

Original articles

Observation of increased venous gas emboli after wet dives compared to dry dives

Andreas Møllerløgken, Toni Breskovic, Ivan Palada, Zoran Valic, Zeljko Dujic and Alf O Brubakk

Key words

Bubbles, decompression sickness, diving tables, diving research

Abstract

(Møllerløgken A, Breskovic T, Palada I, Valic Z, Dujic Z, Brubakk A. Observation of increased venous gas emboli after wet dives compared to dry dives. *Diving Hyperb Med.* 2011;41(3):124-8.)

Introduction: Testing of decompression procedures has been performed both in the dry and during immersion, assuming that the results can be directly compared. To test this, the aim of the present paper was to compare the number of venous gas bubbles observed following a short, deep and a shallow, long air dive performed dry in a hyperbaric chamber and following actual dives in open water.

Methods: Fourteen experienced male divers participated in the study; seven performed dry and wet dives to 24 metres' sea water (msw) for 70 minutes; seven divers performed dry and wet dives to 54 msw for 20 minutes. Decompression followed a Bühlmann decompression procedure. Immediately following the dive, pulmonary artery bubble formation was monitored for two hours. The results were graded according to the method of Eftedal and Brubakk.

Results: All divers completed the dive protocol, none of them showed any signs of decompression sickness. During the observation period, following the shallow dives, the bubbles increased from 0.1 bubbles per cm² after the dry dive to 1.4 bubbles per cm² after the wet dive. Following the deep dives, the bubbles increased from 0.1 bubbles per cm² in the dry dive to 2.4 bubbles per cm² in the wet dive. Both results are highly significant ($P = 0.0001$ or less).

Conclusions: The study has shown that diving in water produces significantly more gas bubble formation than dry diving. The number of venous gas bubbles observed after decompression in water according to a rather conservative procedure, indicates that accepted standard decompression procedures nevertheless induce considerable decompression stress. We suggest that decompression procedures should aim at keeping venous bubble formation as low as possible.

Introduction

Testing of decompression tables is often done with the binominal end-point of decompression sickness (DCS) or no-DCS. This binominal, DCS/no-DCS, outcome is dependent on the clinical judgment of the investigator performing the medical investigation after test dives, and the signs and symptoms reported by the diver. It is commonly accepted that the primary cause of DCS is gas bubbles in blood and tissues. Such bubbles can be measured using ultrasound. Ultrasound, both Doppler and imaging, has been proven valid for indicating the risk of DCS after dives, and is also commonly used to indicate the level of stress that the diver has been exposed to.^{1,2} The use of ultrasound is also attractive because it allows the number of dives needed to validate a procedure to be markedly reduced when using detection of venous gas emboli (VGE) as the end-point.³

The aim of the present study was to compare the number of VGE detected following a short, deep air dive and a shallow, long air dive in the same individuals in two different environments. All dives were performed by recreational divers and both profiles were tested both in a dry hyperbaric chamber and in open water. The decompression procedure

tested was a Bühlmann diving algorithm, which, because of its long decompression obligations, has been considered one of the more conservative decompression algorithms.⁴

Methods

STUDY POPULATION

All experimental procedures were conducted in accordance with the Declaration of Helsinki, and were approved by the Ethics Committee of the Split School of Medicine, Croatia. All procedures and potential risks were explained to the participants in detail and they all gave written informed consent before the experiments. None of the divers participated in any other diving activity for a minimum of seven days before the start of the experiment.

A total of 14 experienced, male recreational divers were randomly divided into two groups ($n = 7$ in each). All had a valid medical certificate for diving and were clear of all symptoms of acute illness. Two of the divers were smokers (10 and 15 cigarettes per day), but all lung function values were within normal ranges (Table 1).

Table 1
Overview of the physiological characteristics of the divers;
FVC - forced vital capacity; FEV₁ - forced expiratory
volume in 1 sec

	Median	Range
Age (years)	34.1	26–46
BMI (kg m ⁻²)	26.7	23.8–32.6
FVC (% predicted)	115.1	102.2–139.2
FEV ₁ (% predicted)	106.3	88.5–127.8
FEV ₁ /FVC ratio (% predicted)	96.5	78.9–109.2

DIVING PROTOCOLS

Two different dive protocols were compared in this study. One deep, short dive (54 metres’ sea water (msw) with 20 min bottom time) and one shallow, long dive (24 msw for 70 min bottom time). Compression rates were 10 msw min⁻¹, and decompression rates 9 msw min⁻¹. The decompression profile for the deep dive consisted of one stop for 1 min at 12 msw for 1 min, 5 min at 9 msw, 9 min at 6 msw and 27 min at 3 msw before ascent to the surface. In the shallow, long dive, the decompression consisted of a decompression stop at 6 msw for 22 min followed by a stop at 3 msw for 46 min before reaching the surface.

Both profiles were tested first in a dry hyperbaric chamber and then under field conditions (wet dives). During the chamber trials, the divers were seated resting at room temperature (approximately 20°C), performing no exercise. During the field trials, conducted in Split, Croatia, the divers were equipped with personal 7 mm neoprene wet suits, the water temperature being 16–18°C. Each diver was supplied with a dive computer (Galileo™, UWATEC, Switzerland) for verification of the dive profiles and monitoring of heart rate (HR). No exercise was performed during the bottom and decompression phases apart from any swimming needed to stay in position. No noticeable currents were recorded. Each diver performed two dives, one week apart. Seven divers completed matched dry- and open-water shallow dives and seven others completed matched dry- and open-water deep profiles. The sequence of the dives was the same under both dive conditions.

POST-DIVE MONITORING AND BUBBLE ANALYSIS

Observations for VGE commenced within 5 minutes following hyperbaric chamber exposures, while after the

field dives, the divers were transported to the on-shore diving facility by boat, and it took approximately 15 min to begin VGE monitoring. The divers were placed in the supine position and a phase-array ultrasonic probe (1.5–3.3 MHz) was positioned to obtain a clear view of all four chamber walls of the heart. The transducer was connected to a Vivid 3 Expert ultrasonic scanner (GE, Milwaukee, USA). The same, experienced cardiologists performed all echocardiographic investigations. Monitoring was performed every 20 min after reaching the surface for a total period of 2 hours, giving six recordings for each diver after each dive. Bubbles were observed in the pulmonary artery and the right ventricle as high intensity echoes. The cardiac images were recorded on S-VHS videotape for 60 seconds at rest and after two coughs. The bubbles were graded using the method described by Eftedal and Brubakk.⁵ This grading system has been used extensively in several animal species as well as in man. The grading system uses the following definition:

- 0 – no bubbles;
- 1 – occasional bubbles;
- 2 – at least one bubble per 4th heart cycle;
- 3 – at least one bubble per cycle;
- 4 – continuous bubbling, at least one bubble cm⁻² in all frames;
- 5 – “white-out”, individual bubbles cannot be seen.

High-quality images were obtained in all subjects. The data were saved on tape and digitized on a personal computer (ATI Multimedia Center, ATI Technologies, USA). After grading, the values were transferred to a linear scale (bubbles per cm²) as described previously.⁶ The number of bubbles was determined at the end of each of the measurement points, and an average bubble number for the whole observation period was obtained. The divers were carefully monitored and asked about any symptoms of decompression sickness (DCS) by a diving medical specialist.

STATISTICAL ANALYSIS

Data are presented as median (range) and mean ± standard deviation (SD). Inter-dive comparisons of bubble grade were done with Mann-Whitney U tests. Differences in bubble grade, expressed as bubbles per cm², and HR between dry dives and in-water dives were compared using a Student t-test for unpaired samples. Statistical significance was set at *P* < 0.05. All analyses were done using Statistica 7.0 software (Statsoft Inc., Tulsa, USA).

Table 2
Changes in the amount of VGE during deep and shallow dives; * – *P* = 0.0001

	54 msw/20 min		24 msw/70 min	
	Dry dive	In-water dive	Dry dive	In-water dive
Median bubble grade (range)	0.0 (0–3)	3.0 (0–4)	0.0 (0–3)	2.0 (0–4)
Mean bubbles per cm ⁻² (SD)	0.1 (0.3)	2.4 (2.6)*	0.1 (0.3)	1.4 (2.0)*

Table 3
Comparison of the different decompression tables (in minutes) for the shallow-long dive (24 msw/70 min);
total decompression time does not include swimming time between stops

Depth (msw)	Bühlmann	US Navy	Norwegian	DCIEM
6	22	-	5	7
3	47	23	20	30
Total decompression (min)	69	23	25	37

Table 4
Comparison of the different decompression tables (in minutes) for the deep-short dive (54 msw/20 min);
total decompression time does not include swimming time between stops

Depth (msw)	Bühlmann	US Navy	Norwegian	DCIEM
12	2	-	-	6
9	5	1	5	6
6	9	5	10	8
3	27	17	15	25
Total decompression (min)	43	23	30	45

Results

All subjects completed both the wet and dry protocols without any symptoms or signs of DCS. Bubbles were observed in the right heart only (Table 2). The mean bubble number over the whole observation period increased from 0.1 bubbles per cm² after dry dives to 1.4 bubbles per cm² after wet dives on the shallow profile and from 0.1 to 2.4 bubbles per cm² on the deep profile ($P = 0.0001$ and $P < 0.0001$ respectively).

During the bottom phase of the in-water dives, the divers were instructed not to do any strenuous exercise, as is supported by HR data. The mean HR during the bottom phase was similar during the 24/70 dive (91.2 ± 15.6 beats per minute, bpm) and the 54/20 dive (93.6 ± 13.6 bpm) ($P = 0.8$). Heart rates from the dry chamber dives were not recorded.

The decompression obligation in the shallow, long dive is largest following the Bühlmann decompression table, compared to other tables (Table 3). For the short, deep dive, the Bühlmann decompression table gave almost the same obligations as the DCIEM table (Table 4).

Discussion

In this study, the number of VGE detected was used as an indicator of decompression stress after two different dive profiles tested in both a dry hyperbaric chamber and in open-water conditions. None of the dry dives led to large numbers of VGE being detected in any of the divers. However, when performing the same dives in water, the number of VGE increased dramatically. Despite this increase in decompression stress related to the amount of VGE, none of the divers developed any symptoms or signs of DCS.

There are obviously considerable haemodynamic differences between sitting in a dry hyperbaric chamber and swimming in water. At present, we have no good explanation as to why these differences should lead to differences in bubble formation. Even using wet-suits, divers in water will probably be colder during both the bottom and the decompression phase of the dives than divers resting at room temperature. In fact, an increase in bubble scores has been observed in warm conditions compared to cold conditions.⁷ It was suggested that cold-induced peripheral vasoconstriction reduced inert gas uptake and hence the number of bubbles. A study which measured both DCS as an endpoint and VGE suggested beneficial effects of warm conditions during decompression (enhanced off-gassing from tissues) compared with deleterious effects of warm conditions during the bottom time (enhanced on-gassing during deepest part of the dive).⁸ We did not monitor the core or skin temperatures of our divers, which is a clear weakness of this study. Regardless of temperature differences, we were surprised that immersion resulted in such a significant increase in the numbers of VGE following the dive.

In the development of the DCIEM air tables, schedules with >50% incidence of Kisman Masarel (KM) grading system grade 2 bubbles or higher were rejected.⁹ In relation to the findings in the present study, the in-water schedules would both have been rejected. Initial experiments prior to the testing of the two dive profiles, together with the knowledge that Bühlmann procedures have apparently been used safely for many years, convinced us that the procedures we tested were safe to test on humans. The Bühlmann diving algorithm is recognised as one of the more conservative decompression algorithms. As can be seen in Tables 3 and 4, there are large differences between different decompression tables regarding these two specific dives. The first dive in the dry hyperbaric chamber supported this view. When the same procedure was tested in water in the same divers,

considerably higher bubble grades were observed. The present study points in the same direction as a previous study by Weathersby et al.¹⁰

The traditional endpoint for testing of decompression procedures has been the occurrence of DCS, based on the assumption that procedures that give no symptoms of DCS will have no effect upon the health of the individual. This outcome is dependent on the clinician performing the clinical investigation, as well as the symptoms reported and signs elicited from the diver. A challenge using VGE as a stress indicator after dives is that there is a large individual variability; the same dive can result in many bubbles or none at all, and the response to bubbles may also differ. At an Undersea and Hyperbaric Medicine Society 1989 workshop, the validation of decompression procedures was discussed in detail.¹¹ The workshop concluded that validation should primarily involve extensive, dedicated laboratory testing before putting the procedures into the field for “operational evaluation”. By using ultrasound to detect VGE after decompression, the extent of the laboratory testing will be dramatically reduced compared to testing with the binominal endpoint DCS or no-DCS. Data from Sawatzky suggests that the absence of detectable bubbles is a good indicator of decompression safety.¹ In recent years, ultrasonic imaging systems have become more available and have been shown to be well suited to the detection of VGE.¹² While the ultrasonic Doppler method requires extensive training, both with regards to the monitoring itself and to the interpretation of the Doppler signals, the use of ultrasound imaging techniques requires less training and it has been demonstrated that the bubble grades from the different detection methods can be directly compared at rest.^{6,12,13}

Recently, a reduction in ventricular and arterial endothelial function has been observed in divers when they returned to the surface.^{14,15} This subclinical, asymptomatic alteration in cardiovascular function lasted up to three days after a single air dive, but was partially reversible with pre-dive antioxidant administration (vitamins C and E).¹⁶ Thus, although high in this study, bubble scores after diving at the limits of the Bühlmann table do not appear to be predictive of DCS; their effects on cardiovascular and biochemical function should be studied in more detail to find the adverse effects of VGE. Such studies should also include the fitness level of the divers, as this has been reported to be negatively correlated with bubble formation after a dive.¹⁷

Hennessy and Hempleman postulated in their model for estimating risk in dives that $p\sqrt{t}$ (where p is absolute pressure in bar and t is time in minutes) can be used for evaluating dive stress.¹⁸ The success of this model was demonstrated by Shields, who pointed out the inadequacy of existing tables to provide sufficient decompression as the pressure-duration exposure increases.¹⁹ In North Sea professional divers, the incidence of DCS from air dives increased sharply above a $p\sqrt{t}$ value of 25.²⁰ That has led to a depth/duration limitation

of nitrogen/oxygen dives to less than these levels for all professional dives in the United Kingdom.¹⁹

In the present study, the dives had a $p\sqrt{t}$ of 28.6 (54/20) and 28.8 (24/70) respectively. Despite the fact that both dive profiles in this study were above a $p\sqrt{t}$ 25 value, and generated a fair amount of VGE when conducted in water, no DCS occurred. This observation suggests that one should look for other possible test criteria than DCS/no-DCS when analysing a decompression schedule. However, these observations are based on a very small data set.

In Tables 3 and 4, the decompression requirements of four commonly used decompression tables are compared. The results from the present study would seem to indicate that the decompression requirements of most tables may be inadequate and could expose divers to considerable decompression stress in the extreme depth/duration ranges studied here. VGE is a practical way to evaluate decompression procedures, given the sometimes difficult clinical diagnosis of DCS, and we would suggest that less than grade III in 80% of a representative group of divers would be a useful set of criteria to distinguish acceptable dives from stressful dives.

Acknowledgements

This study was supported by StatoilHydro, Philips Norge and the Norwegian Oil Directorate through the programme Research and Development in Diving, contract no. 4600002328, the Norwegian Underwater Intervention and Croatian Ministry of Science, education and sports No 216-2160133-0130. UWATEC AG, Switzerland is acknowledged for computer support. We thank Duska Glavas, Ante Obad, Darija Bakovic and Sergio Angelini for their support and advice with this project.

References

- 1 Sawatzky KD. *The relationship between intravascular Doppler-detected gas bubbles and decompression sickness after bounce diving in humans*. MSc Thesis, Toronto, Ontario: York University; 1991.
- 2 Eftedal OS, Lydersen S, Brubakk AO. The relationship between venous gas bubbles and adverse effects of decompression after air dives. *Undersea Hyperb Med*. 2007;34:99-105.
- 3 Eftedal OS, Tjelmeland H, Brubakk AO. Validation of decompression procedures based on detection of venous gas bubbles: A Bayesian approach. *Aviat Space Environ Med*. 2007;78:94-9.
- 4 Bühlmann AA. *Decompression – decompression sickness*. Berlin; Springer Verlag, 1984.
- 5 Eftedal O, Brubakk AO. Detecting intravascular gas bubbles in ultrasonic images. *Med Biol Eng comput*. 1993;31:627-33.
- 6 Nishi RY, Brubakk AO, Eftedal OS. Bubble detection. In: Brubakk AO, Neuman TS, editors. *Bennett and Elliott's physiology and medicine of diving*, 5th ed. Toronto: Saunders; 2003. p. 501-30.
- 7 Dunford R, Hayward J. Venous gas bubble production

- following cold stress during a no-decompression dive. *Undersea Biomedical Research*. 1981;8:41-9.
- 8 Gerth WA, Ruterbusch VL, Long ET. *The influence of thermal exposure on diver susceptibility to decompression sickness*. Technical report 06-07. Panama City: Office of Naval Research; 2007. p. 70. Available from: < <http://archive.rubicon-foundation.org/5063>>
 - 9 Nishi RY, Eatock BC. The role of bubble detection in table validation. In: Schreiner HR, Hamilton RW, editors. *Validation of decompression tables. The 37th Undersea and Hyperbaric Medical Society Workshop*. Bethesda, MA: Undersea and Hyperbaric Medicine Society; 1989. p. 133-8.
 - 10 Weathersby PK, Survanshi SS, Nishi RY. Relative decompression risk of dry and wet chamber air dives. *Undersea Biomedical Research*. 1990;17:333-52.
 - 11 Hamilton RW, Schreiner HR. Editorial summary: validation of decompression tables. In: Schreiner HR, Hamilton RW, editors. *Validation of decompression tables. The 37th Undersea and Hyperbaric Medical Society Workshop*. Bethesda, MA: Undersea and Hyperbaric Medicine Society; 1989. p. 163-7.
 - 12 Eftedal O, Brubakk AO. Agreement between trained and untrained observers in grading intravascular bubble signals in ultrasonic images. *Undersea Hyperb Med*. 1997;24:293-9.
 - 13 Brubakk AO, Eftedal O. Comparison of three different ultrasonic methods for quantification of intravascular gas bubbles. *Undersea Hyperb Med*. 2001;28:131-6.
 - 14 Dujic Z, Obad A, Palada I, Valic Z, Brubakk AO. A single open sea air dive increases pulmonary artery pressure and reduces right ventricular function in professional divers. *Eur J Appl Physiol*. 2006;97:478-85.
 - 15 Brubakk AO, Duplancic D, Valic Z, Palada I, Obad A, Bakovic D, et al. A single air dive reduces arterial endothelial function in man. *J Physiol*. 2005;566(Pt 3):901-6.
 - 16 Obad A, Palada I, Valic Z, Ivancev V, Bakovic D, Wisloff U, et al. The effects of acute oral antioxidants on diving-induced alterations in human cardiovascular function. *J Physiol*. 2007;578(Pt 3):859-70.
 - 17 Carturan D, Boussuges A, Vanuxem P, Bar-Hen A, Burnet H, Gardette B. Ascent rate, age, maximal oxygen uptake, adiposity, and circulating venous bubbles after diving. *J Appl Physiol*. 2002;93:1349-56.
 - 18 Hennessy TR, Hempelman HV. An examination of the critical released gas volume concept in decompression sickness. *Proc R Soc Lond B Biol Sci*. 1977;197:299-313.
 - 19 Shields T, Duff P, Lee W, Wilcock S. *Decompression sickness from commercial offshore air-diving operations on the UK continental shelf during 1982 to 1986*. Aberdeen: Robert Gordon's Institute of Technology; 1989. UK Health and Safety Executive; <<http://www.hse.gov.uk/diving/acop.htm>>
 - 20 Shields TG, Lee, WB. *The incidence of decompression sickness arising from commercial offshore air-diving operations in the UK sector of the North Sea during 1982/83*. Sheffield: Health and Safety Executive; 1997. p. 95.

Submitted: 27 January 2011

Accepted: 21 July 2011

Andreas Møllerløkken, PhD, Department of Circulation and Medical Imaging, Faculty of Medicine, Norwegian University of Science and Technology, Trondheim, Norway
Toni Breskovic, MD, PhD, Ivan Palada, MD, PhD, Zoran Valic, MD, PhD, and Professor Zeljko Dujic, MD, PhD, Department of Physiology, University of Split School of Medicine, Split, Croatia.

Alf O Brubakk, MD, PhD, Professor Emeritus, Department of Circulation and Medical Imaging, Norwegian University of Science and Technology, Norway.

Address for correspondence:

*Andreas Møllerløkken
 Department of Circulation and Medical Imaging
 Norwegian University of Science and Technology
 Postbox 8905, Medisinsk teknisk forskningscenter
 7491 Trondheim, Norway*

Phone: +47-(0)72-828071

Fax: +47-(0)72-828372

E-mail: <andreas.mollerlokken@ntnu.no>