Hyperbaric oxygen treatment of nonacute central retinal artery occlusion

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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-day 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

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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% (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


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, does diminish 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 there 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

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Abstracts

DECOMPRESSION AND THE DEEP STOP WORKSHOP PROCEEDINGS

JUNE 24-25, 2008
OVERVIEW

DECOMPRESSION AND THE DEEP STOP WORKSHOP

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 add 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 decompressed 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. 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 introduced 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 Kissman 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%.

**BBBLE DDETECTION 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 therepetitivedives 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
partial pressures (PtN2) exceeding 80% of the allowed M-value.

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:
In Memoriam

IVAN W. BROWN M.D.

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 Problem Wound
Management & 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
• 41 hours AMA PRA Category 1 Credits™
  for physicians
• 41 Prescribed credit hours by the AAFP
• 41 contact hours by the Florida Board
  of Nursing

Contact
• drobinson@diversifiedcs.com
• Phone: +1-904-296-6526
• www.diversifiedcs.com/healthcare-providers/continuing-education.php
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
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Undersea & Hyperbaric Medicine

The Journal of the Undersea and Hyperbaric Medical Society, Inc.

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2. Clinical research should be conducted only by scientifically qualified persons and under the supervision of a qualified medical man.

3. Clinical research cannot legitimately be carried out unless the importance of the objective is in proportion to the inherent risk to the subject.

4. Every clinical research project should be preceded by careful assessment of inherent risks in comparison to foreseeable benefits to the subject or to others.

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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.

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Lisa D. Tidd, Office Manager, UHMS

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).