine school.

“He was energetic and innovative, and everybody who worked with him just loved him,” said Wade Harris, who served under Capt. des Granges when he was division commander and Harris was an executive officer. “He was one of the finest naval officers I ever met. He had a quiet, unassuming personality, but he was outstanding at his job.”

Maino des Granges was born Aug. 2, 1918, in Fullerton, Calif., graduating from Fullerton High School. He married the former Dorothy Beckley in 1942. They settled in San Diego in 1964, when he was stationed as commanding officer of the submarine tender Nereus. He was a member of the Yacht Club and enjoyed golf and bridge. He is survived by his wife, Dorothy, of Point Loma; daughters, Jeanne Vivoli and Anne Chambers of San Diego; son, Paul of Portland, Ore.; four grandchildren and one great-grandchild. He was predeceased by a sister, Pauline des Granges, a former director of the San Diego Parks and Recreation Department.

Friends and relatives said Capt. des Granges was a quiet, unassuming, self-made man who enjoyed coming up with solutions to problems. Growing up in Southern California, Maino des Granges developed a love for scuba diving, inventing and building early on.

As a Navy officer and entrepreneur, he used his skills to create inventions to assist in diving and construction. As a retiree who wanted to improve his golf game, des Granges designed and built a collapsible driving-range cage and putting green for his yard, complete with sand trap.

“He was a fearless, remarkable man’s man,” son-in-law Ned Chambers said. “I think I hit the father-in-law lotto . . . He was the most honorable, phenomenal human being.”

Sources
Hyperbaric Introductory Courses for 2010

These courses have been approved by the UHMS Education Committee as meeting the rigorous standards of a UHMS Designated Introductory Course in Hyperbaric Medicine

Hyperbaric Medicine Team Training

Dates
Jan 11-15; Feb 15-19; Mar 8-12; Apr 12-16; May 3-7; Jun 14-18; Jul 12-16; Aug 16-20; Sept 13-17; Oct 18-22; Nov 15-19; Dec 6-10

Hours
- 42 hours AMA PRA Category 1 Credits™ for physicians
- 40.5 contact hours by the Texas Nurses Association

Contact
- education@hyperbaricmedicine.com /
- +1-210-614-3688
- www.hyperbaricmedicine.com

Introduction to Hyperbaric Medicine

Dates
TBA – see website listed below

Hours
- 40 AMA PRA Category 1 Credits™ for physicians

Contact
- Ms. Patricia Rooney
- +1-914-664-8000 x 6795 or 6535
- LSTcourses@aol.com
- www.LifeSupport-USA.com

Introduction to Hyperbaric Medicine and Wound Care

Dates
May 3-7; Nov 1-5

Hours
- 40 AMA PRA Category 1 Credits™ for physicians

Location
Long Beach Memorial Medical Center, Long Beach, Calif.

Contact
- SHart@memorialcare.org /
- 1-562-933-6950
Introduction to Hyperbaric Medicine and Wound Care Challenges

Dates
TBA – see information listed below

Hours
• 40 AMA PRA Category 1 Credits™ for physicians

Contact
• serena@healingwounds.com /
• +1-814-688-4000
• www.serenagroup.net

Principles of Wound Healing and Hyperbaric Medicine

Dates
Jan 25-30; Mar 15-20; Apr 12-17; May 24-29; Jun 14-19; Jul 12-17; Aug 16-21; Sept 20-25; Oct 18-23; Nov 15-20; Dec 13-18

Hours
• 8.5 hours AMA PRA Category 1 Credits™ for physicians

Contact
• jleighty@nationalhealing.com
• +1-561-213-8847

Hyperbaric Training for Healthcare Providers

Dates
May 17-21; Sept 20-24

Hours
• 41 hours AMA PRA Category 1 Credits™ for physicians
• 41 Category A by NBDHMT

Contact
• nevada@oxyheal.com
• +1-877) 699-8648

Primary Training in Hyperbaric Medicine

Dates
Jan 25-29; Mar 8-12; May 10-14; Jun 21-25; Aug 9-13; Sept 27-Oct 1; Nov 15-19

Hours
• 40 hours, AMA PRA Category 1 Credits™ for physicians
• 40 hours Category A Credits (CHTs and CHRNs)
• 37.5 contact hours, RNs
• 37.5 CRCE hours, RTs

Contact
• registration@baromedical.com
• +1-803-434-7101
www.baromedical.com

UHMS JOINTLY SPONSORED COURSES FOR 2010

Courses listed have been reviewed by the UHMS Education Committee and meet the standards for quality required for UHMS sponsorship of CME credits

Advanced Diving and Hyperbaric Medical Team Training Program with Chamber Operation

Dates
Jan 4-8; Feb 1-5; Mar 8-12; Apr 26-30; May 3-7; Jun 7-11; Jul 5-9; Aug 16-20; Sept 20-24; Oct 4-8; Nov 15-19; Dec 13-17

Hours
• 40 AMA PRA Category 1 Credits™ for physicians
• 40 CEUs for all allied medical personnel

Contact
Dick Rutkowski
• +1-888-451-2551
• www.hyperbaricsinternational.com
The Wound Care Course

Dates
Jan 15-16; Feb 19-20; Mar 12-13; Apr 16-17; May 7-8; Jun 18-19; Jul 16-17; Aug 20-21; Sept 17-18; Oct 22-23; Nov 19-20; Dec 10-11

Hours
• 16 hours AMA PRA Category 1 Credits™ for physicians
• 12.4 contact hours by the Texas Nurses Association

Contact
• education@hyperbaricmedicine.com
• +1-210-614-3688
• www.hyperbaricmedicine.com

Hyperbaric Safety Director Training Course

Dates
Jan 20-23; May 12-15; Sept 22-25

Hours
• 28 hours AMA PRA Category 1 Credits™ for physicians
• 25.5 contact hours by the Texas Nurses Association
• 28 Cat. A credit hours credit hours by the NBDHMT

Contact
• education@hyperbaricmedicine.com
• +1-210-614-3688
• www.hyperbaricmedicine.com

67th DAN Diving and Hyperbaric Medicine Course

Dates
May 1-8

Hours
• 24 hours AMA PRA Category 1 Credits™ for physicians

Contact
• 1-800-446-2671 or +1-919-684-2948

UHMS DIRECTLY SPONSORED COURSES FOR 2010

Courses listed have been reviewed by the UHMS Education Committee and meet the standards for quality required for UHMS sponsorship of CME credits

Medical Assessment of Fitness for Diving

Dates
TBA – see information listed below

Hours
• 36 hours AMA PRA Category 1 Credits™ for physicians

Contact
• uhms@uhms.org
• +1-919-490-5140

NOAA/UHMS/USRF Physicians’ Training Course in Diving Medicine

Date
Aug. 9-20

Location
Seattle, Wash.

Hours
• 82 hours AMA PRA Category 1 Credits™ for physicians

Contact
• uhms@uhms.org
• +1-919-490-5140
OTHER COURSES AND MEETINGS OF INTEREST IN 2010

This list is for information only. The Undersea and Hyperbaric Medical Society does not accredit or sponsor all or some of the meetings/events listed below.

**Wound Symposium SE**

*Dates*
April 9-10

*Location*
Greensboro, N.C.

*Contact*
www.woundse.com

**SPUMS — South Pacific Underwater Medical Society**

*Combined with the 6th ASM of the Asian Hyperbaric and Diving Medical Association*

*Dates*
May 24-28

*Location*
Redang Island, Malaysia

*Contact*
Glen Hawkins
  • glen@hawkeyemedical.com.au

**Hyperbaric Facility Safety Director Course**

*Dates*
July 19-21; Oct 18-20

*Location*
The OxyHeal University

*Contact*
www.oxyhealuniversity.com

**PVHO Acrylic Inspection Course**

*Dates*
July 22; Oct 21

*Location*
The OxyHeal University

*Contact*
www.oxyhealuniversity.com

**Hyperbaric Facility Fire Safety Course**

*Dates*
July 23; Oct 22

*Location*
The OxyHeal University

*Contact*
www.oxyhealuniversity.com

**Expedition Medicine Conference**

*Dates*
September 17-19

*Location*
Washington, D.C.

*Contact*
www.expedmed.org

**EUBS 37th Annual Meeting — 2011**

*Dates*
Aug. 24-27, 2011

*Location*
Gdansk, Poland

*Contact*
• office@eubs2011.org
  • www.EUBS2011.org

Find more information on these meetings on the UHMS website at www.uhms.org.
Hyperbaric oxygen, oxidative stress, NO bioavailability and ulcer oxygenation in diabetic patients
S. Efrati, N. Gall, J. Bergan, G. Fishlev, A. Bass, S. Berman, R. Abu-Hamad
M. Feigenzon, J. Weissgarten ............................................................... 1

Effective patient blinding during hyperbaric trials
D. Clarke ................................................................. 13

Evaluation of decompression tables by Doppler technique in caisson work in the Netherlands
J.H. Breedijk, G.J.G.M. Van der Putten, M. Schrier, W. Sterk ............................. 19

A twelve-year longitudinal study of hearing thresholds among professional divers
M. Skogstad, T. Eriksen, Ø. Skare .......................................................... 25

Cerebral magnetic resonance imaging of compressed-air diver in diving accidents
GK Gao, D. Wu, Y. Yang, T. Yu, J. Xue, XH Wang, JP Jiang ............................... 33

Transcutaneous oximetry in clinical practice:
Consensus statements from an expert panel based on evidence*
* Based upon proceedings from the workshop “Transcutaneous oximetry: art, science and practice,” June 13, 2007

Albuterol metered dose inhaler performance under hyperbaric pressures
H.M. Dale, G.R. Kracke ............................................................... 55

Design of an acoustic telemetry system for rebreathers
S.M. Egi .......................................................... 65
ISSUE 2 – MARCH/APRIL

Effect of nitrogen narcosis on free recall and recognition memory in open water
  
  M. Hobbs, W. Kneller ................................................................. 73

Risk of decompression sickness in extreme human breath-hold diving
  
  J.R. Fitz-Clarke ................................................................. 83

Time course of carbon monoxide transfer factor after breath-hold diving
  
  R. Prediletto, E. Fornai, G. Catapano, C. Carli, E. Garbella, M. Passera
  D. Cialoni, R. Bedini, A. L’Abbate ........................................ 93

A new method of measure of bubble gas volume shows that interleukin-6
injected into rats has no effect on gas embolism
  
  M. Bondi, A. Cavaggioni, A. Gasperetti, A. Rubini ...................... 103

A single exposure to hyperbaric oxygen increases levels of circulating nucleosomes
but does not induce mononuclear cell apoptosis in divers
  
  A. Hoeft, S. Schröder ......................................................... 117

Dual-frequency ultrasonic detection of stationary microbubbles in tissue
  
  P.J. Magari, D.L. Alvarenga, J.C. Buckey .................................. 127

Decision process to assess medical equipment for hyperbaric use
  
  F. Burman, R. Sheffield, K. Posey ........................................ 137

Book review – Naval Forces Under the Sea: The rest of the story
  
  Reviewed by CRD Stephen Jeffs ............................................. 145

ISSUE – 3 MAY/JUNE

Hyperbaric oxygen improves nasal air flow
  
  P. Vera-Cruz, J. Croca and C. Zagalo .................................... 147

Dual effects of hyperbaric oxygen on proliferation and cytotoxic T lymphocyte
activity of rat splenic lymphocytes
  
  W. Liu, J. Zhang, C. Ma, Y. Liu, R. Li, X. Sun, John Zhang, W. Xu ............ 155

Monthly variations in the diagnosis of carbon monoxide exposures in the
emergency department
  
  Matthew Partrick, Frederick Fiesseler, Richard Shih,
  Renee Riggs, Oliver Hung M.D. ............................................ 161

Retinal angiography: Noninvasive, real-time bubble assessment
from the ocular fundus
  
  J. Travis Parsons, Cameron R. Smith Jiepei Zhu, Bruce D. Spiess .......... 169
Abstracts from the 3rd Conference U.S.-Japan Panel on Aerospace, Diving Physiology & Technology and Hyperbaric Medicine (UJNR) .................. 183

Special Invited Abstract: Orthopedic applications of hyperbaric oxygen therapy
   Michael B. Strauss .................................................. 214

Book Review: Hyperbaric Medicine Practice, 3rd Edition
   Reviewed by Enrico Camporesi .................................. 217

Book Review: Physiology and Medicine of Hyperbaric Oxygen Therapy
   Reviewed by Paul E. Cianci ......................................... 219

In Memoriam: Obituaries
   Rudolph Emil Klare, M.D. and Grace Baldwin Doherty, M.D. ............. 221

ISSUE 4 – JULY/AUGUST

The pathway to drive decompression microbubbles from the tissues to the blood and the lymphatic system as a part of this transfer
   J. Hugon, L. Barthelemy, J.C. Rostain, B. Gardette .......................... 223

Estimates of N₂ narcosis and O₂ toxicity during submarine escapes from 600 to 1,000 fsw
   Christopher W. Connor, Massimo Ferrigno .................................. 237

Abstracts from the UHMS 2009 Annual Meeting ................................. 247
   Session A: Diving/Decompression Illness: Theory & Mechanisms .......... 249
   Session B: HBO₂ Therapy Mechanisms ..................................... 261
   Session C: Wound Healing and HBO₂ Treatments ............................ 273
   Session D: Diving and Diver Management ................................... 299
   Session E: HBO₂ Chambers and Equipment ................................ 313
   Session F: Diving Equipment and Decompression Illness Treatment ...... 327
   Author Index ........................................................................ 337

In Memoriam: Obituary – Dr. Robert F. Goad ................................. 341

ISSUE 5 – SEPTEMBER/OCTOBER

It is possible to perform a double-blind hyperbaric session:
A double-blinded randomized trial performed on healthy volunteers
   T. Jansen, C.R. Mortensen, M. Tvede ........................................ 347

Croatian indications for hyperbaric oxygenation:
Doubts of the world projected in a small country
   Nadan M. Petri .............................................................. 353
Behavioral temperature regulation in humans during mild narcosis induced by inhalation of 30% nitrous oxide

DANIEL YOGEV, IGOR B. MEKJAVIC .............................................................. 361

Pulmonary dysanapsis and diving assessments

Lin Min Ong, Michael H. Bennett, Paul S. Thomas ........................................... 375

Functional changes in microcirculation during hyperbaric and normobaric oxygen therapy

Alin Stirban, Stefan Lentrodt, Simona Nandrean, Alexandra Pop, Diethelm Tschoepe, Werner Alfons Scherbaum .................................................. 381

ISSUE 6 – NOVEMBER/DECEMBER

It is possible to perform a double-blind hyperbaric session:
A double-blinded randomized trial performed on healthy volunteers

T. Jansen, C.R. Mortensen, M. Tvede .............................................................. 347

Case report: Treatment of mild traumatic brain injury with hyperbaric oxygen

Colonel James K. Wright, Eddie Zant, Kevin Groom, Robert E. Schlegel,
Kirby Gilliland .................................................................................................. 391

Hyperbaric oxygen treatment of nonacute central retinal artery occlusion

Jeffrey N. Weis ............................................................................................... 401

Letter to the editor: Air embolism’s new scarlet letter

Peter J. Mariani, Norma Cooney ...................................................................... 407

Abstracts: Decompression and the deep stop workshop

June 24-25, 2008 ........................................................................................... 409

In memoriam — Obituaries:

Ivan W. Brown and Maino des Granges ......................................................... 421

Contents of Volume 36

Issues 36-1 through 36-6 ............................................................................... 427

Continuing education

Hyperbaric introductory courses; UHMS jointly sponsored and directly sponsored courses; other courses and meetings .............................................. 423
Overview

- Manuscripts must be submitted in MS WORD via electronic transmittal.
- Only manuscripts in the English language will be considered.
- Address email to: renee@uhms.org.

Please be sure to include:
- correct attributions;
- mailing address;
- telephone numbers; and
- email address of corresponding authors.

Language

The language of the journal is standard American English. Please write in a clear and concise style: Well-written papers have the best chance of acceptance. UHM does not provide translation or writing services; authors who are not fluent in the language should have the manuscript edited before submission by a native speaker of English or professional language editor.

The journal will decline to review manuscripts that are not written clearly enough for an informed reader to follow the line of arguments.

Manuscript guidelines

Membership in the Undersea and Hyperbaric Medical Society (UHMS) is not a prerequisite for publication in the journal. Manuscripts are accepted for publication on the condition that they are contributed solely to this journal. Authors submitting a manuscript do so with the understanding that if it is accepted for publication, copyright for the article is assigned exclusively to the UHMS. On request, permission will be given to quote from papers or to use tables and illustrations in other publications, providing credit is given to the original source.

Acceptance of a manuscript is based on originality and quality of the work as well as the clarity of presentation. All manuscripts will be evaluated for significance, soundness and conformance to journal format by two or more members of the Editorial Board or guest referees. Authors should recommend three qualified persons to act as independent referees for their papers; the Editor-in-Chief welcomes these suggestions but is not obliged to follow such recommendations.

After manuscripts have been accepted, authors are asked to submit the final version of the paper electronically.

Fees

Authors of accepted papers will be assessed a flat publication fee of $250 US dollars. The Editor-in-Chief may consider waiving the fee on a case-by-case basis for undue hardship. Editorial consideration of a paper is in no way related to the payment of page charges. Additional charges may be incurred for color reproduction, where necessary.

Proofs

Proofs are sent to authors to be checked carefully. Necessary changes must be clearly indicated on the galley, with corrections typed in different color text. Proofs must be sent back within the time specified by the managing editor. Authors receive reprint instructions after their papers are in pages.

Treatment of subjects

The UHMS endorses the principles of the Declaration of Helsinki on the treatment of human subjects and the guiding principles in the care and use of animals approved by the Council of the American Physiological Society.

Types of Articles

- Research Reports: Results of experimental, theoretical and clinical investigations on topics important to the understanding of undersea, submarine, and hyperbaric medicine. Short reports that make a substantial scientific contribution as well as extensive studies will be considered.
- Clinical communications and clinical case reports: Observations of an exceptionally revealing nature.
• **Review articles**: May cover scientific and practical subjects and may express the personal opinion of the author.

• **Current issues**: Well-reasoned essays on topics of interest to the journal’s readers; may draw on new or published experimental data and may be controversial in nature.

• **Technical communications**: Descriptions of new methods or equipment; must include data to support contentions.

• **Proceedings of symposiums or workshops**: Usually a group of short communications that have the flavor of reviews.

• **Letters to the editor**: Discussion of scientific papers that have appeared in the journal or scientific issues of interest to the journal’s readers; should include an informative title and be as short as possible. References may be used if necessary, but tables and figures are discouraged.

**Preparation of manuscripts**

The overriding principles are that the composition is correct and unambiguous, clear and concise. The active voice is usually preferable to the passive voice. Parallel construction of groups of like items or concepts aids in comprehension. Figures should be uncomplicated and legible. Abbreviations and acronyms should not be overused, be clearly defined at first appearance in the abstract and in the text and should be avoided in the title.

Specific items of information should appear only once in the manuscript. There should not be verbatim repetition of Copyright © 2008 Undersea and Hyperbaric Medical Society, Inc. in the text of material that appears in a table or figure, duplication of data in graphs and tables, or repetition in Discussion of information that appears in Results.

Authors are encouraged to use papers that have appeared in recent issues of Undersea & Hyperbaric Medicine as models for their manuscript preparation. All accepted manuscripts are subject to final editing in the Editorial Office to improve readability and to conserve space.

Manuscripts must be formatted on 8½ by 11-inch letter-size document with 1-inch margins. Double-spacing facilitates reading by reviewers. References and legends for illustrations must be adjacent to the graphics which can be embedded in the text or their placement clearly marked at the spot in the text where they are to appear.

A cover sheet must accompany the manuscript. It should give the title of the paper, the names and affiliations of the authors, a short title (referred to as “running head”) and the name, address, telephone and fax numbers, as well as the e-mail address of the corresponding author. (Please note: Reviewers for Undersea & Hyperbaric Medicine are blinded to the identity of the author(s); therefore authors’ names should appear only on the cover sheet.)

An accompanying letter must include a statement that all authors have read and approved the manuscript, that the material in the paper has not been published elsewhere (except as an abstract), and that the paper is not currently being considered for publication by another journal.

**Author Responsibility**: If a submission is the work of a group within one center or at multiple centers, that group should select one individual who accepts direct responsibility for the manuscript’s content as well as the agreed sequence of contributing authors. This person will serve as “corresponding author” or “guarantor,” and this designation must be clearly stated on the front page of the manuscript, together with the following contact information (mailing address, email address, telephone number and fax number).


**Title page**: The title page should include:

1. title of no more than 85 characters, including spaces;
2) authors’ names;
3) laboratory or institution of origin, with city and state or country;
4) a running head, not to exceed 50 characters, including spaces;
5) a complete address for mailing proofs; plus
6) a telephone fax number and email address.

Titles should be informative; the implication that a manuscript is one of a series of related papers is discouraged (e.g., “Decompression sickness studies I”).

**Abstracts**: An informative abstract of 200 words or fewer, suitable for abstracting agencies without rewording, should state the purpose of the research, what was done, what was found, and what was concluded. Titles should contain indexable words.
Text: Except in unusual situations, the manuscript should be divided into Introduction, Methods, Results, and Discussion. Long stretches of text should be broken by suitable subheadings but subheadings should not be overused.

Unusual symbols should be avoided. Statistical methods should be described in Methods; information about presentation of statistical material can be found in Bailar J, Mosteller F. Guidelines for statistical reporting in articles for medical journals: amplifications and explanations. Adv Intern Med 1988; 108:268–273.

References: Authors are responsible for supplying complete references and verifying them against the original documents. References must be numbered consecutively in the order in which they first appear in the text, and identified in the text by Arabic numerals in parentheses.

References cited only in tables or legends should be numbered in accordance with a sequence corresponding to the first mention of the table or figure in the text.

List names and initials of all authors when six or less; when seven or more, list only the first three authors and add et al. Citations in the reference list are to be in the form used by the U.S. National Library of Medicine and Index Medicus.


Manuscripts that have been accepted should be cited in the reference list as regular references, with “in press” in place of journal pages. Citations such as “unpublished observations,” “personal communication,” “manuscript in preparation” or “to be published” are not to appear in the reference list, although reference to such a communication, if it exists in written form, may be cited in the text in parentheses. References to government reports should not be cited unless such reports are easily available to all readers.

Equations: Equations should appear in the text in an appropriate type style (italics, bold type, etc.). Authors should carefully distinguish between capital and lowercase letters, Roman and Greek characters and letters and numerals.

Number equations sequentially, in parenthesis on the left edge of the text. All constituent terms should be defined when they initially appear. Authors are responsible for correct formatting of each term in the equation and, because of potential conversion problems, they must be sent using Times New Roman font in a TIFF file. Equations should be considered “camera-ready” when they are submitted.

Tables: Tables should be limited to material needed to make the point of the paper and should be nearly self-explanatory. They should be numbered consecutively in Arabic numerals and bear a short title. Explanatory matter, excluding definitions of abbreviations, should appear in table footnotes. Statistical measures of variation (SD, SE, etc.) should be stated. Tables should be one or two-column width and no more than 8 rows by 8 columns of data with one row for the column headings. Headings should use only horizontal text, no vertical text. Preferred font is Times New Roman.

Acknowledgments: Acknowledgments of persons who aided in the work and of funding agencies, along with any other special considerations about the manuscript, should appear at the end of the text, before the references.

Footnotes: Footnotes to material in the text are discouraged. Footnotes to tables are acceptable and should be identified in sequence by lower-case letters of the alphabet in italic superscript.

Graphics: All graphics, which include anything other than text, should be numbered in Arabic numerals, in sequence as they appear in the text and must conform to one-column (3.125”) width or two-column width (6”). Each is to be accompanied by a suitable legend not exceeding 40 words. Please note that text within graphic should be in the Times New Roman font. Symbols used should be defined in the legend. Diagrams, charts, and other line drawings should be sharp and clear. Upon acceptance of the manuscript, authors must be prepared to submit graphics in TIFF format, 300 dpi or better. Grayscale is preferable to color, both for simplicity and because the author will be assessed a substantial charge for color printing. If color is to be used, however, graphics must be in CMYK, 300 dpi or better. Authors are responsible for visual clarity.
**Line drawings:** Animals must be depicted by line drawings only. It is the journal’s policy not to publish photographs that might be perceived as raising animal welfare concerns. Good line drawings of equipment are usually more effective than photographs. Freehand or typewritten lettering on figures is not acceptable. Lettering must be proportional to the size of the illustration to ensure that it is legible after reduction, and size to fit the journal page should be considered. An internal scale marker (a bar of defined length) should be drawn directly on all micrographs, and the length specified in the legend.

**Units of measure:** The Système Internationale d’Unités (SI units) format will be used to express pressure, depth, length, weight, time, temperature, energy, power, force, and concentration [Standard Practice for Use of the International System of Units (SI) Document E380-89a, American Society for Testing and Materials, Philadelphia, PA 1989].

If the subject matter makes it appropriate to use non-SI units such as fsw, msw, atm or bar, a parenthetical conversion to pascals, kilopascals, or megapascals should accompany the first mention of a pressure value in the abstract and in the text.

Units of fsw and msw should not be used to express partial pressure or when the nature of the subject matter requires precise evaluation of pressure. The proper method for the expression of other units or appreciations may be found in Br Med J 1978; 1:1334–1336 and Aviat Space Environ Med 1984; 55: 93–100. Authors must include after all units a small parenthetical (a) or a small parenthetical (g) to indicate whether units are in absolute or gauge terms.

**Auxiliary publications:** Detailed tables, appendixes, mathematical derivatives, extra figures and other supplementary matter may be deemed too voluminous to be included in the journal article. Such material may be submitted for deposition with the American Society for Information Sciences (ASIS), National Auxiliary Publication Service, at no charge. The information is deposited by the editorial office with the consent of the author, and a footnote will appear in the published article to the effect that photoprint or microfiche copies are available at a moderate cost.

Revised November 2009
SCOPES OF THE JOURNAL

Undersea & Hyperbaric Medicine accepts manuscripts for publication related to the areas of diving research and physiology, hyperbaric medicine and oxygen therapy, submarine medicine, naval medicine and clinical research related to the above topics. Scientific papers must deal with significant and new research in an area related to biological, physical and clinical phenomena related to the above environments.

The following types of papers are published: Original Research (theoretical and experimental); Clinical Communications (which may include case reports if they include control observations of a revealing nature); Current Issues; Technical and Preliminary Notes; Letters to the Editor; and Book Reviews.

Reports of major contributions or symposiums will be considered and may even be published as supplements to regular issues. Authors are referred to “Instructions for Authors” for more details on the categories of papers.

Undersea & Hyperbaric Medicine is abstracted and/or indexed in Chemical Abstract Service, Excerpta Medica, Oceanic Abstracts, Bioscience Information Service of Biological Abstracts, Current Contents, Index Medicus and Current Awareness in Biological Sciences. Undersea & Hyperbaric Medicine is available on 16-, 35- and 105mm microfiche from University Microfilms International, 300 North Zeeb Road, Ann Arbor, MI 48106.

On file in the administrative offices of the Society are two documents pertaining to Institutional Review Board regulations CFR50 and 21CFR56. The UHMS, as publisher of the UHM, acknowledges that all human research requires informed consent and IRB approval in accordance with the laws of the country in which the work was performed. His would include abstracts since they are published in UHM.

The Society endorses the principles embodied in the Declaration of Helsinki and expects that all investigations involving man reported in its journal will have been conducted in conformity with these principles. The Society expects that the Guiding Principles in the Care and Use of Animals will have been observed in all animal experimentation reported in its journal.

Printed on acid free paper since 1993.
2. The nature, the purpose, and the risk of clinical research must be explained to the subject by the doctor.

3a. Clinical research on a human being cannot be undertaken without his free consent after he has been informed; if he is legally incompetent, the consent of the legal guardian should be procured.

3b. The subject of clinical research should be in such a mental, physical, and legal state as to be able to exercise fully his power of choice.

3c. Consent should, as a rule, be obtained in writing. However, the responsibility for clinical research always remains with the research worker; it never falls on the subject even after consent is obtained.

4a. The investigator must respect the right of each individual to safeguard his personal integrity, especially if the subject is in a dependent relationship to the investigator.

4b. At any time during the course of clinical research the subject or his guardian should be free to withdraw permission for research to be continued.

The investigator or the investigating team should discontinue research if in his or their judgment, it may, if continued, be harmful to the individual.

GUIDING PRINCIPLES IN THE CARE AND USE OF ANIMALS

Only animals that are lawfully acquired shall be used in this laboratory, and their retention and use shall be in every case in strict compliance with state and local laws and regulations.

Animals in the laboratory must receive every consideration for their bodily comfort; they must be kindly treated, properly fed, and their surroundings kept in a sanitary condition.

Appropriate anesthetics must be used to eliminate sensibility to pain during operative procedures. Where recovery from anesthetics is necessary during the study, acceptable technique to minimize pain must be followed. Curarizing agents are not anesthetics. Where the study does not require recovery from the anesthesia, the animal must be killed in a humane manner at the conclusion of the observation.

The postoperative care of animals shall be such as to minimize discomfort and pain, and in any case shall be equivalent to accepted practices in schools of Veterinary Medicine.

When animals are used by students for their education or the advancement of science such work shall be under the direct supervision of an experienced teacher or investigator. The rules for the care of such animals must be the same as for animals used for research.
**Statement of Ownership, Management, and Circulation**  
*(Requester Publications Only)*

<table>
<thead>
<tr>
<th>1. Publication Title</th>
<th>2. Publication Number</th>
<th>3. Filing Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undersea and Hyperbaric Medicine</td>
<td>1 0 6 6 2 9 3 6</td>
<td>9/25/2009</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>4. Issue Frequency</th>
<th>5. Number of Issues Published Annually</th>
<th>6. Annual Subscription Price (If any)</th>
</tr>
</thead>
<tbody>
<tr>
<td>bi-monthly</td>
<td>6</td>
<td>$250.00</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>7. Complete Mailing Address of Known Office of Publication (Not printer) (Street, city, county, state, and ZIP+4®)</th>
</tr>
</thead>
<tbody>
<tr>
<td>21 West Colony Place, Suite 280, Durham, NC 27705</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>8. Complete Mailing Address of Headquarters or General Business Office of Publisher (Not printer)</th>
</tr>
</thead>
<tbody>
<tr>
<td>same as above</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>9. Full Names and Complete Mailing Addresses of Publisher, Editor, and Managing Editor (Do not leave blank) (Publisher (Name and complete mailing address))</th>
</tr>
</thead>
<tbody>
<tr>
<td>same as above</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Editor (Name and complete mailing address)</th>
</tr>
</thead>
<tbody>
<tr>
<td>George Mychaskiw, MD</td>
</tr>
<tr>
<td>101 Bridgeview Circle, Ridgeland, MS 39157</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Managing Editor (Name and complete mailing address)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renee Duncan</td>
</tr>
<tr>
<td>21 West Colony Place, Ste. 280, Durham, NC 27705</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>10. Owner (Do not leave blank. If the publication is owned by a corporation, give the name and address of the corporation immediately followed by the names and addresses of all stockholders owning or holding 1 percent or more of the total amount of stock. If not owned by a corporation, give the names and addresses of the individual owners. If owned by a partnership or other unincorporated firm, give its name and address as well as those of each individual owner. If the publication is published by a nonprofit organization, give its name and address.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full Name</td>
</tr>
<tr>
<td>Complete Mailing Address</td>
</tr>
<tr>
<td>Undersea and Hyperbaric Medical Society</td>
</tr>
<tr>
<td>21 West Colony Place, Ste. 280, Durham, NC 27705</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>11. Known Bondholders, Mortgagors, and Other Security Holders Owning or Holding 1 Percent or More of Total Amount of Bonds, Mortgages, or Other Securities. If none, check box</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full Name</td>
</tr>
<tr>
<td>Complete Mailing Address</td>
</tr>
<tr>
<td>□ None</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>12. Tax Status (For completion by nonprofit organizations authorized to mail at nonprofit rates) (Check one)</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ Has Not Changed During Preceding 12 Months</td>
</tr>
<tr>
<td>□ Has Changed During Preceding 12 Months (Publisher must submit explanation of change with this statement)</td>
</tr>
</tbody>
</table>

PS Form 3526-R, September 2007 (Page 1 of 3 (Instructions Page 3))  PSN: 7530-09-000-0655  PRIVACY NOTICE: See our privacy policy on www.usps.com
## Extent and Nature of Circulation

<table>
<thead>
<tr>
<th></th>
<th>Average No. Copies Each Issue During Preceding 12 Months</th>
<th>No. Copies of Single Issue Published Nearest to Filing Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Total Number of Copies (Net press run)</td>
<td>1900</td>
<td>1900</td>
</tr>
<tr>
<td>b. Legitimate Paid and/or Requested Distribution (By Mail and Outside the Mail)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1)</td>
<td>Outside County Paid/Requested Mail Subscriptions stated on PS Form 3541. (Include direct written request from recipient, telemarketing and internet request, paid subscriptions including nominal rate subscriptions, employer requests, advertiser's proof copies, and exchange copies.)</td>
<td>459</td>
</tr>
<tr>
<td>(2)</td>
<td>In-County Paid/Requested Mail Subscriptions stated on PS Form 3541. (Include direct written request from recipient, telemarketing and internet requests from recipient, paid subscriptions including nominal rate subscriptions, employer requests, advertiser's proof copies, and exchange copies.)</td>
<td>1298</td>
</tr>
<tr>
<td>(3)</td>
<td>Sales Through Dealers and Carriers, Street Vendors, Counter Sales, and Other Paid or Requested Distribution Outside USPS® (e.g. First-Class Mail®)</td>
<td>0</td>
</tr>
<tr>
<td>(4)</td>
<td>Requested Copies Distributed by Other Mail Classes Through the USPS</td>
<td>0</td>
</tr>
<tr>
<td>c. Total Paid and/or Requested Circulation (Sum of 15b (1), (2), (3), and (4))</td>
<td>1757</td>
<td>1757</td>
</tr>
<tr>
<td>d. Nonrequested Distribution (By Mail and Outside the Mail)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1)</td>
<td>Outside County Nonrequested Copies Stated on PS Form 3541 (Include Sample copies, Requests Over 3 years old, Requests induced by a Premium, Bulk Sales and Requests including Association Requests, Names obtained from Business Directories, Lists, and other sources)</td>
<td>10</td>
</tr>
<tr>
<td>(2)</td>
<td>In-County Nonrequested Copies Stated on PS Form 3541 (Include Sample copies, Requests Over 3 years old, Requests induced by a Premium, Bulk Sales and Requests including Association Requests, Names obtained from Business Directories, Lists, and other sources)</td>
<td>100</td>
</tr>
<tr>
<td>(3)</td>
<td>Nonrequested Copies Distributed Through the USPS by Other Classes of Mail (e.g. First-Class Mail, Nonrequestor Copies mailed in excess of 10% Limit mailed at Standard Mail® or Package Services Rates)</td>
<td>0</td>
</tr>
<tr>
<td>(4)</td>
<td>Nonrequested Copies Distributed Outside the Mail (Include Pickup Stands, Trade Shows, Showrooms and Other Sources)</td>
<td>0</td>
</tr>
<tr>
<td>e. Total Nonrequested Distribution (Sum of 15d (1), (2), (3) and (4))</td>
<td>110</td>
<td>110</td>
</tr>
<tr>
<td>f. Total Distribution (Sum of 15c and e)</td>
<td>1867</td>
<td>1867</td>
</tr>
<tr>
<td>g. Copies not Distributed (See Instructions to Publishers #4, (page #3))</td>
<td>33</td>
<td>33</td>
</tr>
<tr>
<td>h. Total (Sum of 15f and g)</td>
<td>1900</td>
<td>1900</td>
</tr>
<tr>
<td>i. Percent Paid and/or Requested Circulation (15c divided by f times 100)</td>
<td>94%</td>
<td>94%</td>
</tr>
</tbody>
</table>

16. Publication of Statement of Ownership for a Requester Publication is required and will be printed in the 6th issue of this publication.

17. Signature and Title of Editor, Publisher, Business Manager, or Owner

Lisa D. Tidd, Office Manager, USMS

Date

9/25/2009

I certify that all information furnished on this form is true and complete. I understand that anyone who furnishes false or misleading information on this form or who omits material or information requested on the form may be subject to criminal sanctions (including fines and imprisonment) and/or civil sanctions (including civil penalties).