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**SAFE INNER EAR GAS TENSIONS FOR SWITCH FROM
HELIUM TO AIR BREATHING DURING
DECOMPRESSION**



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14. ABSTRACT Inner ear decompression sickness (DCS) has followed deep breathing gas switches to air (or other nitrogen-oxygen mixtures) during decompression from deep heliox dives. This study investigates the safety of heliox-to-air breathing gas switches at moderate depths. Divers immersed in 80 °F (27 °C) water were compressed to 150, 170, or 190 fsw (561, 622, 683 kPa) breathing 79% helium-21% oxygen or to 220 fsw (775 kPa) breathing 84% helium-16% oxygen for a 60-minute bottom time — a time estimated to allow 99% equilibration of inner ear gas tensions with breathing gas. Divers were then decompressed at 30 fsw/min to 70 fsw (316 kPa) from 150 fsw, or to 100 fsw (408kPa) from the other depths. At 70 or 100 fsw, divers exited the water and breathed chamber air for 60 minutes. Following this air stop, divers were decompressed breathing 50% nitrogen-50% oxygen and 100% oxygen. Profiles were tested in order of increasing maximum depth. Each profile was accepted if 25 man-dives were free from inner ear or other central nervous system DCS during the air stop — a design chosen to detect greater than 0.2% probability of central nervous system DCS with approximately 3% significance and 96% power. There was no diagnosed DCS during or following any of the 104 man-dives completed. On the 220 fsw dive profile, one diver complained of “fullness” in the ear at the air stop that resolved during subsequent decompression and one diver complained of fleeting, mild shoulder pain (niggles) during decompression from 100 fsw. Equilibration of the inner ear with 651 kPa inspired helium followed by no-stop decompression to 100 fsw and switch to air breathing appears to have a low risk of inner ear DCS.					
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INTRODUCTION

Many commercial and military helium-oxygen (heliox) decompression procedures include breathing gas switches from heliox to air (or other nitrogen-oxygen mixtures) during decompression. The U. S. Navy has not used heliox-to-air breathing gas switches during decompression since the Sealab and Deep Diving Systems programs of the 1960's and 1970's, but has a renewed need to understand the conditions under which such gas switches are safe. For bounce diving, switching from heliox to air during decompression has practical and hypothetical advantages: air is cheaper than heliox, some decompression algorithms prescribe shorter decompressions with a heliox-to-air switch than without¹ (although there is experimental evidence to the contrary²), and there are some indications that air decompression results in a reduced incidence of Type II decompression sickness (DCS) than does heliox decompression.³ A potential disadvantage of switching from heliox to air during decompression is that it is associated with symptoms of injury to the vestibulocochlear apparatus (inner ear) such as vertigo, nausea, tinnitus, and hearing loss (inner ear DCS).⁴⁻⁶ Inner ear DCS poses a substantial risk because it is debilitating and can occur during the dive.

Vertigo, nausea, and other symptoms consistent with injury to the vestibulocochlear apparatus have been described during 1200 feet sea water (fsw, 3.68 MPa) heliox saturation dives following an isobaric switch to neon-rich or nitrogen-rich (1.01 MPa) breathing gas mixtures.⁷ This is accounted for by a physiological model which indicates that the inner ear is unusual in having diffusion-limited gas exchange, and that transient supersaturation can develop (and therefore bubbles may form) without decompression, principally due to diffusion of helium from the avascular fluid filled spaces into the surrounding vascular tissue exceeding the diffusion of nitrogen in the opposite direction.⁶ The magnitude of this supersaturation is proportional to the increase in inspired nitrogen partial pressure. Modeling indicates that counterdiffusion alone following a heliox-to-air breathing gas switch during decompression from bounce dives makes a trivial contribution to inner ear supersaturation.⁶ However, isolated inner ear DCS also follows decompression without a heliox-to-air breathing gas switch⁸⁻¹¹, indicating that inadequate decompression *per se* can also be an important cause of inner ear DCS. The association of inner ear DCS with a heliox-to-air breathing gas switch during decompression⁴⁻⁶ may be coincidental, or may be because counterdiffusion of helium and nitrogen exacerbates supersaturation from inadequate decompression and enhances growth of already formed bubbles.

Although there are successful decompression procedures that employ a switch from heliox to air during decompression, safe inner ear decompression immediately prior to a heliox-to-air gas switch has not been systematically characterized. The purpose of this study was to find a set of conditions under which a heliox-to-air breathing gas switch could be made safely, not necessarily to probe the limits of such a procedure. Inner ear DCS appears to occur more often following a breathing gas switch deeper than 100 fsw (408 kPa) than at shallower depths⁴ and 100 fsw was chosen as a practical depth for a heliox-to-air breathing gas switch. This study was designed to assess whether counterdiffusion causes unsafe potentiation of supersaturation at a 100 fsw gas switch depth under controlled conditions.

METHODS

EXPERIMENTAL DESIGN AND DECOMPRESSION SCHEDULES

Although the kinetics of gas exchange in the inner ear are complex, they can be approximated for longer time periods from the overall perfusion time constant, which is estimated to be 12.7 minutes.⁶ After a change in arterial gas tension, a compartment is 99% equilibrated with arterial blood in a period equal to 4.6 times the time constant. The inner ear is consequently equilibrated with arterial blood approximately 58 minutes after a change in arterial gas tension. Also, onset of inner ear symptoms generally occurs within 60 minutes after decompression or a breathing gas switch.^{4,8,9,12} The experimental philosophy was to conduct heliox dives to various depths for 60-minute bottom times, then conduct no-stop ascents to the depths at which the breathing gas is switched from heliox to air. This allowed near-complete equilibration of the inner ear with inspired gas partial pressures at depth and minimal change during ascent, so that the inner ear helium tension (concentration/solubility) and gas supersaturation on arrival at the heliox-to-air switch depth could be readily estimated. A 60-minute hold at the gas switch depth after the switch to air breathing allowed near-complete exchange of helium for nitrogen in the inner ear and likely onset of any symptom before continued decompression.

The decompression schedules tested were calculated using the Linear Exponential Multigas probabilistic model parameterized with the he8n25 data set (LEM-he8n25), the model that underlies the U. S. Navy MK 16 MOD 1 He-O₂ Decompression Tables.¹³ Standard fixed-fraction heliox mixtures were chosen to give approximately 1.3 atm PO₂ on the bottom. The diving was planned in two phases and the decompression schedules designed differently in each phase. The 150 – 190 fsw schedules (phase 1) were calculated for a target probability of DCS (P_{DCS}) of 2.3% and the 220 fsw schedule (phase 2) was calculated for a target P_{DCS} of 4.7%; see Appendix A for details.

Profiles were tested in order of increasing maximum depth and therefore increasing risk of DCS at the gas switch depth. Each profile was accepted if 25 man-dives were free from inner ear or other central nervous system DCS during the gas switch stop. DCS was diagnosed by the duty Diving Medical Officer. Monte Carlo simulations of this sequential stopping rule¹⁴ indicated that this trial design would detect greater than 0.2% probability of central nervous system DCS with approximately 3% significance and 96% power.

Table 1. Schedules tested

Bottom Depth (fsw)	BT (min)	He / O ₂ (%)	Stops (fsw, mins*)									TST	
			Air		50% N ₂ / 50% O ₂					†100% O ₂			
			100	70	70	60	50	40	30	20	10		
150	60	79 / 21	-	60	1	-	-	-	-	-	157	-	218
170	60	79 / 21	60	-	1	-	-	-	-	-	189	-	250
190	60	79 / 21	60	-	1	-	-	-	-	-	226	-	287
220	61	84 / 16	60	-	1	3	3	11	-	-	173	70	321

Descent rate 40 fpm. Ascent rate 30 fpm.

*Stop time does not include travel to stops. At gas switches, stop times begin once divers are confirmed on gas.

†A 15 minute air-break was taken after every 60 minutes of 100% oxygen breathing. Air breaks were not counted towards stop time.

DIVING

Forty-four qualified U. S. Navy divers gave informed consent under NEDU Institutional Review Board approved protocols 10-16/32241 and 11-27/32253 and completed experimental dives. At the time of their first dive in this study, divers mean (SD) age was 35 (7) years, body weight was 196 (25) pounds or 89.1 (11.3) kg, height was 70.5 (2.4) inches or 1.79 (0.06) m, BMI was 28 (3), and body fat estimated from body dimensions¹⁵ was 20 (4) %. Details about diver-subjects are given in Appendix B. A Diving Medical Officer judged all divers to be physically qualified for diving on the basis of review of medical records and a physical examination. Immediately before each experimental dive, diver-subjects reported any current injury or illness and their amounts of exercise and sleep, any alcohol consumed, and any medications used in the previous 24 hours. On the bases of this self-report and a brief interview, a Diving Medical Officer either cleared or disqualified diver-subjects for participating in each experimental dive. Divers refrained from any hyperbaric exposure for a minimum of 48 hours before and following any experimental dive. Divers also refrained from any hypobaric exposure for a minimum of 48 hours before and 72 hours following any experimental dive. Diving took place over an 18 month period and divers participated in one to six experimental dives (median = 2). See Appendix C for individual diving intensity.

All experimental dives were completed in the Ocean Simulation Facility (OSF) at the Navy Experimental Diving Unit. One dive per day was conducted, generally on Monday through Thursday, with up to four divers participating in each dive. Wet pot water temperature was actively controlled with a target of 80 °F (27 °C) ± 2 °F. Divers dressed for comfort in neoprene wetsuits and wore an equipment harness and breathed heliox via an umbilical and full face mask equipped with an oronasal mask, demand valve, and diver communications (U.S. Navy MK 20 underwater breathing apparatus). One at a time, divers donned the MK 20, entered the wet pot, and assumed a prone position fully submerged with mid-chest approximately two feet (0.6 m) below the wet pot water surface. Once all divers were situated, the wet pot air space, trunk, and C chamber of the OSF were compressed by the introduction of compressed air, at a target descent rate of 40 fsw/min, until the pressure at diver mid chest level (chamber air pressure plus 2 fsw hydrostatic pressure) was equivalent to the specified maximum depth.

Upon reaching bottom the divers began exercising on custom-built, hysteresis-braked (model HB210, Magtrol; Buffalo, NY), underwater cycle ergometers. The ergometers were mounted to position the divers in a semi-prone position (approximately 15° head-up inclination) during pedaling to mimic underwater fin swimming. Divers pedaled at a target cadence of 60 rpm with the ergometer hysteresis brake controller (W.E. Collins; Braintree, MA) set at 25 watts so that divers' work rate (incorporating the extra power required due to submersion in this diving dress) was approximately 100 watts.¹⁶ Divers exercised intermittently (10 minutes on / 10 minutes off) for an estimated average diver oxygen consumption of 1.1 L/min.^{17,18} Divers exercised until five minutes before ascent and then rested in either seated or prone positions with mid chest level two feet below the wet pot water surface.

The wet pot, trunk, and C chamber were decompressed at 30 fsw/min until the air pressure above the wet pot water surface was 70 fsw or 100 fsw according to the appropriate schedule given in Table 1. At 70 or 100 fsw, divers stood with head out of water and simultaneously removed their full face masks and began breathing chamber air. One at a time the divers climbed the ladder out of the wet pot into the trunk and then into C chamber. After changing into dry clothes the divers opened the hatch to, and moved into B chamber, which had been compressed with air to the air switch depth. There the divers remained at rest, generally seated, for the remainder of the decompression. Approximately 50 minutes after reaching the air switch depth, divers were interviewed, guided using a brief questionnaire including questions regarding vision or hearing changes. Sixty minutes after the divers reached the air switch depth, B chamber was decompressed to 70 fsw (if not already at 70 fsw) and divers began breathing 50% N₂ / 50% O₂ via the Built in Breathing System (BIBS) masks. On reaching 20 fsw divers removed BIBS for a two-minute air break while BIBS gas was shifted to 100% oxygen. A 15 minute air-break was taken after every 60 minutes of 100% oxygen breathing. Air breaks were not counted towards oxygen stop times. During the periods of breathing chamber air, divers could eat and drink water, juice or sports drinks.

A Diving Medical Officer interviewed all divers at 10 minutes and two hours after surfacing, and again the following day, generally between 21 and 24 hours after surfacing (mean 19; range 15–26, n = 92: 12 missing). Divers who did not report any symptom of decompression sickness (DCS) within 48 hours after completing a dive were given the diagnosis of “no DCS”.

Wet pot depth (C chamber pressure plus 3 fsw), water temperature, pedaling cadence, and cycle ergometer hysteresis break settings were digitized and recorded with a microcomputer based data acquisition system every two seconds throughout the in water phase of the dive. When divers reached the wet pot surface at the gas switch depth, B chamber air pressure in fsw and air temperature were recorded every two seconds for the remainder of the dive. B and C chamber atmospheres were continuously sampled and analyzed using a quadrupole mass spectrometer (Extrel CMS; Pittsburg, PA) tuned to detect helium, nitrogen, oxygen, argon, and carbon dioxide.

RESULTS

No diver was diagnosed with DCS by the Diving Medical Officer during or following any of the 104 man-dives completed on the test schedules (Table 2). On the 220 fsw dive profile, one diver complained of “fullness” or reduced hearing in the left ear at the air stop that resolved during subsequent decompression. This diver had complained of left-sided neck pain prior to diving and complained of altered sensation on the left side of the face several days following diving. At 20 fsw during a 220 fsw profile, one diver admitted to transient, mild right shoulder pain (niggles) during decompression from 100 fsw but which had resolved. During a 190 fsw dive, one diver complained of dizziness and nausea upon standing during the air break following the first 60-minute oxygen breathing period at 20 fsw. Examination revealed nystagmus (a pre-existing condition in this diver), but no other neurological signs or symptoms. During a 220 fsw dive, one diver developed abdominal cramps and nausea during oxygen breathing. This was attributed to the cold pizza the diver ate during the 100 fsw stop. Details of these medical incidents are given in Appendix D.

Table 2. Number and outcome of dives

Heliox Excursion		Air Switch	DCS / dives
He% / O ₂ %	fsw	fsw	
79 / 21	150	70	0 / 26
79 / 21	170	100	0 / 25
79 / 21	190	100	0 / 26
84 / 16	220	100	0 / 27

The LEM-h8n25 predicted incidence of DCS for this dive series was 3.0 (95% prediction limits [C.L.] 1.9, 4.5) DCS in 104 man-dives. These binomial confidence limits overlap substantially with those for the diagnosed incidence of zero DCS in 104 man-dives (95% C.L. 0, 2.8), and it is not possible to say that the observed and estimated incidences are different. On retrospective review, the incident of transient, mild right shoulder pain lasting less than 60 minutes during decompression (see Appendix D, Diver ID 5, 9 November 2011) meets the U. S. Navy primary decompression database diagnostic criteria¹⁹ for marginal DCS (not requiring recompression). Such cases are usually assigned a fractional value of 0.1 DCS. Using this convention the observed incidence of DCS was 0.1 in 104 man-dives.

Seven divers reported minor symptoms of pulmonary oxygen toxicity (cough or mild discomfort on inspiration) and two divers reported Draeger ear following the 104 man-dives. Four divers had ear or sinus squeeze on descent but were able to complete the dive and one had tooth ‘squeeze’ on ascent. Five divers had ear squeeze and could not complete the descent. In two cases, the un-afflicted divers were able to continue, but in three cases the dive was aborted for the afflicted and un-afflicted divers. Overall, 14 man-dives were aborted, none of which are included in the 104 man-dives completed.

Several dives deviated from the intended schedule. During four compressions of the OSF, accounting for 16 man-dives, holds caused delays in descent of between 58 s and 155 s; bottom time was not adjusted. On another occasion, one diver had ear squeeze at 15 fsw, the OSF was decompressed to the surface, the afflicted diver removed, and the remaining three divers compressed to 150 fsw; in this instance, bottom time started at the original leave surface time, and as a result the divers had 245 s less time on the bottom. In phase 2, one diver had ear squeeze at 26 fsw, the OSF was decompressed to the surface (158 s total dive time), the afflicted diver removed, and the remaining three divers compressed to 220 fsw; the phase 2 protocol allowed this delay to be ignored (see Appendix A), so the remaining three divers conducted the dive according to the original schedule starting from the beginning of the second compression. On two occasions (three man-dives each), the 20 fsw decompression stop was extended with additional air breathing time. During a 190 fsw dive, an additional 20 minute air break was started five minutes into the second 60-minute oxygen breathing period while the diver who complained of dizziness and nausea was examined. During a 220 fsw dive, the first oxygen breathing period was stopped after 50 minutes and followed by a 25-minute air break while the diver who complained of cramps and nausea was examined. Subsequent oxygen periods were adjusted to give the scheduled oxygen breathing time at 20 fsw and 10 fsw.

Since the divers breathed heliox via open circuit underwater breathing apparatus, exhaled gas contaminated the OSF atmosphere with helium. Although monitored during all dives, OSF atmosphere composition was only recorded for the deepest dives, during which the greatest mass of helium was exhausted into the OSF. Helium accumulated to approximately 10% of C chamber atmosphere which is directly above the wet pot and was open to the wet pot throughout the dive. Once the hatch between C and B chamber was opened, helium from C chamber, trunk, and wet pot contaminated the B chamber air atmosphere and B chamber helium levels ranged from 4.0 to 5.8%.

DISCUSSION

This study was designed to assess whether counterdiffusion after a heliox-to-air gas switch at 100 fsw during decompression from a heliox dive causes unsafe potentiation of inner ear gas-supersaturation and undue risk of inner DCS. Experimental conditions allowed 1) ready estimation of inner ear gas-supersaturation at the time of the heliox-to-air gas switch, and 2) completion of counterdiffusion processes in the inner ear — and hence likely onset of inner ear DCS symptoms and signs — at the gas switch depth before further decompression. It was found that switches to air breathing at 100 fsw after no-stop decompressions from heliox dives to modest depths did not pose undue risk of inner ear DCS. Moreover, decompressions scheduled with the LEM-h8n25 decompression model were successful with no diagnosed cases of DCS requiring recompression in the 104 completed man-dives.

INNER EAR DECOMPRESSION

Because of the experimental conditions in the present dive series, the absence of inner ear symptoms has implications for the safety of heliox-to-air gas switches during decompressions from dives with longer bottom times and deeper depths than those tested. To develop these implications, it is useful to consider the two plausible mechanisms of inner ear DCS. First, injury may result from bubble formation in the inner ear structures as a result of local gas supersaturation (autochthonous bubble formation). A second mechanism may involve passage of venous gas emboli (VGE) into the arterial circulation (right-to-left shunt) and then to the inner ear where the bubbles grow by acquiring more gas.^{10,11,20} Both of these mechanisms are dependent on the level and duration of gas supersaturation in the inner ear, where the kinetics of inert gas exchange have never been measured. Figure 1 shows the inner ear inert gas partial pressures during the 220 fsw dive through to the end of the 100 fsw air stop as estimated with a physiological model of inner ear perfusion and diffusive gas exchange between the different fluid compartments in the inner ear.⁶ The inner ear inert gas partial pressures at both 200 fsw and 100 fsw are 99% equilibrated with breathing gas after 60 minutes, which corresponds to a perfusion-limited time constant of 12.7 minutes. Even if the overall perfusion time constant of the inner ear is two times this value, the inner ear would be nearly (92%) equilibrated with breathing gas after sixty minutes. In the present experiments, 60 minutes of bottom time and time at the gas switch depth therefore allowed near-complete equilibration of the inner ear with breathing gas.

If inner ear DCS is caused principally by autochthonous bubble formation, the present results suggest that unlimited bottom times at depth up to 220 fsw with no-stop ascent to 100 fsw would be possible with low risk of inner ear DCS. Because the present sixty-minute bottom times produced near-complete equilibration of the inner ear with the prevailing breathing gas, longer bottom times would result in only trivial increases in inner ear inert gas tensions. Therefore, ascent to 100 fsw following any bottom time of 60 minutes or longer would produce essentially the same level and duration of gas supersaturation and the same kinetics of the individual inert gases in the inner ear. Autochthonous bubble formation and growth in the inner ear, and therefore risk of inner ear DCS caused by such bubble formation and growth, should be practically independent of bottom time longer than sixty minutes.

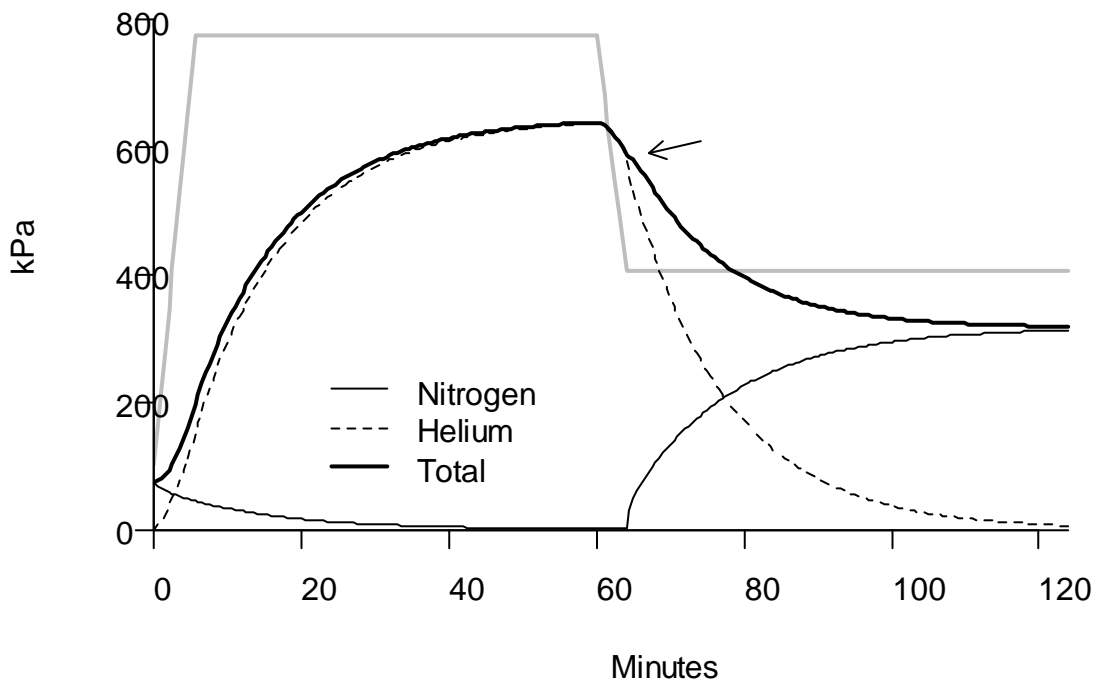


Figure 1. Inert gas tensions in the vascular compartment (membranous labyrinth) of a three-compartment inner ear model⁶ during the 220 fsw heliox dive with switch to air breathing on arrival at 100 fsw. Ambient pressure is indicated by the grey line. Total inert gas tension (heavy solid line) is the sum of the nitrogen (thin solid line) and helium (dashed line) tensions. The vascular compartment is 99% equilibrated with inspired helium partial pressure (651 kPa) at the end of bottom time. Total vascular compartment inert gas tension is 588 kPa upon arrival at 100 fsw (408 kPa). Slowing of total inert gas washout from the vascular compartment due to differences in helium and nitrogen kinetics is just evident as an inflection in the heavy solid line (arrow). Helium and nitrogen partial pressures in the perilymph and endolymph are not shown but lag only slightly behind those in the vascular compartment.

In contrast, if inner ear DCS is caused by right-to-left shunting of VGE, the risk of inner ear DCS may be higher after no-stop ascents to 100 fsw from dives with bottom times longer than sixty minutes at the dive depths tested in the present study. Right-to-left shunting of VGE has been implicated in inner ear DCS because a high prevalence of “major” (based on large number of bubbles detected in the middle cerebral artery) right-to-left shunting of venous bubble contrast medium is found in divers who suffered inner ear DCS following relatively shallow dives.^{10,11} Interestingly, two reported cases of inner ear DCS with onset at depth during deep heliox dives (one during decompression from 686 fsw and one following a downward excursion from 1306 to 1502 fsw) were preceded by detection of high VGE grades.²¹ Tissues that equilibrate more slowly than the inner ear will continue to on-gas during bottom times longer than sixty minutes, which will result in greater supersaturations in such tissues upon subsequent no-stop ascent to 100 fsw. VGE could form in greater profusion with such supersaturations in these tissues and, to the extent that right-to-left shunt may increase with VGE profusion, increase the risk of shunt-induced inner ear DCS.

However, circumstantial evidence indicates that arterialized VGE only cause injury in the inner ear if the bubbles can grow by acquiring more gas²⁰ — so called “Type III” DCS. The vascular anatomy overwhelmingly favors embolization of the brain rather than the inner ear, yet inner ear DCS characteristically presents without cerebral symptoms — a difference explained by faster gas washout from the brain than from the inner ear such that negligible supersaturation develops in the brain with standard decompression rates. In open-chamber cardiac surgery, a situation where many bubbles distribute to the cerebral vessels but no tissues are supersaturated, patients often develop signs indicative of brain injury, but inner ear injury is never reported. Typically, bubbles can only grow while tissue is supersaturated; in Figure 1, the period of supersaturation roughly coincides with the 17-minute period when the heavy solid line is greater than ambient pressure (Figure 1 does not include metabolic gas tensions). It is theoretically possible for arterialized, helium-rich VGE to grow transiently if they reach undersaturated, nitrogen-rich tissue in which the product of tissue solubility and diffusivity for nitrogen greatly exceeds that for helium, but this is physiologically implausible based on published values of solubility and diffusivity.²² Therefore, increased risk of inner ear DCS via a Type III mechanism requires increased VGE production at a time when the VGE can be arterialized and impact the inner ear during its brief period of supersaturation. There are relatively few published data concerning VGE detection at hyperbaric pressure during decompression: VGE have been detected about 15 minutes after a large decompression to the first stop, but are not usually detected until much later.^{21,23,24} VGE consequently do not appear to coincide with the period of inner ear supersaturation, so that dives with bottom times longer than 60 minutes at up to 220 fsw may not pose a greater risk of inner ear DCS upon ascent to 100 fsw and switch to air breathing. This is not to say that dives with longer bottom times would not place divers at risk for other forms of DCS. One diver developed marginal symptoms during decompression following 60 minutes at 220 fsw.

Dives deeper than 220 fsw with no-stop ascent to 100 fsw and a heliox-to-air breathing gas switch should present no greater risk of inner ear DCS than encountered in the present dives as long as bottom times are limited so that inner ear gas tensions upon leaving bottom are no higher than those in the present dives (approximately 651 kPa of inert gas). Under such limits, the same level and duration of gas supersaturation, the same kinetics of the individual inert gases, and the same autochthonous bubble formation and growth in the inner ear will prevail at 100 fsw. To take up the same inert gas into the inner ear as during a 60-minute bottom time at 220 fsw, deeper dives will require shorter bottom times, and therefore VGE production also will likely be no greater. Tissues that equilibrate with breathing gas more slowly than the inner ear will take up less inert gas during the deeper, shorter bottom times, and therefore be less supersaturated, and a reduced source of VGE upon arrival at 100 fsw than during the tested 220 fsw for 60-minute bottom time dives. Tissues that exchange gas faster than the inner ear will take up more gas during deeper, shorter bottom times but also washout gas quickly and therefore develop negligible supersaturation during decompression, and therefore are probably not an important source of VGE. Using a 12.7 minute time constant and 60 fsw/min descent rate to estimate inner ear inert gas tensions, the approximate bottom times that produce a similar inner ear inert gas tension as 220 fsw for 60 minutes, and therefore might allow no-stop ascent to 100 fsw with heliox-to-air breathing gas switch are given in Table 3. These bottom times are untested, and

unknown whether these would be safe with respect to all forms of DCS. However, these bottom times would result in no more supersaturation in most tissues than the tested 220 fsw for 60 minutes bottom time. The bottom times in column two are slightly longer than the longest tabulated bottom times with the first decompression stop at 100 fsw in the Canadian Forces Helium-Oxygen Diving Tables²⁵, which use 84% He / 16% O₂, have a heliox-to-air breathing gas switch at the first decompression stop, and have been man-tested without incidents of inner ear DCS.²⁶ The bottom times in column three are in general agreement with longest tabulated bottom times with a first decompression stop at 100 fsw in the U. S. Navy MK 16 MOD 1 He-O₂ Decompression Tables, which are 1.3 atm PO₂-in-helium schedules and do not include a breathing gas switch.^{13,27} It would seem that these latter decompression procedures also limit inner ear supersaturations to levels that would allow a heliox-to-air breathing gas switch at 100 fsw.

Table 3. Potential (untested) bottom times for depths deeper than 220 fsw

Depth (fsw) fsw	Bottom Time (minutes)	
	84% He / 16% O ₂	1.3 atm PO ₂ -in-He
230	44	47
240	35	34
250	30	29
260	26	25
270	24	23
280	22	21
290	20	19
300	19	18

LEM-he8n25 DECOMPRESSION

The LEM-h8n25-predicted incidence of DCS for this dive series was 3.0 (95% prediction limits [C.L] 1.9, 4.5) DCS compared to the observed incidence of 0.1 DCS. Even though these predicted and observed incidences are not significantly different, the lower observed than predicted DCS incidence may be attributable to the particular diving conditions. The present dives were conducted with the divers dry and warm during decompression. Immersed divers who are warm during decompression have significantly lower P_{DCS} compared to being cold during decompression.²⁸

In accord with occurrence of the marginal DCS case at the 100 fsw gas switch stop, a substantial portion of the LEM-he8n25-estimated risk accumulates during this stop. However, LEM-he8n25 may overestimate the effects of the helium-to-air switch at this stop. For instance, if the 220 fsw schedule is conducted with 79% He / 21% O₂ instead of air breathing at 100 fsw, the LEM-he8n25- estimated P_{DCS} is 1.6% rather than 4.7%. This increased P_{DCS} arises from faster uptake of nitrogen than washout of helium in some LEM-he8n25 compartments, an imbalance that results in an increase in the gas burden (a notional tissue tension representing both dissolved and free gas as if all were dissolved^{13,29}) in excess of ambient pressure in these compartments: The instantaneous risk of DCS is a function of this excess gas burden. A physiological interpretation of this model behavior would be greater nitrogen influx than helium efflux from tissue bubbles, but LEM-he8n25 may overestimate any risk attributable to this phenomenon.

CONCLUSIONS AND RECOMMENDATIONS

This study was designed to assess whether helium-nitrogen counterdiffusion after a heliox-to-air gas switch at 100 fsw during decompression causes unsafe potentiation of inner ear gas-supersaturation with undue risk of inner ear DCS. The results indicate that:

1. Dives to depths up to 220 fsw (704 kPa) with 60-minute bottom time and 651 kPa inspired helium partial pressure followed by no-stop decompression to 100 fsw (408 kPa) and switch to air breathing have low risk of inner ear DCS.
2. The LEM-h8n25 model may be used to compute decompression schedules with a heliox-to-air breathing gas switch at 100 fsw for heliox dives to depths up to 220 fsw and bottom times up to 60 minutes without special consideration of the risk of inner ear DCS. Additional man-testing is required to confirm that the model may also be used to compute schedules for such dives with longer than 60-minute bottom times and for such dives to depths up to 300 fsw with short bottom times.

REFERENCES

1. A. A. Buhlmann, G. P. Michel, translator, *Decompression - Decompression Sickness* (Springer-Verlag, Berlin, 1984) (English translation of Dekompression - dekompressionkrankheit, first published 1983).
2. S. S. Survanshi, E. C. Parker, D. D. Gummin, E. T. Flynn, C. B. Toner, D. J. Temple, R. Ball, and L. D. Homer., *Human Decompression Trial with 1.3 ATA Oxygen in Helium*, Technical Report 98-09, Naval Medical Research Institute, Jun 1998.
3. J. S. Shannon, R. D. Vann, C. F. Pieper, E. D. Thalmann, and W. A. Gerth, "Relationship Between Inert Gas Type, Venous Gas Emboli (VGE), and Decompression Sickness (DCS)" [abstract], *Undersea and Hyperbaric Medicine*, Vol. 31 (2004), pp. 336-337.
4. J. C. Farmer, W. G. Thomas, D. G. Youngblood, and P. B. Bennett, "Inner Ear Decompression Sickness," *The Laryngoscope*, Vol. 86, No. 9 (1976), pp. 1315-1327.
5. P. B. Bennett, R. D. Vann, J. Roby, and D. Youngblood, "Theory and Development of Subsaturated Decompression Procedures for Depths in Excess of 400 Feet," *Underwater Physiology VI, Proceedings of the Proceedings of the 6th Symposium on Underwater Physiology*, C. W. Shilling and M. W. Beckett, eds., (Federation of American Societies for Experimental Biology, Bethesda (MD), 1978), pp. 367-381.
6. D. J. Doolette and S. J. Mitchell, "A Biophysical Basis for Inner Ear Decompression Sickness," *Journal of Applied Physiology*, Vol. 94, No. 6 (2003), pp. 2145-2150.

7. C. J. Lambertsen and J. Idicula, "A New Gas Lesion Syndrome in Man, Induced by "Isobaric Gas Counterdiffusion", " *Journal of Applied Physiology*, Vol. 39, No. 3 (1975), pp. 434-443.
8. E. D. Thalmann, "Testing of Revised Unlimited-Duration Upward Excursions During Helium-Oxygen Saturation Dives," *Undersea Biomedical Research*, Vol. 16 (1989), pp. 195-218.
9. Z. Nachum, A. Shupak, O. Spitzer, Z. Sharoni, I. Doweck, and C. R. Gordon, "Inner Ear Decompression Sickness in Sport Compressed-Air Diving," *The Laryngoscope*, Vol. 111, No. 5 (2001), pp. 851-856.
10. C. Klingmann, P. J. Benton, P. A. Ringleb, and M. Knauth, "Embolitic Inner Ear Decompression Illness: Correlation With a Right-to-Left Shunt," *The Laryngoscope*, Vol. 113 (2003), pp. 1356-1361.
11. E. Cantais, P. Louge, A. Suppini, P. P. Foster, and B. Palmier, "Right-to-Left Shunt and Risk of Decompression Illness With Cochleovestibular and Cerebral Symptoms in Divers: Case Control Study in 101 Consecutive Dive Accidents," *Critical Care Medicine*, Vol. 31, No. 1 (2003), pp. 84-88.
12. C. Klingmann, "Inner Ear Decompression Sickness in Compressed-Air Diving," *Undersea and Hyperbaric Medicine*, Vol. 39, No. 1 (2012), pp. 589-594.
13. W. A. Gerth and T. M. Johnson, *Development and Validation of 1.3 ATA PO₂-in He Decompression Tables for the MK 16 MOD 1 UBA*, NEDU TR 02-10, Navy Experimental Diving Unit, Aug 2002.
14. D. J. Doolette and W. A. Gerth, "Significance and Power of Sequential Bernoulli Trials" [abstract], *Undersea and Hyperbaric Medicine*, Vol. 36, No. 4 (2009), p. 257.
15. J. A. Hodgdon, "Body Composition in the Military Services: Standards and Methods," in *Body Composition and Physical Performance: Applications for the Military Services*, B. M. Marriott, J. Grumstrup-Scott, eds. (National Academy Press, Washington DC, 1992), Ch. 4, pp. 57-70.
16. B. E. Shykoff, *Underwater Cycle Ergometry: Power Requirements With and Without Diver Thermal Dress*, NEDU TR 09-01, Navy Experimental Diving Unit, Jan 2009.
17. B. E. Shykoff, *Oxygen Consumption As a Function of Ergometer Setting in Different Diver's Dress: Regression Equations*, NEDU TM 09-06, Navy Experimental Diving Unit, Aug 2009.
18. D. J. Doolette, W. A. Gerth, and K. A. Gault, *Addition of Work Rate and Temperature Information to the Augmented NMRI Standard (ANS) Data Files in the "NMRI98" Subset of the USN N₂-O₂ Primary Data Set*, NEDU TR 11-02, Navy Experimental Diving Unit, Jan 2011.

19. D. J. Temple, R. Ball, P. K. Weathersby, E. C. Parker, and S. S. Survanshi, *The Dive Profiles and Manifestations of Decompression Sickness Cases After Air and Nitrogen-Oxygen Dives*, Technical Report 99-02, Naval Medical Research Center, 1999.
20. S. J. Mitchell and D. J. Doolette, "Selective Vulnerability of the Inner Ear to Decompression Sickness in Divers With Right to Left Shunt: the Role of Tissue Gas Supersaturation," *Journal of Applied Physiology*, Vol. 106, No. 1 (2009), pp. 298-301.
21. B. Gardette, "Correlation Between Decompression Sickness and Circulating Bubbles in 232 Divers," *Undersea Biomedical Research*, Vol. 6, No. 1 (1979), pp. 99-107.
22. T. Lango, T. Morland, and A. O. Brubakk, "Diffusion Coefficients and Solubility Coefficients for Gases in Biological Fluids: a Review," *Undersea and Hyperbaric Medicine*, Vol. 23, No. 4 (1996), pp. 247-272.
23. T. S. Neuman, D. A. Hall, and P. G. Linaweaver, "Gas Phase Separation During Decompression in Man: Ultrasound Monitoring," *Undersea Biomedical Research*, Vol. 3, No. 2 (1976), pp. 121-130.
24. M. R. Powell, W. Thoma, H. D. Fust, and P. Cabarro, "Gas Phase Formation and Doppler Monitoring During Decompression With Elevated Oxygen," *Undersea Biomedical Research*, Vol. 10, No. 3 (1983), pp. 217-224.
25. *DCIEM Diving Manual* (Universal Dive Techtronics, Richmond, British Columbia, 1992)
26. P. Tikuisis and R. Y. Nishi, *Role of Oxygen in a Bubble Model for Predicting Decompression Illness*, Report 94-04, Defence and Civil Institute of Environmental Medicine, Jan 1994.
27. Naval Sea Systems Command, *U.S. Navy Diving Manual, Revision 6, NAVSEA 0910-LP-106-0957/SS521-AG-PRO-010 ed.* (Naval Sea Systems Command, Arlington (VA), 2008)
28. W. A. Gerth, V. L. Ruterbusch, and E. T. Long, *The Influence of Thermal Exposure on Diver Susceptibility to Decompression Sickness*, NEDU TR 06-07, Navy Experimental Diving Unit, Nov 2007.
29. E. D. Thalmann, E. C. Parker, S. S. Survanshi, and P. K. Weathersby, "Improved Probabilistic Decompression Model Risk Predictions Using Linear-Exponential Kinetics," *Undersea and Hyperbaric Medicine*, Vol. 24, No. 4 (1997), pp. 255-274.
30. J. K. Summitt and M. N. Kahn, *Report of Experimental Dives for ADS-IV Decompression Schedules*, Research Report 4-70, Navy Experimental Diving Unit, Aug 1970.
31. P. K. Weathersby, J. R. Hays, S. S. Survanshi, L. D. Homer, B. L. Hart, E. T. Flynn, and M. E. Bradley, *Statistically Based Decompression Tables II. Equal Risk Air Diving*

Decompression, Technical Report 85-17, Naval Medical Research Institute, Mar 1985.

32. W. A. Gerth and R. D. Vann, *Development of Iso-DCS Risk Air and Nitrox Decompression Tables Using Statistical Bubble Dynamics Models*, Final Report National Oceanic and Atmospheric Administration, Office of Undersea Research, 1996.

APPENDIX A DECOMPRESSION SCHEDULES

HELIOX-TO-AIR GAS SWITCH

The primary purpose of this study was to find a set of conditions under which a heliox-to-air breathing gas switch could be made safely — not necessarily to probe the limits of such a procedure. Operational considerations motivated the choice of 100 fsw for the first decompression stop and gas switch depth. The target maximum depth was selected by evaluation of previous, well-documented experimental dives that resulted in inner ear decompression sickness (inner ear DCS). The gas kinetics of the inner ear for longer time periods can be approximated as a single compartment with perfusion time constant estimated to be 12.7 minutes.⁶ Using this time constant for both helium and nitrogen, and zero tensions of metabolic gases, hypothetical inner ear gas supersaturation was calculated at various pertinent time points from the logged dive profiles from previous studies, as well as for candidate test schedules for the present series.

NEDU testing of upward excursions from saturation diving storage depth resulted in several incidents of inner ear DCS from 1976 to 1982. Summaries of these excursions are given by Thalmann⁸ and more detailed descriptions were assembled from the NEDU diving logs. Storage depths ranged from 640 fsw (1.96 MPa) to 1400 fsw with upward excursion distances of 123 fsw to 234 fsw at ascent rates near 60 fsw/min and with decompression continuing at saturation decompression rates (typically 6 fsw/h). Calculated inner ear gas tensions resulted in peak supersaturations of between 2.7 atm (274 kPa) and 5.7 atm with some level of supersaturation sustained for over 30 minutes. A more recent incident of inner ear DCS (1996) followed a more modest upward excursion consistent with procedures in the current U. S. Navy Diving Manual (840 fsw to 713 fsw at 2 fsw/min). This excursion resulted in peak inner ear supersaturation of 1.9 atm.

Development of decompression schedules for the Advanced Diving System IV involved testing of 200 fsw to 400 fsw heliox dives with a switch to air breathing during decompression³⁰, typically not at the first decompression stop. These dives resulted in one case of diagnosed inner ear DCS and one probable inner ear DCS (both manifest as hearing loss). The inner ear DCS cases followed 350 fsw / 30 minute bottom time dives with first decompression stops at 200 fsw (calculated inner ear supersaturation 2.2 atm) and 190 fsw (calculated inner ear supersaturation 2.6 atm). Only a few other dives in that series resulted in higher calculated inner ear supersaturations at the first decompression stop.

Based on the foregoing analysis, 1.9 atm was chosen as the maximum allowed inner ear supersaturation at the time of the heliox-to-air gas switch for the present study. The 220 fsw schedule tested has an estimated 1.9 atm supersaturation on arrival at the 100 fsw first stop.

LEM-he8n25

A secondary objective of this study was to test the use of the Linear Exponential Multigas probabilistic model parameterized with the he8n25 data set (LEM-he8n25) for dives that include a heliox-to-air breathing gas switch (including the Advanced Diving Systems IV dives mentioned above). In principle, LEM-he8n25 is applicable to computing decompression schedules for these dives because it tracks helium, nitrogen, and oxygen and the he8n25 data set includes dives with fixed-fraction breathing gas and some dives with heliox-to-air breathing gas switches. However, LEM-he8n25 was developed for MK 16 MOD 1 closed-circuit underwater breathing apparatus diving and validated by successful completion of constant PO₂-in-helium dives only.¹³

Probabilistic decompression models are used to estimate the probability of DCS (P_{DCS}) of a dive profile and, in conjunction with a search algorithm, find decompression schedules with the minimum total stop time (TST) that does not exceed a target P_{DCS}.^{31,32} The diving was planned in two phases and the decompression schedules designed differently in each phase. The 150 – 190 fsw schedules (phase 1) were calculated by specifying a 60 minute bottom time and initial ascent breathing 79% He / 21% O₂ to a 60-minute first stop breathing air. Subsequent decompression was the minimum TST for a target P_{DCS} of 2.3%, the same target used in calculating the MK 16 MOD 1 He-O₂ Decompression Tables.¹³ Decompression was calculated assuming 50% N₂ / 50% O₂ breathing from 70 to 20 fsw and 98% oxygen at a 20 fsw last stop depth and to the surface (although the actual inspired oxygen fractions breathed using BIBS were probably lower). Air break time between oxygen breathing periods was considered dead time (no gas exchange). Due to a typographical error, the 20 fsw stop times for the 170 fsw and 190 fsw dives in Table A-1 are one minute longer than calculated.

It was not possible to calculate a decompression schedule with target P_{DCS} of 2.3% for the 220 fsw dive (phase 2) because 3.4% LEM-he8n25-estimated risk accrues by the end of the 60 minute air-breathing first stop at 100 fsw. Therefore, the 220 fsw schedule was calculated for a target P_{DCS} of 4.7%. Also, the 220 fsw schedule shown in Table A-1 is not the search algorithm optimum (which had three minutes shorter TST and did not have stops and 50 and 40 fsw). Instead, the dive profile was first calculated as if the 100 fsw stop and ascent to 70 fsw was conducted breathing 79% He / 21% O₂ instead of air, and the resulting 50% N₂ / 50% O₂ stop times used. This was done for a possible follow-up comparison of a 79% He / 21% O₂ gas switch with the air switch tested — a comparison that did not eventuate. The final 220 fsw schedule was calculated by specifying 84% He / 16% O₂ breathing during descent, time on the bottom and initial ascent, a 60-minute first stop breathing air, and the 50% N₂ / 50% O₂ stop times at 70–40 fsw shown in Table A-1. Subsequent decompression was the minimum TST for a target P_{DCS} of 4.7, assuming 50% N₂ / 50% O₂ breathing continuing from 40 fsw until reaching 20 fsw and then 98% oxygen during the 20 fsw stop and to the surface with a 10 fsw last stop depth. The four dive profiles are illustrated in Figure A-1.

Table A-1 Schedules tested

Bottom Depth (fsw)*	BT (min)	He / O ₂ (%)	Stops (fsw, mins*)									TST	
			Air		50% N ₂ / 50% O ₂					100% O ₂			
			100	70	70	60	50	40	30	20	10		
150	60	79 / 21	-	60	1	-	-	-	-	-	157	-	218
170	60	79 / 21	60	-	1	-	-	-	-	-	189	-	250
190	60	79 / 21	60	-	1	-	-	-	-	-	226	-	287
220	61 [†]	84 / 16	60	-	1	3	3	11	-	-	173	70	321

Descent rate 40 fpm. Ascent rate 30 fpm.

*Stop time did not include travel to stops. Air stop time began when all divers began breathing chamber air. 70 fsw stop time began once all divers were breathing 50% N₂ / 50% O₂. A two-minute air break was taken on arrival at 20 fsw. 20 fsw stop time began once all diver were breathing 100% oxygen. A 15 minute air-break was taken after every 60 minutes of 100% oxygen breathing. If a final calculated oxygen period was less than 10 minutes, it was combined with the previous 60 minute oxygen period without an air break. Air breaks were not counted towards stop time. Divers surfaced breathing oxygen.

[†]Schedule calculated for 55 minutes time on the bottom (60.5 min BT), see below.

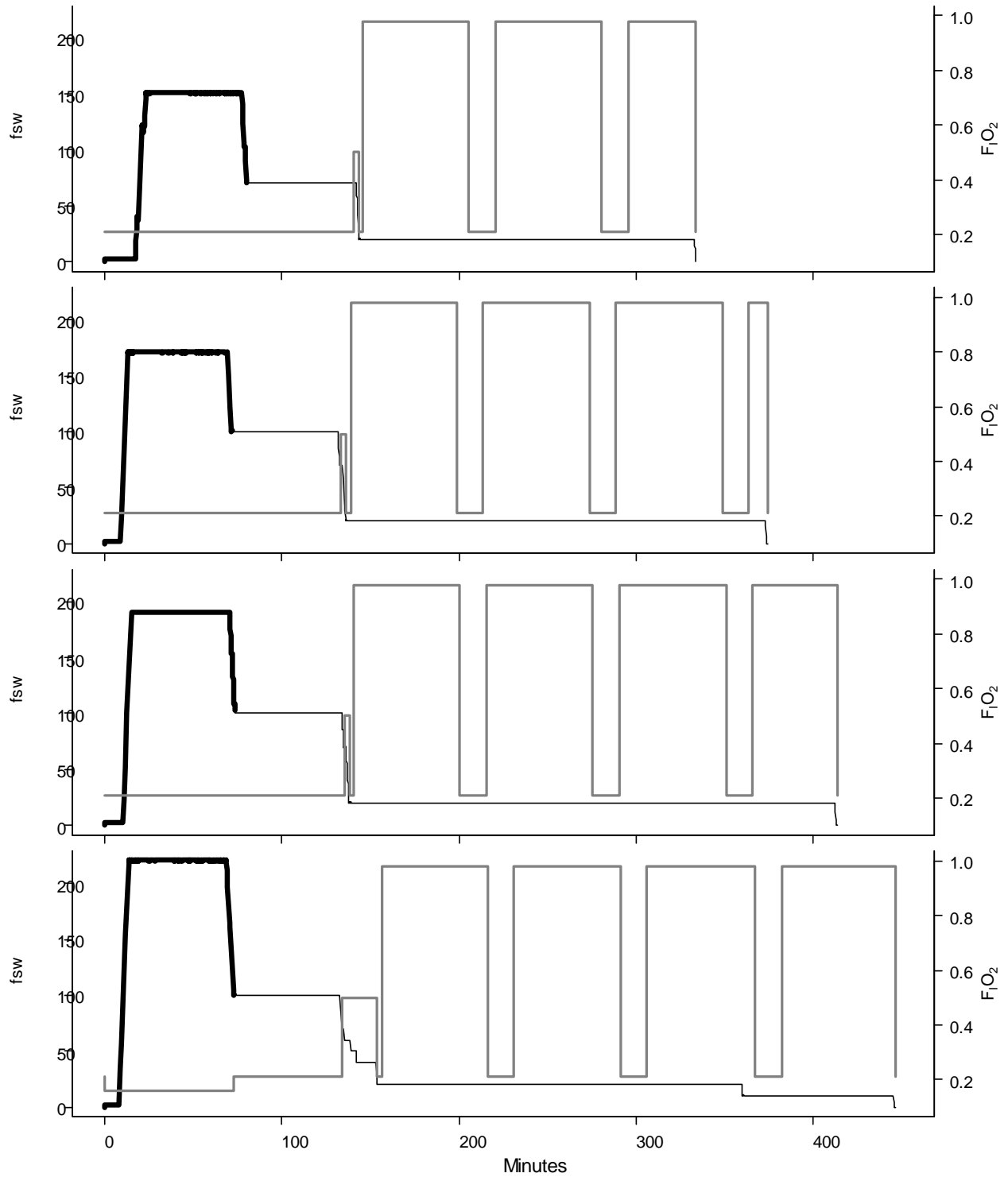


Figure A-1. Representatives of each of the four schedules, taken from Medical Deck logs. Black lines indicate depth (right axis): the heavy lines indicate the period during which divers were submerged in the wet pot and breathing heliox and the thin lines indicate the period during which divers were dry and breathing nitrox, oxygen, or chamber air. Grey lines indicate the assumed inspired oxygen fraction (right axis).

CORRECTION FOR DELAYS IN DESCENT

The 150 – 190 fsw schedules (phase 1) were conducted using a 60-minute bottom time but for phase 2, a 55-minute time on the bottom (not including the 5.5 minutes descent time) was specified. The reason for this change was that, in the event of a delay during descent, it is a simple calculation to adjust time on the bottom so that compartmental inert gas uptake is the same as for the planned schedule. The resulting small adjustments to time on the bottom were not important to the primary objective of attaining near-complete equilibration of the inner ear with breathing gas, but were considered important for testing the application of LEM-he8n25 to decompression schedules that include a heliox-to-air breathing gas switch.

Adjusted time at bottom (t_{adj}) is calculated for descent delayed by short holds at constant depths (such as to prevent an ear squeeze) but otherwise at the specified descent rate. Under these conditions, gas uptake during travel can be ignored with only a small error, so that the target tension of a single inert gas (or equivalently the sum of multiple inert gases with the same compartmental time constant [TC]) in any compartment (P_t) is approximated as:

$$P_t = P_a + (P_{hold} - P_a) \times e^{-\frac{t_{adj}}{TC}},$$

where P_a is the arterial inert gas tension on the bottom and P_{hold} is the compartmental inert gas pressure at the end of the hold.

Rearranging and inserting the solution for P_{hold} for any particular depth and duration (t_{hold}) of a hold gives:

$$t_{adj} = -\ln \left\{ \frac{P_t - P_a}{\left[P_{a_hold} + (P_0 - P_{a_hold}) \times e^{-\frac{t_{hold}}{TC}} \right] - P_a} \right\} \times TC,$$

where P_{a_hold} is the arterial inert gas tension at the hold depth and P_0 is the compartmental inert gas pressure at the beginning of the dive (at equilibrium with air at surface).

Table A-2 gives t_{adj} subtracted from the planned 55-minute time on the bottom, for holds at 10 fsw increments (deeper value indicated) and up to five minutes duration. In this range, values of t_{adj} calculated using the different compartmental time constants in the LEM-he8n25 model (range 4.7–327.7 minutes) differ by only a few seconds.

Table A-2. Decrease in time at 220 fsw to accommodate holds during descent

Hold Depth	Hold Time (minutes)				
	1	2	3	4	5
	DECREASE TIME AT BOTTOM BY:				
0-30	0	0	0	0	0
31-40	0	0	1	1	1
41-50	0	0	1	1	1
51-60	0	1	1	1	1
61-70	0	1	1	1	2
71-80	0	1	1	1	2
81-90	0	1	1	2	2
91-100	0	1	1	2	2
101-110	1	1	2	2	3
111-120	1	1	2	2	3
121-130	1	1	2	2	3
131-140	1	1	2	3	3
141-150	1	1	2	3	3
151-160	1	1	2	3	4
161-170	1	1	2	3	4
171-180	1	1	2	3	4
181-190	1	1	3	3	4
191-200	1	1	3	4	5
201-210	1	1	3	4	5
211-220	1	2	3	4	5

APPENDIX B DIVER CHARACTERISTICS

DiverID	Age*	Height (inch)	Height (m)	Weigh (lb)	Weight (kg)	Waist (inch)	Waist (m)	Neck (inch)	Neck (m)	BMI	Body Fat (%) [†]
2	33	70	1.78	197	89.4	36	0.91	16	0.41	28	19
3	37	68.5	1.74	248	112.5	39.50	1.00	16.5	0.42	37	25
4	39	70	1.78	168	76.2	36	0.91	15	0.38	24	21
5	27	71	1.80	205	93.0	38	0.97	16	0.41	29	22
6	27	67	1.70	177	80.3	35	0.89	16	0.41	28	18
7	29	75	1.90	251	113.9	44.50	1.13	17	0.43	32	29
8	29	69	1.75	180	81.6	33	0.84	16	0.41	27	14
9	40	68.5	1.74	197	89.4	40	1.02	16	0.41	30	27
10	37	67	1.70	178	80.7	35	0.89	15	0.38	28	21
11	39	66.25	1.68	176	79.8	36	0.91	15	0.38	28	23
12	20	68	1.73	174	78.9	33.50	0.85	15	0.38	26	17
13	40	69	1.75	191	86.6	36	0.91	16.5	0.42	28	18
14	46	68	1.73	160	72.6	35	0.89	15	0.38	24	20
15	35	74	1.88	201	91.2	32	0.81	16	0.41	26	9
16	39	71.25	1.81	243	110.2	40.50	1.03	17.5	0.44	34	24
17	27	73	1.85	179	81.2	31.50	0.80	15.5	0.39	24	10
18	42	73	1.85	214	97.1	39	0.99	16	0.41	28	23
19	30	70	1.78	183	83.0	35	0.89	15	0.38	26	19
20	43	69	1.75	205	93.0	37	0.94	18	0.46	30	18
21	32	73	1.85	192	87.1	34	0.86	15	0.38	25	16
22	35	67	1.70	163	73.9	33.75	0.86	15.5	0.39	26	18
23	35	71	1.80	155	70.3	32	0.81	15	0.38	22	13
24	50	69	1.75	172	78.0	35	0.89	16	0.41	25	18
25	28	70	1.78	189	85.7	35.50	0.90	14	0.36	27	21
26	30	69	1.75	175	79.4	37	0.94	14	0.36	26	25
27	34	75	1.90	219	99.3	38	0.97	16.5	0.42	28	20
28	28	73	1.85	205	93.0	35.50	0.90	16	0.41	27	17
29	45	68	1.73	186	84.4					28	
30	39	72	1.83	207	93.9	36	0.91	15	0.38	28	20
31	40	74	1.88	201	91.2	34	0.86	13.5	0.34	26	18
32	40	74	1.88	250	113.4	41	1.04	16	0.41	32	26
33	19	68	1.73	184	83.5	34.5	0.88	15	0.38	28	19
34	31	66.75	1.70	231	104.8	41	1.04	17	0.43	36	27
35	36	72	1.83	180	81.6	36	0.91	15	0.38	24	20
36	33	73	1.85	238	108.0	39	0.99	17	0.43	32	22
37	22	69	1.75	195	88.5	32	0.81			29	
38	40	71	1.80	220	99.8	37	0.94	18	0.46	31	17
39	44	69	1.75	175	79.4	33.5	0.85	15	0.38	26	17
40	30	72	1.83	210	95.3	38	0.97	17.5	0.44	28	20
41	39	74	1.88	220	99.8	35	0.89	16.5	0.42	28	15
45	44	72	1.83	210	95.3	37	0.94	16	0.41	28	20
46	42	69	1.75	174	78.9	36	0.91	14.5	0.37	26	22
47	29	73	1.85	165	74.8	33.5	0.85	14	0.36	22	17
48	28	70	1.78	190	86.2	34	0.86	16	0.41	27	15
49	42	69	1.75	202	91.6					30	

*age at first dive in this study; [†]calculated from height, waist circumference, and neck circumference according to U.S. Navy method¹⁵

APPENDIX C DIVING INTENSITY

The following tables show the dates on which each diver-subject participated in the dive trial.

DiverID	150 fsw						
	2010-06-09	2010-06-10	2010-06-14	2010-06-15	2010-06-16	2010-06-17	2010-06-21
2					X		
3		X		X			
4					X		
5					X		X
6	X		X				
7							
8						X	
9				X			
10		X					
11						X	
12					X		
13						X	
14				X			
15	X						
16	X		X				
17	X					X	
18		X					X
19		X		X			
20			X				
21							X
22							
23							
24							
25							
26							
27							
28							

DiverID	170 fsw					
	2010-07-20	2010-07-21	2010-07-22	2010-07-26	2010-07-27	2010-07-28
2		X			X	
3	X					
4						
5						
6						X
7						
8	X					
9						
10						
11		X				X
12				X		
13						
14			X			
15						
16						
17						
18						
19						
20						X
21				X		
22	X				X	
23	X					X
24		X			X	
25			X			
26				X		
27				X		
28					X	

APPENDIX D MEDICAL INCIDENTS

The tables below give the case narratives written by the attending Diving Medical Officer (DMO) for each medical incident (narratives of squeezes not given). Editorial interventions by one of the authors (DJD) are indicated by <tags>, as follows:

Tag	Meaning
<gap>	material deleted, typically description of dive
<add>...<add>	added in clarification
<corr>...<corr>	correction
<supplied>...<supplied>	material from other sources supplied by editor
<reg>...<reg>	regularized expression

Names removed and common short-hand expanded without indication.

DIVER ID 38, 15 MARCH 2011, 190 FSW SCHEDULE

<supplied>Diver complained of dizziness during first air break at 20 fsw. Second oxygen period was started on schedule but stopped after five minutes when DMO arrived to interview diver.<supplied><gap> Diver interview confirmed symptoms: approximately 30s to one minute “dizziness, unsteady gait, and nausea” after going from sitting to standing. Symptoms relieved after walking from B to A chamber. Has not noted any further exacerbation since that time. Denies pain, weakness, numbness, or tingling. Neurological examination performed by HM3 <supplied>(GREEN diver).<supplied> Cranial nerves II–XII intact. Noted eight beats nystagmus - asymptomatic. Sensory intact. Cerebellar intact. Dix-Hallpike maneuver performed - negative. Assessment: likely orthostatic intolerance; DCS unlikely. Considerable amount of water drained from diver’s ear during Dix-Hallpike maneuver <add>At postdive interview<add>diver admits <add>similar<add> “dizziness” has occurred off and on for last month upon standing. Approximately 5 beats lateral nystagmus – doubt dive related.

DIVER ID 5, 9 NOVEMBER 2011, 220 FSW SCHEDULE

<add>Principal Investigator (DJD) noted that this diver appeared uncomfortable and fidgety towards the end of the 100 fsw air breathing stop but exact time not noted. Subsequently, diver denied any symptoms at DMO interview conducted at end of 100 fsw stop (09:50 am). Following switch to 50% N₂ / 50% O₂ at 70 fsw (09:51 am) diver covered head with sweatshirt hood and seemed withdrawn. Later during an air break following an oxygen period (first air break at 20 fsw started at 11:13 am), afflicted diver admitted to BLUE diver (a DMO) that he had right-shoulder pain and identified the onset at about 10:10 am but it had now subsided.<add> <add>At post-dive interview<add> diver states that at approximately 10:10 am he had right shoulder pain. Within a few minutes the pain subsided. The diver states the actual duration was possibly 5-10

minutes. No symptoms now. Has a history of bends in same shoulder approximately one year ago. <supplied>At post-dive interview with Principal Investigator (DJD) diver recalled pain as 2/10 intensity and of a nature that would not have been noticed during an in-water dive.<supplied>

DIVER ID 39, 14 NOVEMBER 2011, 220 FSW SCHEDULE

<supplied>This diver noted left-sided neck pain during his pre-dive interview.<supplied> <add>During standard interview at the end of the 100 fsw gas switch stop,<add> diver complained of left ear "fullness" between <corr>reaching 100 fsw<corr> and Dombero <add>(otitis externa prophylaxis)<add>. <supplied>He later described this as "reduced hearing" as if he had "an ear full of water" but qualified this description as uncertain because he was unfamiliar with being under pressure in a dry chamber.<supplied> Left ear symptoms resolved during oxygen breathing decompression stops and diver had no complaints at the scheduled, 10-minute, 2-hour, and 22-hour postdive interviews.

<add>On 21 November 2011, the diver contacted the duty DMO.<add> <gap>Diver denied any signs and symptoms following the dive until Friday afternoon (18 NOV 2011), 4 days after surfacing. Friday diver noticed some "absent mindedness", 1/10 throbbing right knee pain, and unilateral left-sided headache just posterior to left ear. Headache dissipated with 800 mg ibuprofen. On Saturday headache returned and knee pain worsened to 2/10. Knee pain not increased with movement or specific position, although relief obtained briefly while resting or changing position. Diver note he ran on 16 NOV 2011 after infrequently exercising over past 2 weeks. Diver noticed some feelings of altered sensation on left face over cheek and masseter associated with the headache. Denies paresthesia or pain, just "not feeling right". Diver reported sensation intact over this area. Ibuprofen again effectively treated headache. Knee pain, left cheek altered sensation continued Sunday but has mostly resolved by time of this interview. Slight residual altered sensation localized to left lower lip. Diver denies Nausea/Vomiting/Dizziness/Fatigue/Confusion. No shortness of breath, chest pain, palpitations, abdominal pain, changes in mood, or changes in gait. Diver's wife noted diver had appeared "tired" over weekend. Physical and neurological examination was normal. Signs and symptoms not consistent with DCS I/II due to time course, with no signs or symptoms for four days ,add> following dive<add>.

DIVER ID 3, 15 NOVEMBER 2011, 220 FSW SCHEDULE

<add>Thirty-five minutes into first oxygen period at 20 fsw, diver complained of stomach ache. Oxygen breathing discontinued 15 minutes later (50 minutes total oxygen time) and diver interviewed by DMO.<add> Diver complained of generalized crampy abdominal pain and undulating nausea. No other symptoms. No numbness, tingling, weakness or vertigo. Symptoms attributed to large amount of food <supplied>cold pizza<supplied> intake at 100 fsw stop. Symptoms not relieved when oxygen breathing discontinued. Oxygen breathing continued after 25-minute air break and improved. Nausea waxed and waned as decompression continued. Diver administered Maalox as tolerated. Tolerating oxygen breathing. Reports complete resolution of all symptoms <add>after 96 minutes<add>.