

## Letter to the Editor

# **A deep stop during decompression from 82 fsw (25m) significantly reduces bubbles and fast tissue gas tensions.**

Dear Sir:

A large number of strategies for improving decompression procedures for surface oriented diving have been suggested, including deeper stops and a slower ascent rate during the deepest part of the ascent. However, if there is considerable theoretical support for this, limited experimental evidence has been presented to support the efficiency of such procedural changes. It was therefore much appreciated that Marroni et al (1) investigated the effect of different ascent rates and decompression stops on 22 recreational divers completing 16 different dives to 25 m in open water. Independent variables were 8 different combinations of bottom time (either 25 or 20 min), ascent rates (3, 10 or 18 m/min) and staged decompression stops (at 15 or 6 m for 5 min). Outcome measures were the amount of venous gas embolism monitored precordially and estimated saturation of inert gas in eight tissues with half-times calculated according to the Bühlmann algorithm. The main conclusion was that “The introduction of a deep stop during decompression ascent appears to significantly decrease Doppler

recorded bubbles and predicted gas tensions in the fast ‘tissues’ which may relate to actual gas exchange within the spinal cord.” The authors conclude that such a deep stop may therefore significantly reduce the incidence of spinal related decompression sickness.

We believe that the manuscript has a few flaws that could misdirect the reader. First of all, the title suggests that the authors have investigated a procedure that will reduce “fast tissue” gas tensions while, in fact, the authors have mathematically calculated gas tension in a “fast tissue” based on classic Haldanian theory of flow-limited gas kinetics. Fig 4b/Profile 6 shows less supersaturation in tissues with half times ( $T_{1/2}$ ) of 5 and 10 min than Fig 4a/Profile 2. However it is not easily understood whether this is due to the staged decompression at 15 and 6 m of Profile 6 or whether it was due to a total decompression time being ~50% longer in Profile 6 compared with Profile 2 (12.5 vs 8 min). Actually Table 3 shows that allowed average surfacing saturation in the tissues with  $T_{1/2}$  of 5 and 10 min are closely related to total decompression time and varies less with ascent rate and length of the staged decompression stops. We don’t see Table 3

as a convincing evidence for the effects of deep stops on “fast tissue” supersaturation at surface.

Venous gas embolism has been measured by acoustic detection of continuous Doppler ultrasound. The authors mention that the signals were scored in real time and re-investigated by a “blinded” specialist at a later time. This information is however of little use since no information is given on whether the scores from the first investigator were used or whether the specialist score was used. As pointed out by Sawatzky and Nishi (2) there is a risk of significant inter-rater disagreement. The reader should be advised by a quantitative measure (e.g. Kappa score) of the agreement between raters in this case. The authors have introduced a new VGE scoring system, the “Expanded Spencer Scale.” Firstly, it is difficult to appreciate the benefit of yet another VGE grading system, since most published work on acoustic VGE detection is based on either the Spencer or Kisman-Masurel (K-M) scoring system (3). Due to the general difficulty in scoring Doppler recordings mentioned above, fewer categories (none, few or many bubbles) should be used. While the association between decompression sickness and VGE scored with K-M grading has previously been reported (3), the introduction of a new grading system (ESS) complicates interpretation of VGE data for the reader without any obvious benefit. Rather, the use of a numerical grading system (0-4) confuses the reader, suggesting that the categorized VGE data could be transformed to a continuous variable. Secondly, the reader is invited to assume that a statistical association (between bubble grade and tissue supersaturation has been demonstrated (Figs. 2 and 3), while this conclusion is based on an, in our opinion, erroneous transformation of the categorical VGE bubble data to a continuous BSI variable. ANOVA and Kruskal-Wallis

tests assuming continuous data were incorrectly applied in Fig 1. Accordingly, a conclusion of statistical significance is drawn using an inappropriate test. A method for transforming repetitive VGE measurements into a continuous variable (KISS) has been developed as discussed by Nishi et al (3), but a statistical relationship between VGE and health outcome (DCS) has only been described using maximum VGE score as the independent variable. We believe that the readers should be given the opportunity to assess maximum VGE scores in a study like this and not only a transformation of the original data. The information provided does not allow us to evaluate whether there was a statistical difference in maximum VGE score between the different profiles tested and we are unable to conclude whether the authors’ statement in the title (a deep stop significantly reduces bubbles) is correct.

In the discussion of the 10 min tissue, the authors state, “Therefore decompression profiles may need to focus more closely on this ‘tissue’ as not only being a critical factor in the production of bubbles, but also possibly reflecting supersaturation within the spinal cord.” In the conclusion they say that “... such a deep stop may therefore significantly reduce the incidence of spinal cord related decompression sickness.” We believe that these statements invite the reader to believe that the data presented in some way suggests relationship between spinal DCS and VGE. We agree that there is a relationship between high bubble grades and DCS and that there is a relationship between a PFO and serious neurological DCS, indicating that such a relationship may exist (4,5). However, the data presented do not show any relationship between spinal DCS and VGE. We challenge the authors to document a relationship based on the data in their study.

The authors should be given credit for

the much needed effort to assess the benefit of deep stops in diving. However, we believe that the conclusions are not substantiated by the data provided or the statistical analyses used, and we would suggest proper care when disseminating the findings to the diving community, more care than shown in the recent edition of *Alert Diver* (6).

JAN RISBERG, M.D, PhD  
NUI AS and Norwegian Armed Forces  
Medical Service  
Bergen, Norway

ALF BRUBAKK, M.D., PhD  
Department of Circulation and Medical  
Imaging  
Norwegian University of Science and  
Technology  
Trondheim, Norway

#### REFERENCES

1. Marroni A, Bennett PB, Cronje FJ et al. A deep stop during decompression from 82 fsw (25 m) significantly reduces bubbles and fast tissue gas tensions. *Undersea Hyperb Med* 2004; 31:233-43.
2. Sawatzky KD, Nishi RY. Assessment of inter-rater agreement on the grading of intravascular bubble signals. *Undersea Biomed Res* 1991; 18:373-96.
3. Nishi RY, Brubakk AO, Eftedal OS. Bubble detection. In: Brubakk AO, Neuman TS, Eds. Bennett and Elliott's physiology and medicine of diving. 5th edition. London: Saunders, 2003: 501-29.
4. Moon RE, Camporesi EM, Kisslo JA. Patent foramen ovale and decompression sickness in divers. *Lancet* 1989; 1:513-4.
5. Wilmshurst P, Bryson P. Relationship between the clinical features of neurological decompression illness and its causes. *Clin Sci (Lond)* 2000; 99:65-75.
6. Marroni A. Use of a deep stop during the ascent from 25 metres could significantly reduce the incidence of DCI of the neurological type in recreational diving. *Alert Diver* 2004; 147

## REFERENCES

1. Divers Alert Network. Report on Decompression Illness and Diving Fatalities: DAN's annual review of recreational diving injuries and fatalities based on 1998 data. Durham, NC: Divers Alert Network, 2000.
2. Melamed Y, Shupak A, Bitterman H. Medical problems associated with underwater diving. *New Engl J Med* 1992; 326:30-35.
3. Clenney TL, Lassen LF. Recreational scuba diving injuries. *Am Fam Phys* 1996;53: 1761-1766.
4. Greer HD, Massey EW. Neurologic injury from undersea diving. *Neurol Clin* 1992;10: 1031-1045.
5. Newton HB. Neurologic complications of scuba diving. *Am Fam Phys* 2001;63: 2211-2218.
6. Todnem K, Nyland H, Skeidsvoll H, et al. Neurological long term consequences of deep diving. *Br J Indian Med* 1991;48: 258-266.
7. Barratt DM, Harch PG, Van Meter K. Decompression illness in divers: A review of the literature. *The Neurologist* 2002;8: 186-202.
8. U. S. Navy. Recompression treatments when chamber available. U. S. Navy Diving Manual volume 1 (Air Diving). Revision 1, ch. 8, rev. 15. February 1993; Naval Sea Systems Command Publication NAVSEA 0994-LP-001-9110.
9. Miller NR. Walsh and Hoyt's clinical neuro-ophthalmology. Vol 3. Baltimore: Williams & Wilkins, 1985:652-682.
10. Yamana T, Murakami N, Itoh E, Takahashi A. Weber's syndrome of ischemic vascular origin – a clinical and neuroradiologic study. *No To Shinkei* 1993;45: 349-354.
11. Brazis PW. Localization of lesions of the oculomotor nerve: Recent concepts. *Mayo Clin Proc* 1991;66: 1029-1035.
12. Bennett JL, Pelak VS. Neuro-ophthalmology. Palsies of the third, fourth, and sixth cranial nerves. *Ophthalmol Clin N. Am.* 2001;14: 169-185.
13. Parent A. Carpenter's human neuroanatomy. Ninth Edition. Baltimore: Williams & Wilkins, 1996:469-582.
14. Levin HS, Goldstein FC, Norcross K, Amparo EG, Guinto FC, Mader JT. Neurobehavioral and magnetic resonance imaging findings in two cases of decompression sickness. *Aviat Space Environ Med* 1989;60: 1204-1210.
15. Hutzelmann A, Tetzlaff K, Reuter M, Müller-Hülsbeck S, Heller M. Does diving damage the brain? MR control study of divers' central nervous system. *Acta Radiologica* 2000;41: 18-21.
16. Brown RD, Evans BA, Wiebers DO, Petty GW, Meissner I, Dale AJD. Transient ischemic attack and minor ischemic stroke: An algorithm for evaluation and treatment. *Mayo Clin Proc* 1994;69: 1027-1039.
17. U. S. Department of the Navy. 1999. U. S. Navy Diving Manual. Revision 4. NAVSEA 0901-LP-708-8000. Naval Sea Systems Command, 2531 Jefferson Davis Hwy. Arlington, VA 22242-5160.
18. Walker R. Pulmonary Barotrauma. In: Edmonds C, Lowry C, Pennefather J, Walker R, eds. Diving and subaquatic medicine. Fourth edition. London: Arnold, 2002:55-71.
19. Aksenov V, Parsa N, Nicholson W, Saba GW. Arterial gas embolism in tender during treatment of decompression sickness: Case report (abstract). *Proc Undersea Hyperbar Med Soc Meeting* 2002;82.
20. Bernard N, Grammond P, Lecours R. Double cerebral air gas embolism (CAGE): Lessons from commercial flight and hyperbaric oxygen therapy complications (abstract). *Proc Undersea Hyperbar Med Soc Meeting* 2003;3.
21. Moon RE. Treatment of diving emergencies. *Critical Care Clin* 1999;15: 429-456.
22. Dick APK, Massey EW. Neurologic presentation of decompression sickness and air embolism in sport divers. *Neurol* 1985;35: 667-671.
23. Boussuges A, Blanc P, Molenat F, Bergmann E, Sainty JM. Haemoconcentration in neurological decompression illness. *Int J Sports Med* 1996;17: 351-355.