Diving and Hyperbaric Medicine

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'Yo-yo' diving in the aquaculture industry

Echocardiography gives new insights into HBO physiology

Transcutaneous oxygen values in legs of healthy subjects

Grommets for middle ear barotrauma in HBOT patients

Danish physicians want more evidence for HBOT in ORN

Splenic platelets may not contribute to DCS in rats

Cortisol changes after warm-water scuba dives

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PURPOSES OF THE SOCIETIES

To promote and facilitate the study of all aspects of underwater and hyperbaric medicine
To provide information on underwater and hyperbaric medicine
To publish a journal and to convene members of each Society annually at a scientific conference

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The Editor's offering

Our last issue contained a series of articles and an editorial on applications of the principles of ultrasonography in enhancing our understanding of the pathophysiology of decompression and decompression stress. This theme is taken further in this issue in which ultrasonography is used to investigate decompression stress in working divers and to assess the effects of hyperbaric oxygen on cardiac physiology.[1,2] Smart et al have taken ultrasonography into the aquaculture diver's workplace to assess the risks and contribute to the safe management of what is known as 'yo-yo' diving.[1] As a result of this research, they have developed, in cooperation with the Tasmanian aquaculture industry itself, new decompression rules for this type of diving, that have markedly reduced the risks of decompression sickness and enormously enhanced productivity in relation to diving operations in that industry. This ground-breaking work will have, I believe, important implications for diving operations in the huge international seafood farming industry, not just salmon farming.

Recently three important aspects of scientific publication have been raised for myself and the Editorial Board (EB) to consider. Firstly the appropriate use of statistics is an important ethical component of good research. No study should be embarked upon without considering the analysis to be used and assessing the sample sizes needed to investigate an hypothesis effectively. All too often studies are of insufficient power to satisfactorily address the null hypothesis underlying the research. Researchers, whether in the laboratory or in a clinical setting, are strongly encouraged to seek professional help from a biostatistician for this. In much of my own research career, my biostatistical colleagues have played an integral part in the design, analysis and reporting of research (my own knowledge of biostatistics being at the somewhat basic level of many of my medical colleagues), such that their names appear as co-authors.

In the letters column, the statistical analysis of the study on the effects of vinegar on discharged nematocycts of *Chironex fleckeri* is strongly challenged and this rebutted by the authors.[3] Independent biostatistical advice was also sought. This all highlighted the diverse views of biostatisticians to a single problem. Nevertheless, the data in that paper suggests that it should be regarded as preliminary work that, because of its potentially important implications for first-aid care of these envenomations, clearly requires further laboratory and clinical research. Unfortunately this paper resulted in sensationalist, erroneous reporting in the Australian media that was picked up internationally. Authors need to take great care in how they report their work to the public at large.

The second issue discussed recently is that of multiple authorship. *Diving and Hyperbaric Medicine* follows the recommendations regarding authorship of the International Committee of Medical Journal Editors (ICMJE).[4] All authors should meet the ICMJE's four criteria for authorship and those who do not should be acknowledged. The ICMJE document also states that "*It is the collective responsibility of the authors, not the journal to which the work is submitted, to determine that all people named as authors meet all four criteria; it is not the role of journal editors to determine who qualifies or does not qualify for authorship or to arbitrate authorship conflicts.*" Nevertheless, a journal needs some reassurance that this determination has been made appropriately. DHM also requires that there be strong justification for more than six (6) authors and may seek further information from co-authors. Anyone undertaking research should be familiar with the full ICMJE guidelines.

Thirdly in the past few years we have received potentially publishable clinical studies, including one prospective RCT, for which the authors had not sought ethical approval. DHM also regularly receives individual case reports and case series in which patient consent to publish their medical details, however anonymously, has not been obtained. Clinicians are reminded that prior ethics approval and/or patient consent are essential prerequisites for acceptance for publication.

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Michael Davis

Erratum

In *Diving and Hyperbaric Medicine* 2014 June;44(2):63, the title of Professor Costantino Balestra's editorial is incorrect in referring to "*nitrous oxide*". It should read: "*Just say NO to decompression bubbles: is there a real link between nitric oxide and bubble production or reduction in humans?*" We apologise for this error in the print issue, which has been corrected on Medline, SciSearch and Embase/Scopus.

Photo of *Arctocephalus pusillus*, the Australian fur seal, under an Atlantic Salmon pen in Tasmania. Reproduced with kind permission from Jon Bryan Photography, Newnham, Tasmania.

The Presidents' pages

Is there a need for more diving science for divers?

Costantino Balestra, President EUBS

Decompression illness (DCI)/dysbaric disorders represent a complex spectrum of pathophysiological conditions with a wide variety of signs and symptoms related to dissolved gas and its subsequent phase change.[1,2] Any significant organic or functional decrement in individuals who have recently been exposed to a reduction in environmental pressure (i.e., decompression) must be considered as evidence of DCI until proven otherwise. However, apart from the more obvious acute manifestations, individuals who have experienced repetitive exposures (e.g., commercial or professional divers and active recreational divers) may also develop sub-acute or chronic manifestations sub-clinically – insidious, even if subtle, and almost symptomless.[3]

It is, in fact, generally accepted that sub-clinical forms of DCI exist, with little or no reported symptoms, and that these may cause changes in the bones, the central nervous system and the lungs. All this has led us to analysing 'decompression stress', the actual way of understanding decompression. Current research into decompression sickness (DCS) is focused on biological markers that can be detected in the blood. Investigators are exploring the potential association between decompression stress and the presence of membrane microparticles (membrane-bound vesicles shed from a variety of cell types) in the blood.[4–6]

Microparticle levels increase in association with many physiological disease states as well as with the shearing stress caused by bubbles in the blood. The working hypothesis is that certain microparticles (possibly induced by inert gas bubbles) may initiate, be a marker of or contribute to the inflammatory response that leads to DCS. This investigation goes beyond the pure bubble model. While bubbles in the blood certainly play a key role in the development of DCS, their presence or absence does not reliably predict DCS symptom onset. Investigating this process at the molecular level may teach us a great deal more about DCS, providing insights that we hope will improve the effectiveness of both prevention and treatment.

Approaches to evaluating decompression stress have considered a wide range of 'markers': different physiological changes after the dive (flow mediated dilatation reduction, blood pressure); personal susceptibility (VO_{2max}, age); environmental factors (altitude, temperature); various physiological states (dehydration, increased vascular resistance as well as bubble counts, predictive decompression models, etc., etc. All this shows how far today's approach to decompression is removed from 'traditional' understanding. It reflects both the need to consider the phenomenon of decompression in a different way than previously and the advances in knowledge over the past 20 years of diving science research.

The 14 researchers who have been working for three years under the PHYPODE European Project reached a point where they felt the need to publish a new book in English to allow divers to learn more about the modern approaches to understanding decompression and its problems. Almost every young scientist participating in the PHYPODE project had the responsibility of writing a chapter. This was by no means a simple job considering the different linguistic origins of this group of young researchers, many of whom had their own doctoral theses or research programmes to complete in parallel. Authors also include renowned and established scientists and diving medicine specialists. The intended readership is divers, as well as medically or scientifically educated individuals, interested in increasing their knowledge of the science behind diving and decompression.

One may question this project considering the huge amount of information available on the internet on such a topic. Let us illustrate our motivation by means of a story from Japan where one of the major cosmetic companies received a customer complaint because he received an empty soap box. They launched a huge investigation and discovered that the defect arose in the packaging department. The plan was to develop a robust and reliable system ensuring zero defects in the process of product packaging and the company invested heavily in the design and implementation of a solution. A few weeks later, a similar problem occurred in a small soap-manufacturing company in India. This time the approach was very different. The manufacturer bought a big industrial fan and placed it facing the soap box chain. Boxes that were empty simply blew off the chain and the rest moved ahead to the storage house!

Our aim was to keep the concepts as clear as possible but maintain the scientific integrity of the subject. References are limited and proposed as further reading. As many of those conceiving some of the new approaches are authors, this is our opportunity to be the "*fan that blows empty boxes*".

As the PHYPODE Project has no means to receive profits from book sales or rights, the book will be published under the name of EUBS/PHYPODE, with EUBS being the beneficiary. The tentative title could be "*Diving science for divers – What your diving instructor never told you"*. The final editor has to be decided during the Excom meeting in Wiesbaden and the book will then be published shortly after.

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Key words
Medical society, diving research, decompression sickness, general interest

David Smart, President SPUMS

This is my first report as President of SPUMS. I am humbled and honoured to have been elected President and there is a very high standard to maintain after the distinguished service provided by my predecessor, Associate Professor Mike Bennett. I would first of all like to congratulate Mike for his achievements whilst at the helm of SPUMS. Mike has led the Society through some turbulent times, and there is no doubt SPUMS is now a stronger and more robust organisation, with better governance structures. SPUMS also has a sound financial base, a credit to the previous executive. It has not been necessary to raise membership fees in the next financial year. Mike has also overseen the closer relationship with EUBS, co-publishing the *Diving and Hyperbaric Medicine* Journal.

There is still much to be done to continue growing the relationship with our European colleagues. We are in the process of establishing a joint Journal Committee, so that there is a clear structure for the governance of the Journal. There is also work being undertaken to create at least one point of parity in diving medical training for physicians across Europe and the South Pacific. There are considerable differences in the processes of training in diving and hyperbaric medicine, ranging as they do from short courses to clinical attachments, web-based packages, university- and college-based courses. All have merits, but there are points of commonality to work with in order to achieve some form of mutual professional recognition.

The incoming executive committee has an immediate task – to revise the SPUMS Purposes and Rules to match new "*Model Rules*" from Consumer Affairs Victoria (CAV). This has commenced and, although not onerous, will require some careful negotiation of the necessary processes. Another task is to grow our membership. Hopefully we have turned the corner after a number of years of contraction of membership as the past year has seen an increase in numbers. I would encourage all members to approach colleagues who share mutual interests and recommend they join SPUMS. There are membership brochures downloadable from the web site.

I would also offer my congratulations to Mike Davis for the sterling job he is doing as Editor of the Journal. It is a quality production, managed on the barest of budgets, and has risen in Impact Factor to become the premier journal of its type in the world. In addition, Mike has had terrific support from his assistant, Nicky McNeish in producing the Journal. Nicky has also been instrumental in improving the SPUMS website. We are in need of renewal and further improvement to the website, a task for the SPUMS Executive to navigate over the next year or two. Hopefully this can be a multipurpose site to serve our members better; combining subscriptions, administrative processes, education, and multiple other functions.

Finally, I had the privilege to attend another SPUMS ASM, this year in Bali, and I offer my congratulations and thanks to Neil Banham and his colleagues who convened the meeting. It was successful in all areas; scientific content, practical diving workshops, standard of accommodation and amenities and the social programme. In addition, numbers of delegates this year were 50% higher than recent trends. Well done Neil, you have set a very high benchmark for future convenors.

Key words
Medical society, general interest

Original articles

Field validation of Tasmania's aquaculture industry bounce-diving schedules using Doppler analysis of decompression stress

David R Smart, Corry Van den Broek, Ron Nishi, P David Cooper and David Eastman

Abstract

(Smart DR, Van den Broek C, Nishi R, Cooper PD, Eastman D. Field validation of Tasmania's aquaculture industry bounce-diving schedules using Doppler analysis of decompression stress. *Diving and Hyperbaric Medicine*. 2014 September:44(3):124-136.)

Introduction: Tasmania's aquaculture industry produces over 40,000 tonnes of fish annually, valued at over AUD500M. Aquaculture divers perform repetitive, short-duration bounce dives in fish pens to depths up to 21 metres' sea water (msw). Past high levels of decompression illness (DCI) may have resulted from these 'yo-yo' dives. This study aimed to assess working divers, using Doppler ultrasonic bubble detection, to determine if yo-yo diving was a risk factor for DCI, determine dive profiles with acceptable risk and investigate productivity improvement.

Methods: Field data were collected from working divers during bounce diving at marine farms near Hobart, Australia. Ascent rates were less than 18 $m \cdot min^{-1}$, with routine safety stops (3 min at 3 msw) during the final ascent. The Kisman-Masurel method was used to grade bubbling post dive as a means of assessing decompression stress. In accordance with Defence Research and Development Canada Toronto practice, dives were rejected as excessive risk if more than 50% of scores were over Grade 2.

Results: From 2002 to 2008, Doppler data were collected from 150 bounce-dive series (55 divers, 1,110 bounces). Three series of bounce profiles, characterized by in-water times, were validated: 13–15 msw, 10 bounces inside 75 min; 16–18 msw, six bounces inside 50 min; and 19–21 msw, four bounces inside 35 min. All had median bubble grades of 0. Further evaluation validated two successive series of bounces. Bubble grades were consistent with low-stress dive profiles. Bubble grades did not correlate with the number of bounces, but did correlate with ascent rate and in-water time.

Conclusions: These data suggest bounce diving was not a major factor causing DCI in Tasmanian aquaculture divers. Analysis of field data has improved industry productivity by increasing the permissible number of bounces, compared to earlier empirically-derived tables, without compromising safety. The recommended Tasmanian Bounce Diving Tables provide guidance for bounce diving to a depth of 21 msw, and two successive bounce dive series in a day's diving.

Key words

Occupational diving, repetitive diving, surface supply breathing apparatus (SSBA), Doppler, decompression tables, diving tables, decompression sickness, diving research

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Introduction

Tasmania's salmonid aquaculture industry commenced in 1986 and now employs over 900 people. The industry is Australia's highest value fishery, producing 43,989 tonnes of salmon (22% of total Australian fisheries production in 2011–12) with an export value of AUD513 million.[1] Marine aquaculture is diving intensive, and divers have made a significant contribution to product quality. There are currently over 100 divers employed in the Tasmanian industry. Aquaculture divers breathe surface-supplied air, and perform repetitive short-duration dives in fish pens, to depths of up to 21 metres' sea water (msw), in water temperatures as low as 8^{O}C. They move from pen to pen in the course of their normal duties (Figures 1 and 2), and undertake multiple decompressions as they transit between pens (Figure 3 shows a typical dive profile). This makes 'bounce' or 'yo-yo' diving potentially more risky than traditional 'square-profile' diving (a single descent followed by a single ascent) with increased potential for bubble formation.[2] Initially, there were high levels of decompression illness (DCI) in the industry.[3,4] All currently available decompression tables are based on square dive profiles. Hence, prior to this study, there were no validated dive tables to guide the type of diving undertaken by aquaculture divers.

The Defence and Civil Institute of Environmental Medicine (DCIEM, now Defence Research and Development Canada, Toronto, DRDC Toronto) has had extensive experience in the development and validation of decompression tables using Doppler bubble detection, culminating in the production of the DCIEM air diving tables for single descent-ascent (square) and limited-repetitive dive profiles based on decompression stress.[5–9] Empirically derived dive tables based on the DCIEM no-stop times were implemented in the early 1990s for the Tasmanian aquaculture industry on the advice of diving medical specialists at the Royal Hobart Hospital (RHH).[3–5] In response to the high initial DCI rates in the fledgling aquaculture industry, these empirical diving tables were made more conservative than the usual DCIEM

Figure 1
Aquaculture diver about to enter a salmon pen

Figure 2
Aerial view of a salmon farm lease

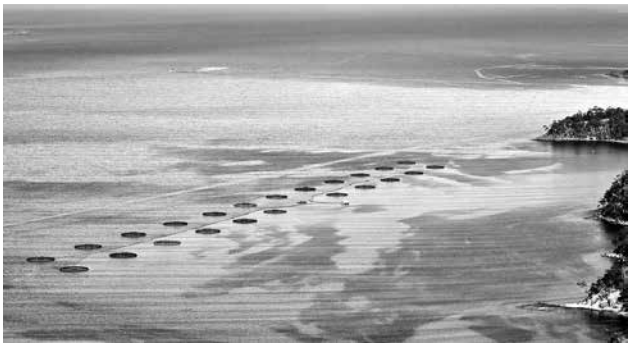
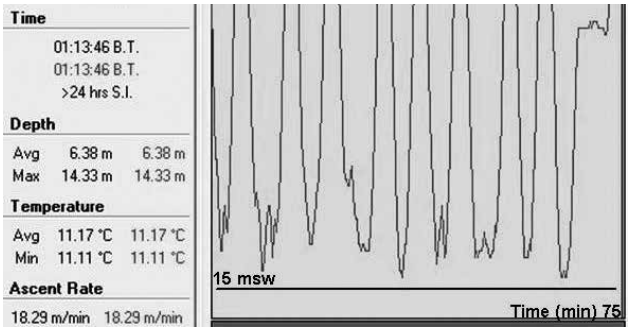


Figure 3
Dive data picture from Sensus Pro dive data recorder



no-stop table limits, and it became common practice to add an extra decompression stop as a risk-reduction measure. From 1988 to 1998, after implementation of the new bounce-diving decompression schedules, there was a 98% reduction in the incidence of DCI, theoretically preventing up to 44 divers per annum from contracting this illness (and up to 200 recompression treatments). The incidence of DCI fell from 26.19 to 0.57 cases per 10,000 dives, from 11.0 to 0.62 cases per 100 divers per year, and from 17.46 to 0.06 cases per 1,000 tonnes of annual fish production (all P values < 0.0001).[4] The observed reduction in risk came at a cost of reduced diver productivity. It was posited that the empirically derived tables were too conservative.

There was reason to suspect that the decompression stress associated with bounce diving would be greater than for the more traditional dive profiles because of a (theoretical) increased risk of bubble formation produced by multiple decompressions.[2] Bubble nuclei formed during any given decompression to the surface may not necessarily resolve completely during the next descent, and may, therefore, be available to act as a focus for gas coming out of solution during subsequent decompressions.

The best way to investigate this was to undertake field studies of the working divers using Doppler bubble detection. The technology and capability to undertake this validation became available at the RHH Department of Diving and Hyperbaric Medicine (DDHM) when one of the authors (CVdB) undertook training in Doppler monitoring of divers at DRDC Toronto, Canada in 2001.

HYPOTHESES

a. Bounce diving is an independent risk factor for decompression stress.
b. Provided divers maintain in-water dive times that are less than DCIEM no-stop time limits according to the tables, and ascent rates obey DCIEM recommendations of 18 ± 3 msw·min^{-1}, bounce diving will not result in an unacceptable risk of DCI occurrence.

AIMS

- To investigate decompression stress produced from bounce diving using Doppler ultrasonic bubble detection;
- To undertake field assessment and validation of the empirically-derived tables used by Tasmania's aquaculture industry;
- To determine whether bounce diving is an independent risk factor for DCI;
- To investigate methods for improving productivity in the industry, guided by the results of this study.

Methods

DCIEM TABLES: DEFINITIONS AND TERMINOLOGY

Except for (c) and (d) below, the definitions used in the text are consistent with those in the DCIEM dive manual.[5] The following are provided for reference:
a. *Ascent rate – the rate of travel as a diver moves from*

depth to the surface: the recommended rate for the DCIEM tables is 18 m ± 3 m·min^{-1}.
b. *Bounce-dive series*: a series of two or more descents and ascents from a dive, which are separated by less than 15 min surface interval. An example of a bounce-dive series is depicted in Figure 3. If a surface interval (SI) between dives exceeded 15 min, the next dive becomes a repetitive dive, and requires calculation of repetitive groups using the DCIEM tables to ascertain time and depth limits of the subsequent dives.
c. *Bottom time*: the total elapsed time from when the diver first leaves the surface to the time (next whole min) that the diver commences the last ascent (measured in min). For a bounce-dive series, bottom time includes SIs between bounces, provided they are less than 15 min.
d. *In-water time*: this differs from bottom time and is the total time the diver spends in the water, minus the time spent at the surface during surface intervals. It includes the time of the last ascent and the decompression stop.
e. *Repetitive factor*: a numerical figure, used for repetitive diving, determined by the Repetitive Group and the length of a SI after a dive. A value of 1.0 reflects no residual nitrogen in the diver. Values ranging from 1.1 up to 2.0 reflect increasing amounts of residual nitrogen.
f. *Repetitive group*: a letter of the alphabet which relates directly to the amount of residual nitrogen in a diver's body immediately on surfacing from a dive. Letter "A" is lowest.
g. *Repetitive dive*: any dive that has a DCIEM repetitive factor greater than 1.0. This includes any series of more than one dive, where dives are separated by SIs of greater than 15 min, unless the SI was of sufficient duration that the diver's repetitive factor returned to 1.0.
h. *Surface interval*: The time which a diver has spent on the surface following a dive; beginning as soon as the diver surfaces and ending as soon as the diver starts the descent for the next dive.

A prospective, observational, cohort study was conducted over six years using Doppler ultrasound to assess sub-clinical decompression stress. This project was approved by the Institutional Research Ethics Committees at both the RHH and DRDC Toronto (RHH Ethics reference number H6455). All divers provided informed consent for data collection and participation in the study.

Commencing May 2002, field data were collected by one or more of the authors during routine diving activities at marine farms near Hobart, Tasmania. Farm visits were timed to coincide with maximum diving activity, and with dives that were consistent with the most common profiles used in the industry. At the commencement of the study the most common profiles were 12 msw and 15 msw. As the study progressed, there were changes in farming techniques and technology requiring extension of the data collection to deeper profiles. There were no ethical issues arising from this because the farms implemented the technology and diving processes independently of this study.

FIELD DATA COLLECTION

Field data were collected primarily by one of the authors (CVdB), with regular visits by DS to monitor diver health. A questionnaire was completed at the time of Doppler scanning, prior to diving. This collected information about the diver's preceding 24 hours including: exercise prior to diving, medications, alcohol consumption, tobacco usage, sleep, fatigue, food and fluid intake, colds or other infections, diving activity and any physical complaints. All of these factors were considered to be potential confounders that have been reported to increase bubble formation. Anticipated altitude exposures (by air or car) post diving were also documented. Divers also completed a post-dive health questionnaire and were required to report symptoms or signs of DCI in the 24 hours after diving.

Divers undertook their usual, working bounce-dive series breathing surface-supplied air from a pod of high-pressure cylinders. Air utilisation was recorded, providing an indication of the workload of the dive. A routine decompression stop for 3 min at 3 msw was performed at the end of each diver's last bounce dive. Each diver's depth and time underwater were monitored and recorded continuously using a submersible dive data logger (*Sensus Pro*, Reefnet Incorporated, Mississauga, ON, Canada, Figure 3), from which the data could be downloaded into a laptop PC upon completion of the dive. Maximum depth, bottom times, number of bounces, ascent rates and water temperature were recorded. The diver was blind to the data collected.

Data handling, analysis and reporting took place at the DDHM (DS, DC), with expert input from the DRDC Toronto. DRDC scientists (RN, DE) independently validated assigned bubble-grade classifications in a randomly selected 10% of readings, and assisted with statistical analysis.

DOPPLER MONITORING

Doppler sampling was undertaken according to the techniques described by Eatock and Nishi.[10–13] One author (CVdB) received training in Doppler monitoring at DRDC Toronto, and subsequently on several occasions over the course of the study, to maintain his skills. All measurements were performed by this individual, or under his direct supervision. Recordings were undertaken using a 2.5 MHz continuous-wave Doppler ultrasound device (TSI DBM 9008, Techno Scientific Inc., Ontario, Canada) with a Doppler array probe (TSI-DPA7). Doppler recordings were taken over the precordium and both subclavian veins at 20-min intervals for at least 2 hours post dive (or until bubbles were no longer detectable for three successive readings) and recorded onto magnetic audio cassettes. The first recording was performed immediately after the diver exited the water. Each recording at 20 min intervals included the following:

- precordium, at rest – 60 seconds;
- precordium, 3 squats – 30 seconds after each;

- subclavian veins, at rest – 30 seconds;
- subclavian veins, 3 hand clenches – 15 seconds after each.

DOPPLER DATA ANALYSIS

Doppler recordings were aurally graded according to the standard Kisman-Masurel (KM) Code.[9,14] Detected bubbles were subjected to a three-fold classification that analysed (i) frequency, (ii) either percentage of cardiac cycles affected (at rest) or duration (following movement), and (iii) signal amplitude of detected bubbles, to yield a single bubble grade (0 to 4).[14]

It was known from a large series of DCIEM air divers (1,726 subjects) that, based on the maximum recorded bubble grades from all monitoring sites and conditions (rest/movement), grades 2 or less (low stress) were associated with clinical symptoms of DCI in 1.1% of cases, and bubbles of grade 3 have been quoted as having a DCI incidence ranging up to 6.3%. Grade 4 bubbles had a DCI rate of 9.7% at the time DCIEM collected its original data.[9] Grade 4 bubbles may produce a much higher risk of DCI when detected after exceptional or extreme exposure dives.

Bounce tables were defined *a priori* as 'low risk' if the bubble scores complied with DCIEM/DRDC-defined limits of acceptability (grade 2 or fewer bubbles in 50% or more of the subjects). DRDC Toronto defined dive profiles producing Doppler bubble grades 3 or 4 in 50% or more of the subjects as of 'high risk' and were to be rejected for use. This study followed the DCIEM table recommendations and definitions.

DATA CONSISTENCY

Aural scoring is known to be observer-dependent; therefore, all Doppler recordings were graded by a single author (CVdB). A random sample of 10% of all recordings were scored and validated independently by DRDC Toronto to ensure data consistency.

STATISTICS

All data were entered into a Microsoft Access® (Microsoft Corporation – Redmond, Washington, USA) database and analysed using Graph Pad Instat® version 3.0 for Windows and Graph Pad Prism® version 4.03 for Windows (Graph Pad Software, San Diego, California, USA, 2003 and 2005). Bubble grades were treated as categorical data for statistical analysis. The highest KM bubble grade following each dive was tabulated for statistical comparison.

Bubble grades were dichotomized into 'acceptable' (grades 0–2) versus 'unacceptable' (grades 3–4) to facilitate subsequent statistical analysis. The resulting 2 x 2 contingency tables were subjected to Fisher's exact test. All tests were 2-tailed and $P < 0.05$ was considered statistically significant. The bubble grades were also correlated with any symptoms divers noted in the 24 hours following diving. When bubble grades were compared to continuous variables such as numbers of bounces or percentage of DCIEM time limits, Pearson's or Spearman's rank correlation coefficient was calculated and tested for significance of association, depending on whether data were continuous or categorical. Two-way analysis of variance was used to assess the relative contributions of independent variables to the dependent variable, Doppler bubble grade. It was planned to undertake multiple regression analysis to assess factors identified in the pre-dive questionnaire and also dive-related factors that affected bubble grades in this population of divers, if sufficient divers recorded unacceptable bubble grades.

It was predicted that more than 90% of divers would produce KM bubble grades of 2 or less, consistent with low-risk profiles, based on data from chamber attendants diving a 14 msw table at Royal Hobart Hospital.[15] If more than 50% of divers experienced bubble grades 3 or 4, then the profile would be rejected and the industry would be advised to modify their decompression table for that series of bounce dives. If less than 50% of divers had bubble grades 3 or 4, then profiles would be recorded as acceptable risk. Using sample size of 20 dives at each depth, this study had 80% power to detect an absolute 40% difference between the proportion of divers expected to have bubble grades 2 or less, and the point at which we rejected a given dive profile (using $\alpha = 0.05$). Being a field study in a workforce environment, it was recognised that there may be some deviation from the ideal, due to issues beyond our control.

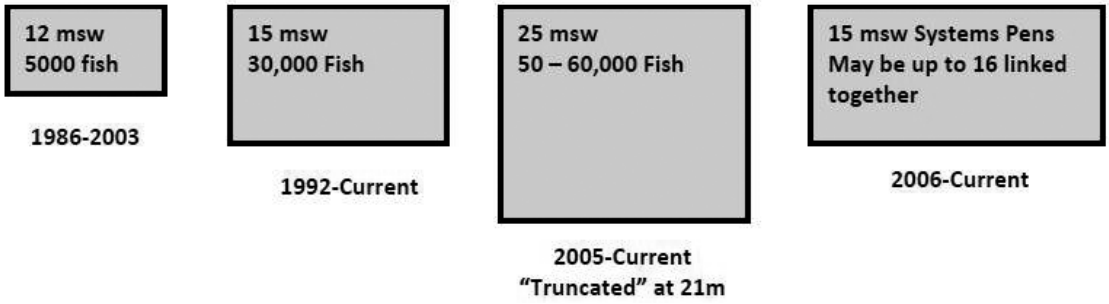
Hence, we aimed for a cohort sample of over 100 bounce-dive series, and a minimum of 20–25 bounce-dive series for each individual depth range. This would allow data collection consistent with DCIEM methods across the four most frequently dived profiles: 10–12 msw, 13–15 msw, 16–18 msw and 19–21 msw. The expected incidence of decompression illness over 100 dives was zero. The 95% confidence limits for 100 samples are 0–3.6 % risk of DCI, and for 20 samples are 0–16.8% risk of DCI, using the binomial distribution. There are recognised statistical limitations to proving dives are acceptable risk by defining DCI as a binary outcome (yes or no), given these wide confidence limits.[16,17] This is further limited if the index event (DCI) has a low incidence. The 1999 study observed only 0.57 cases of clinical DCI per 10,000 dives over the 1996–98 study period.[4]

Results

IMPACT OF EXTERNAL FACTORS ON DATA COLLECTION

This study required some adjustment to keep up with concurrent evolution of technology and dive practices occurring within the industry. At the commencement of the study, only two salmon pen sizes existed: 80 m circumference, 12 msw depth and 120 m circumference,

Figure 4
Evolution of salmon pens by depth and size during the course of the study



15–16 msw depth. The 12-msw pens were superseded in early 2003, preventing sufficient data collection of dive times close to DCIEM no-stop limits for 12 msw (150 min). The evolution of salmon pens is shown in Figure 4. Because of logistical issues, 25-msw pens were 'truncated' with a false bottom at 21 msw.

In addition, some farms expanded significantly, which increased the travel time between pens, and caused many dives to become repetitive dives because surface intervals exceeded 15 min. This reduced the numbers of bounce-dive series that were available for analysis. Unpredictable local events also had a negative impact on data collection. On more than 10 occasions, authors arrived at the salmon farm, ready for data collection, only to discover that work priorities had shifted that day to fix an emergency (e.g., seal strike on a pen – see journal cover image, mooring or other issues), and the bounces for that day had been cancelled. After a full day's expedition, with 80–100 km travel in either direction, no data were collected. This prolonged data collection to 6 years (May 2002 to March 2008).

Complete field data were collected from 55 different divers undertaking 150 bounce-dive series totalling 1,110 bounces (mean 7.4 bounces per series, SD 3.1). The 55 male divers (mean age 27.6 (SD 5.1) years, height 179.0 cm, weight 84.0 kg, BMI 25.7 kg·m^{-2}) were all professionally trained to minimum of AS/NZS 2815.2 (aquaculture-restricted).[18] All divers had not dived for more than 18 hours prior to commencing their bounce-dive series. The average water temperature during data collection was 12.3^{O}C (range: 8–15^{O}C). Four bounce-dive series were excluded from the analysis (total 16 bounces), three because the dive series extended too deep (22 msw – bubble grades 0, 1 and 2) and one because the diver suffered sinus barotrauma.

Figure 3 shows a sample recording from a bounce-dive series of nine individual bounces, to a maximum depth of 14.33 msw and surface-to-surface duration 73 min 46 seconds. The diver undertook a 5-min decompression stop spent at 3 msw in accordance with protocol, during the last ascent. The maximum recorded ascent rate was 18.3 msw·min^{-1}. Note the in-water time for the above dive was 55 min, and bottom time was 65 min.

BUBBLE GRADES AND BOUNCES FOR VARIOUS OPERATIONAL DEPTHS

Table 1 summarises mean in-water times, bottom times and median number of bounces for each depth. Apart from the 12-msw series, all depths had mean in-water durations that exceeded 80% of DCIEM table limits, and bottom times (adding all surface intervals between bounces and in-water times) that exceeded DCIEM limits.

Table 2 summarises the numbers of bounce-dive series and individual bounces undertaken by the divers and their bubble grades, stratified by dive depth. Twenty-two divers were evaluated after diving at different depths on different days; hence there were 77 subjects who contributed data across

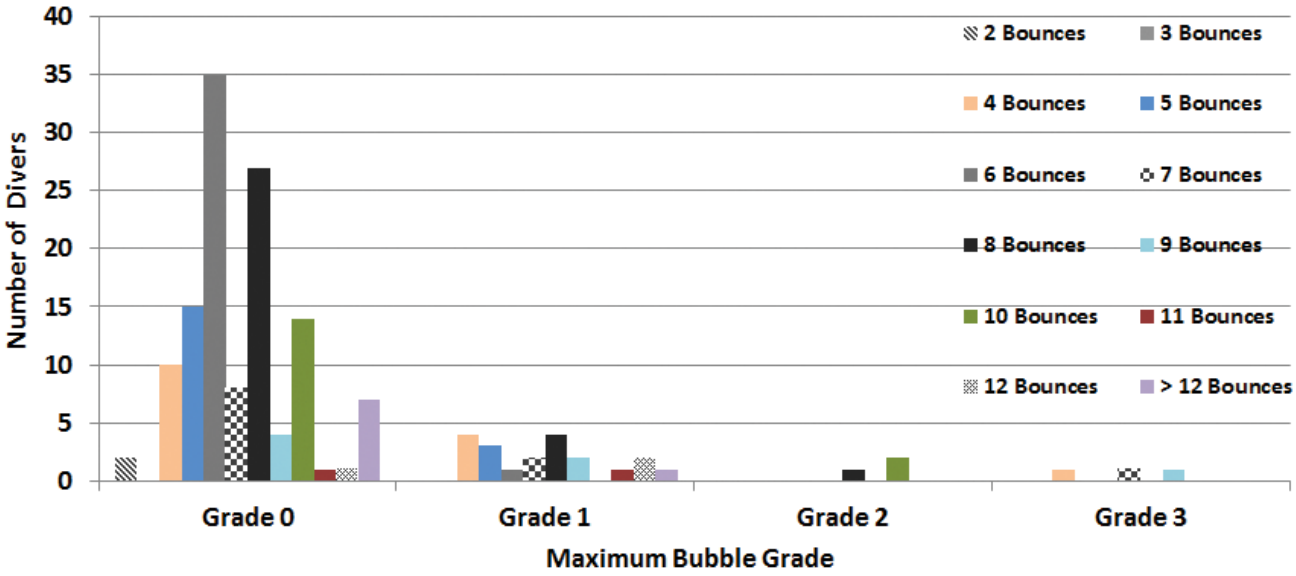
Table 1
Mean in-water times, bottom times and median number of bounces for each depth; Inter-quartile range – IQR

Depth (msw)	Mean in-water time (min)	DCIEM limit (min)	Mean bottom time (min)	Mean surface interval (min)	Median number of bounces (IQR)	Mean in-water time as % DCIEM no-stop limit	Mean bottom time as % DCIEM no-stop limit	Median bubble grade
≤–12	40	150	74	34	8 (8)	26	51	0
13–15	61	75	98	37	7 (6–10)	81	131	0
16–18	56	50	106	50	6 (5–9)	112	215	0
19–21	32	35	77	44	4 (4–6)	91	218	0

Table 2
Bounce dive series and individual bounces undertaken by the divers and their bubble grades, stratified by dive depth

Depth (msw)	Number of divers	Number of bounce dive series	Median number of bounces (range)	Bubble grades 0	1	2	3	Total bounces
≤ 12	10	24	8 (7–12)	22	2	0	0	194
13–15	41	82	7 (4–21)	63	14	3	2	651
16–18	18	32	6 (2–20)	24	7	0	1	207
19–21	8	12	4 (2–8)	8	3	1	0	58
Total	77	150	7 (2–21)	117	26	4	3	1,110

Figure 5
Bubble grades for all 150 dive series split by bounce numbers



the four depth ranges in Table 2.

Overall, 97% of bounce-dive series evaluated were low stress (Doppler grades less than 3), well within DCIEM tolerances. The median bubble grade for all 150 bounce-dive series was 0 (Figure 5). No divers experienced any symptoms suggestive of DCI post dive and none of the subjects required treatment for DCI during the study period.

From available bounce-series data, three dive depth ranges had sufficient data for evaluation of DRDC tolerances (because the in-water times were greater than 80% of DRDC limits) to test hypothesis (a). Data were incomplete for the bounce-dive series conducted up to 12-msw depth, owing to the industry adopting deeper salmon pens early in the study.

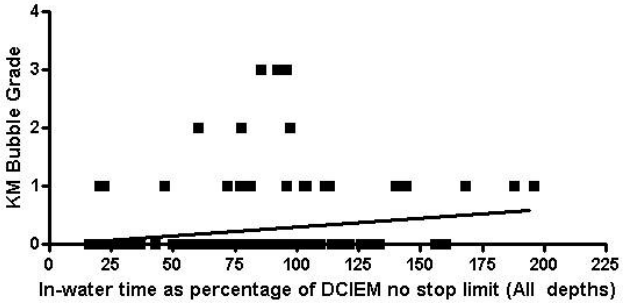
EFFECT OF IN-WATER TIMES

At 13–15 msw, the mean in-water time (61 min) was 81% of the DCIEM no-stop limit, and bounce-dive series up to 10 bounces resulted in a median bubble grade of 0. Twenty per cent of the bounce dive series in this depth range exceeded the DCIEM limit of 75 min; these data were regarded as valid for the reasons outlined in the discussion. The 16 bounce-dives series which exceeded DCIEM limits were evaluated in more detail. In-water times ranged from 76 to 126 min, mean 86.3 min. The mean cumulative SI between bounces was 36 min, and the median number of bounces was 10 (range 6–21). Twelve of these longer-duration bounce-dive series produced grade 0 bubbles and four produced grade 1 bubbles. Divers undertaking bounce-dive series with in-water times less than 75 min had lower median numbers of bounces (six per dive series), and less time on the surface (21.5 min).

At 16–18 msw, divers had a mean in-water time of 56 min, which exceeded DCIEM no-stop limits. All 24 bounce-dive series with six or fewer bounces resulted in a median bubble grade of 0, provided bottom time did not exceed 50 min.

Eighteen of 32 bounce-dive series exceeded DCIEM table limits of in-water times for this bounce-series depth range, so were evaluated in more detail. In-water times ranged from 52 to 98 min, mean 66.0 min. These dives had an average of 67

Figure 6
Graph comparing in-water dive duration (as percentage of DCIEM air table no-stop limit) and bubble grade showing positive correlation trend line



min cumulative SI, and the median number of bounces was six (range 2–20). Twelve of these longer-duration bounce dive series produced grade 0 bubbles and six produced grade 1 bubbles. Divers undertaking bounce dive series with in-water times less than 50 min had median numbers of 5.5 bounces, and less time on the surface (31.2 min).

At 19–21 msw, the mean in-water time (32 min) for the 12 bounce-dive series was 91% of the DCIEM no-stop limits. Ten of the dive series undertook up to six bounces with median bubble grades of 0. Four of the 10 bounce-dive series exceeded the DCIEM no-stop limits in this group, but because of small numbers and only four diver series having six or more bounces, the data are less robust.

There were insufficient data from the less than 12-msw range for evaluation because the average in-water time was only 26% of the DCIEM limit (mean in-water time 40 min, range 24–75 min; mean bottom time 74 min, range 53–139 min). Even allowing for this, it was apparent that a median of eight bounces did not result in significant decompression stress at 12 msw during these short-duration dives.

IMPACT OF BOUNCE DIVING ON DECOMPRESSION STRESS AS MEASURED BY BUBBLE GRADE

The correlation between the number of bounces and bubble grades for all 150 bounce-dive series was not statistically significant (Spearman $r = 0.07$, $P = 0.42$). When stratified by depth ranges, a trend towards significance was identified for the relationship between number of bounces and bubble grade in the 13–15 msw range. (Spearman $r = 0.21$, $P = 0.06$). No depth ranges had statistically significant relationships between numbers of bounces and bubble grades. Bubble grades of 0 were recorded in 78% of divers. There was no significant difference in the mean number of bounces performed by divers with 0 bubble grade (7.3, SD 3.1) compared to those with higher maximum bubble grades (7.4, SD 3.5); difference between the means -0.048 +/- 0.64, 95% CI -1.3 to 1.2, $P = 0.93$).

Figure 6 plots the relationship between in-water dive duration and bubble grade, which was statistically significant (Spearman $r = 0.23$, $P = 0.004$). This suggested that the possible trend observed for number of bounces and bubble grades may have been influenced by in-water dive duration. There was a highly significant relationship between number of bounces and in-water dive duration (Pearson $r = 0.28$, $P = 0.0006$). This was logical because, as divers undertook more bounces in the dive series, their dive duration increased.

The relationship between bubble grade and bottom time as a percentage of DCIEM limit, was statistically significant (Spearman $r = 0.17$, $P = 0.03$). Bottom time included (variable) time that divers spent on the surface during their bounce-dive series.

OTHER VARIABLES AFFECTING DECOMPRESSION STRESS

Pre-dive questionnaires identified that four divers experienced health issues prior to diving: one with gastroenteritis the day prior, one with a hand injury, one with epistaxis and one with a torn thigh muscle. None of these divers had bubble grades > 2. Intra- and post-dive factors included two divers being harassed by seals, and another undertook a very hot shower. Multiple sub-surface bouncing occurred in two bounce-dive series and four divers missed their scheduled decompression stops. None of these divers recorded a bubble grade > 1. The diver with sinus barotrauma was excluded from analysis as no Doppler readings were taken.

We also assessed whether or not recent diving influenced bubble grade. Although all divers commenced their bounce-dive series with a DCIEM repetitive factor of 1.0, some had dived the previous day(s) and some had not. When stratified as two groups – dived previous day versus not dived – there was no significant difference in bubble grades. We did not collect precise data on the time interval from the previous dive if it was greater than 24 hours.

The mean ascent rate for all divers was 18.8 (range 9–40) msw·min^{-1}. Recommended DCIEM ascent rates were exceeded on 12 dive series (8% of total). None of the divers with rapid ascents had higher than grade 2 bubble, but the relationship between ascent rate and bubble grade was statistically significant (Pearson $r = 0.16$, $P = 0.046$).

The data were further analysed for sources of variance. In-water dive duration (per cent of DCIEM limit) accounted for 72.5% of the variance of bubble grades, and was highly significant ($P < 0.0001$). The ascent rate accounted for 13.8% of variance in bubble grade and was not significant ($P = 0.32$). Only 3.7% of variance was attributable to the number of bounces ($P = 0.47$). A multiple regression equation was calculated from available data, and the relationship between maximum bubble grade and the other three variables was significant in the model ($P = 0.04$):

Figure 7
DCI cases treated at Royal Hobart Hospital 1989–2010 showing trends for salmon farm divers

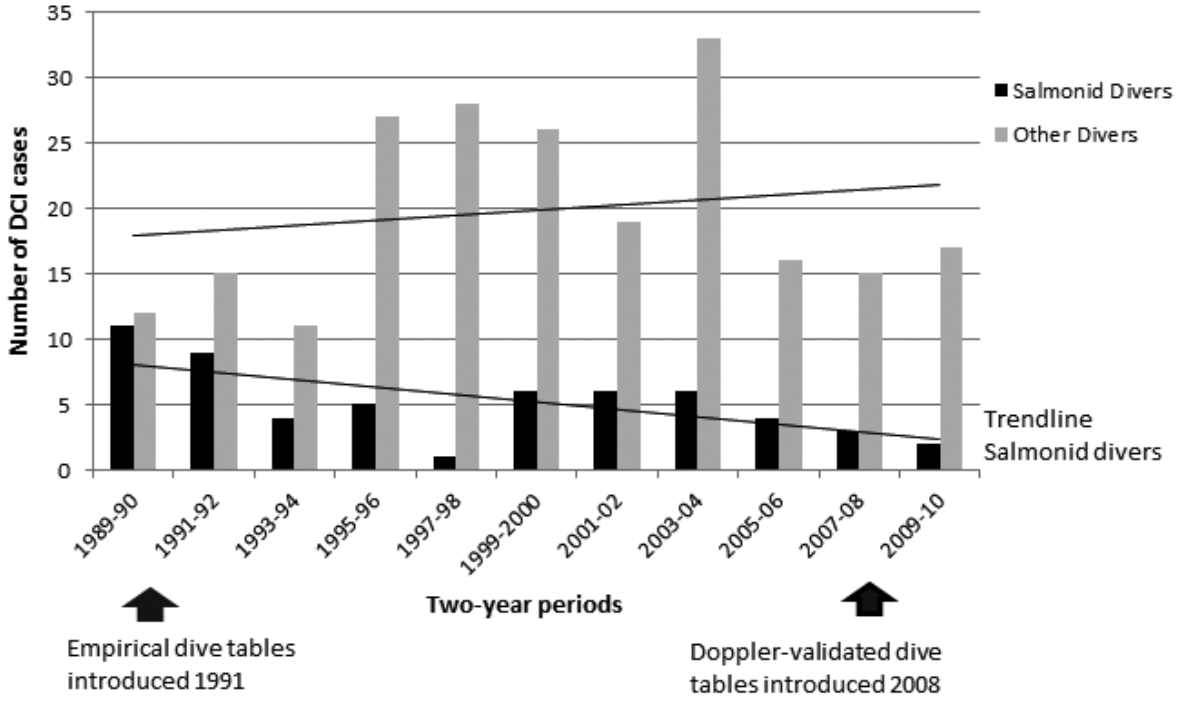


Table 3
DCI incidence compared to number of dives, number of divers and tonnage of fish

Period	Number of divers	Number of dives	Number of cases of DCI	Tonnes of fish	DCI rate per 10,000 dives	DCI rate per 100 diver years	DCI rate per 1,000 tones of fish
1989–1990	50	4,200	11	1,260	26.19	11	17.46
1993–1994	87	11,200	4	8,824	3.57	2.3	0.45
1997–1998	81	17,542	1	16,264	0.57	0.6	0.06
2003–2004	143	44,100	6	29,977	1.36	2.1	0.20
2008–2009	108	33,320	3	59,641	0.90	1.4	0.05

$$MBG = -0.42 - 0.0021 * N + 0.024 * A + 0.0029 * T \quad (1)$$

where *MBG* = maximum bubble grade for dive; *N* = number of bounces; *A* = maximum ascent rate; *T* = in-water time as percentage of DCIEM no-stop time.

DCI EPISODES FROM THE AQUACULTURE INDUSTRY

Over two decades of study, one of the authors (DS) has surveyed the aquaculture industry at 4–5-year intervals to determine the number of divers and number of dives undertaken. The last survey was undertaken in late 2008 at the end of study data collection. Table 3 demonstrates a fall in the incidence of DCI when measured per number of dives, number of dive years and tonnage of fish production. The DCI incidence for 2008–2009 was 1 per 11,106 dives.

Figure 7 depicts all DCI cases treated at the DDHM from 1989 to 2010 in 2-yearly intervals. The population is split into two groups: salmonid divers and all other divers. All other DCI cases include other professional divers (e.g., abalone, inshore, offshore and scientific), recreational scuba and hookah divers. Since 1989, the numbers of cases from the aquaculture industry (salmonid divers) show a statistically significant falling trend (test for trend, $\chi^2 = 23.6$, $P = 0.008$), compared with all other DCI cases, which are increasing. The trend continued to 2010, beyond the end of the study period. The time points at which the empirical and Doppler-validated dive tables were introduced are marked below the X axis.

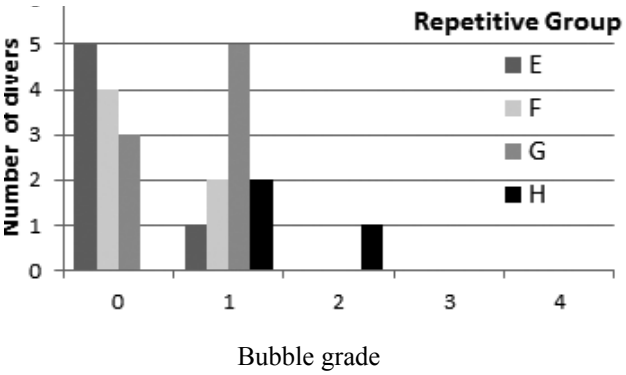
DOPPLER ANALYSIS OF TWO SETS OF BOUNCES

In response to an industry request, we undertook Doppler measurements on divers conducting two series of bounces in a day. This practice was already occurring at one company,

and hence the measurements were observational of an existing (and unchecked) practice, rather than testing a new hypothesis. In response to the request, the authors advised that there should be strict guidelines governing the two sets of bounces, so that results would be reproducible and of practical use. The guidelines to allow two sets of bounces on the same day are outlined in Table 4.

The results of Doppler analysis of two consecutive bounce dive series using the above criteria were available from 23 divers. All first bounce-dive series were 16–18 msw. The depth range for the second bounce-dive series was 16–18 msw (mean 17.7 msw, mean duration 38 min, median number of bounces 4). Following the second set of bounces, the maximum bubble grade for any diver was 2, with a group median of grade 0 and the divers had DCIEM repetitive groups E to H (Figure 8), Bubble grades broadly followed the repetitive groups – as the repetitive group increased, so did the bubble grade.

Figure 8
Doppler grades and DCIEM air table repetitive groups after a second series of bounce dives



Discussion

This study took over six years to obtain sufficient field data in three depth ranges to confirm low risk from bounce diving. Many of the challenges are described in the results, and these demonstrate the difficulties of conducting field research on working divers. It was not logistically possible to evaluate every dive depth range with 100 samples. In addition, it was important that the assessed dives were sufficiently provocative: producing enough decompression stress to provide valid guidance in table development. In developing or testing tables and dive procedures, the number of dives that can actually be done is driven more by practical considerations than by trying to meet statistical criteria. Compromises have to be made and around 20 man-dives per profile without a DCI incident has been considered to be acceptable.[17] This was followed for our field study of aquaculture divers, but limiting depth ranges to those being dived operationally.

Ninety-seven per cent of dive profiles evaluated in this series were low stress (Doppler grades 2 or less), well within DRDC Toronto tolerances. Whilst this provided evidence of acceptable risk, it also led to difficulties with any multivariate analysis of causation of decompression stress, because very few dives were of sufficient stress, as defined by KM bubble grades. The only factors linked to higher decompression stress were the time spent in the water as a percentage of the DCIEM table limit and ascent rates.

A surprising result was the lack of correlation between number of bounces and Doppler bubble grades, and the overall low grades measured in the divers undertaking bounce diving. A number of factors may have influenced this result. Firstly, we may not have 'pushed' the divers into sufficient nitrogen uptake to create high decompression stress (i.e., dives may have been too conservative). In working divers, this conservative approach is justified. The divers were already using DCIEM tables to guide their practice; however, the study was observational so we did not seek to influence their dive practices whilst they were occurring. It was our aim to record a dataset that was close to table limits, and this was achieved for depths 13–15 msw, 16–18 msw and 19–21 msw. Our data support the use of DCIEM no-stop table limits as a guide to risk reduction when bounce diving. Only 8% of divers exceeded recommended ascent rates, and this also would have reduced risk. Even though the divers were blinded to the data recorder they were wearing, they may have been extra careful knowing their dive was being monitored as part of the study.

Some Doppler studies have used integrated scoring systems to evaluate progression of bubble grades over time, rather than peak values. A well known example is the Kisman Integrated Severity Score (KISS).[19,20] KISS provides a broader representation of bubble activity over time by estimating 'the area under the curve'. We did not calculate KISS in this study because we were interested in the maximum bubble grades at rest and with movement. As operational divers, the salmon farm population were active between dives, and this may have led to transient bubble release from activity (similar to the movement case), which is generally greater than the steady state at rest. As bubble grades of 0 were detected in 78% of divers in this study, KISS would have been zero for the majority of the divers. Although KISS provides a broader representation of bubble grades over time, it still does so only at fixed time points, 20 minutes apart. Given that 97% of the dives in this series were grade 2 or less, and there were no cases of DCI, we believe that the outcomes are consistent with acceptable levels of risk in the industry. Our original aims of the study were to investigate what was happening operationally and monitor working divers. There was no significant difference in the number of bounces performed by divers with bubble grade 0 compared to those with higher maximum bubble grades. This suggests that undertaking KISS calculations may not add further to the conclusions; however, we do

Table 4
Tasmanian Bounce Diving Tables
Criteria for two consecutive series of bounce dives

1. Divers are required to be DCIEM Repetitive Factor 1.0 at the commencement of the first bounce-dive series.
2. The maximum depth for the first bounce-dive series is no more than 18 metres.
3. The in-water time for the first bounce-dive series is calculated as the time from commencing first descent to the time of exiting the water, minus the sum of all time spent on surface intervals. The in-water time includes time spent in the water for the decompression stop.
4. The repetitive group for DCIEM tables is calculated from the first bounce-dive series in-water time, after surfacing.
5. A minimum surface interval of 2 hours must occur between the first and second bounce-dive series.
6. The repetitive group is then used to calculate the allowable bottom time for the second bounce-dive series.
7. The maximum depth of the second bounce-dive series shall be no deeper than the maximum depth of the first bounce dive series.
8. The number of allowable bounces in the second bounce dive series shall be restricted to half the number of the first bounce-dive series (maximum of 5 bounces), and with maximum bottom time as defined by the DCIEM repetitive group allowable bottom time.

Table 5
Tasmanian Bounce Diving Tables

Depth (metres)	Number of allowable bounces in dive series	In-water† dive time limit (min)
≤ 9	10*	300‡
10–12	10*	150‡
13–15	10	75
16–18	6	50
19–21	4	35
> 21	Use DCIEM repetitive dive tables	

1. Ascent rates shall be ≤ 18 metres per minute;
2. Surface intervals between bounces shall be < 15 minutes;
3. 3-minute decompression stop at 3 metres shall be performed during the last ascent;
4. A second bounce dive series is possible after a 2- hour surface interval, provided specific criteria are obeyed (Table 4).

Notes:
* Bounce numbers based on validated safety of 13 to 15-metre bounce-dive series;
† In-water time limit defined as: *the total time the diver spends in the water, minus the time spent at the surface during surface intervals. It does include the time of the last ascent and the decompression stop.*
‡ It is recommended bounce-series dive times are less than DCIEM table limits until fully validated.

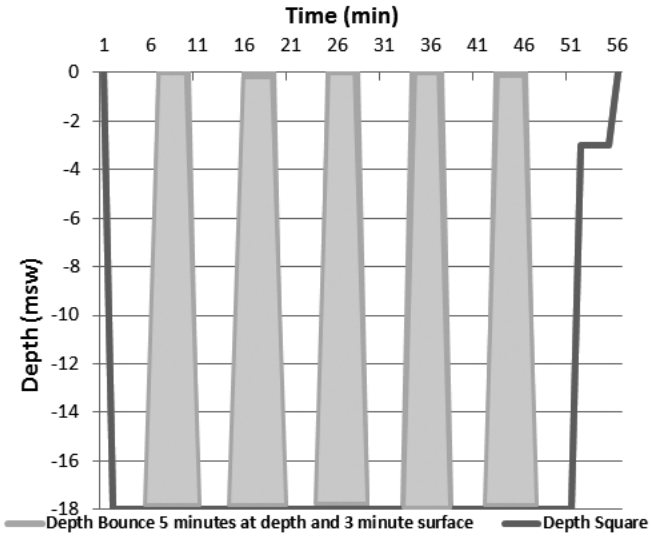
plan to publish the calculations along with additional data, in a subsequent paper.

We also recognise the limitations of our method for calculating decompression stress using Doppler ultrasonic bubble detection. Recent use of 2-D echocardiography has demonstrated good intra- and inter-rater reliability, and may supersede aural grading systems in the future.[21] These systems are still recognised as semi-quantitative. Aural Doppler still has advantages for field research in that it is faster to undertake and divers can assist with accurate probe placement. Although scoring requires an experienced operator, the same is required for accurate images using 2-D echocardiography.

In this study, total in-water time did not take into account the surface interval between dives or that any off-gassing took place during this interval. It appears that there is sufficient off-gassing during the surface intervals, so that the 'effective' bottom time is less than or equal to the DCIEM limit even though the sum of the actual times spent on the bottom may exceed the DCIEM limit. This was illustrated by the data from the 16–18 msw depth range. Divers who exceeded DCIEM time limits for bottom times spent the same amount of time at the surface as they did in the water. There was time to off-gas between each individual bounce dive, which would have reduced nitrogen load in the body.

In addition, the criterion of restricting in-water time to less than the DCIEM limit added conservatism because this time includes all ascent times plus the 3-min decompression

Figure 9
Theoretical bounce-dive series showing five returns to the surface before completing the dive on the sixth ascent (the shaded areas reflect less nitrogen absorbed compared to a square dive profile)



stop on the last bounce. If there are seven bounces, then this reduced the time spent at depth by 10 min less than the DCIEM limit. With 12 bounces, the reduction was 15 min. Because bottom times included surface intervals, with a large number of bounces, the DCIEM limit for *bottom time* could be exceeded, but the *in-water time* could be less than the DCIEM limit. Hence less depth-time exposure may have offset the multiple decompressions. The decompression stop may have independently reduced risk of decompression stress, although this was not assessed in the study.

The above factors provide an explanation for the low decompression stress observed in our data. Bounce diving has reduced the 'area under the curve' compared with a square dive profile – hence, there would be less nitrogen uptake during the ascent, surface interval and descent phases of each surface bounce, compared to staying at depth (Figure 9). In Figure 9, the diver conducting bounce diving has five returns to the surface with a bottom time of 48 mins. Compared to the square dive profile, the bounce diver has less depth-time exposure by the equivalent of 20 min at 18 msw (i.e., the diver had 42% less depth-time exposure). An additional factor may have been that there was insufficient time for bubbles to grow until after the last ascent because divers were under pressure again quickly following their brief surface interval (akin to surface decompression diving). Finally, we cannot rule out other factors such as vibration from boat engines as divers travel between fish pens, which may have a protective effect.[22]

Our data are also consistent with mathematical modelling of yo-yo diving conducted by Flook who concluded: "*yo-yo diving of the type traditionally practised in fish farm diving can be very safe and that dividing the total bottom time into several shorter dives alternating with a surface interval is less of a risk than diving the envelope*."[23] Lower risk of DCI has been demonstrated in rats and pigs undertaking yo-yo diving with 2 or 3 ascents compared with single ascents.[24] Our data are the first to confirm that 'bounce' or 'yo-yo' dive profiles as part of routine occupational diving activities can be conducted with acceptable levels of risk.

A number of divers exceeded the defined DCIEM in-water time limits during this study: 16 of 82 at 13–15 msw, 18 of 32 at 16–18 msw, and 5 of 12 at 19–21 msw. These breaches of rules usually occurred accidentally, because divers were not aware of the exact depth of each salmon pen as they entered. For example, the centre of the pen may have been conical rather than flat, and the dead fish were situated 1–2 m deeper in the "*mort cone*" than the average depth of the bottom of the pen. The real-time dive data in Figure 3 demonstrates how brief some of the dips to maximum depth actually are. DCIEM limits apply only to the maximum depth in a given depth range. At the lower end of the depth range, the DCIEM limit would be considerably longer. For example, DCIEM modelling would allow an additional 10 min of diving at 17 msw compared to 18 msw. The divers' depths were clearly variable during all the bounces, and they were only at maximum depth for brief periods. Given these considerations and the inherent conservatism of the in-water definition, the inclusion of the data which exceeded DCIEM limits is supportable.

Bubble grades and dive duration as a percentage of the no-decompression DCIEM time limit demonstrated a significant positive correlation. This result was expected because previous research has demonstrated that as the diver approaches known decompression limits, their risk of bubbling increases.[6–14,16,17,25] Ascent rates also had a significant correlation with decompression stress. This emphasised the importance of maintaining ascent rates consistent with DCIEM recommendations and adding the routine decompression stop.

We have been able to demonstrate that it is possible to undertake bounce diving using DCIEM tables to guide depths and times. On the basis of these results we have been able to increase the permissible number of bounces at each depth compared with the earlier empirical restrictions. Our recommendations for dive times and numbers of bounces are summarised in Table 5 – *The Tasmanian Bounce Diving Tables*. These recommendations have the proviso that in-water dive times must not exceed DCIEM limits for a given depth, that ascent rates are kept at less than or equal to 18 msw min^{-1}, and that a 3-min decompression stop at 3 msw occurs during the last ascent.

We have been conservative in recommending a maximum of only four bounces in the 19–21 msw range, because our data for six or more bounces was based on only four divers. We also have less certainty regarding the 7–9 msw and 10–12

msw tables and this may be further investigated if industry technology changes in the future to using shallower pens. We recommend no more than 10 bounces in a series, and staying well inside DCIEM no-stop time limits for ≤ 12 msw, until further research has occurred. This recommendation may be overly conservative; however, it does allow some flexibility and a margin for untoward events.

Given that the majority of divers (126/150, 84%) contributing data had undertaken diving the previous day, or on multiple days prior to measurement day, we consider that our data are robust enough to be generalised, and may be applied to industries that require bounce diving as part of their operations on a day-to-day basis. We did not evaluate the possible risk factor for DCI resulting from a prolonged layoff (more than three days) before diving. Given that 97% of the dives in this series were grades 2 or less, and there were no cases of DCI, it is unlikely the study would have had the power to inform this question.

Following implementation of the Doppler-validated Tasmanian bounce diving tables in 2008, the industry benefitted from improvements in productivity, compared with the previous empirical bounce limits set in the early 1990s. The number of pens dived (or allowable bounces per dive series) increased by 25% from eight to 10 at 10–12 msw, by 50% from four to six at 16–18 msw, by 100% from two to four at 19–21 msw and by 150% from four to 10 at 13–15 msw. Dive times in the new tables are based on in-water times, whereas they were previously based on bottom times. There was additional productivity advantage from undertaking a second bounce-dive series in the same day and this was also validated by our research. We have demonstrated that, with strict criteria, it is possible to conduct a second series of bounces after an earlier first series. This will permit a diver to undertake up to 15 bounces in 15-msw-deep pens on the same day, provided the rules set out in Tables 4 and 5 are obeyed.

The improvements in productivity have occurred with continued downward trends in DCI episodes from the industry. This study demonstrated a fall in incidence of DCI when measured per number of dives, number of dive years and tonnage of fish production, over the last two decades. The reductions in DCI incidence have been maintained (Figure 7), despite relaxing the bounce limits as a result of this study. Other factors such as professional training of divers, appropriate use of dive tables, more effective diving procedures and substitution of tasks for some risky diving practices are likely to have contributed to this improvement in safety.[3] Had the industry incidence of DCI remained at 1990 levels, there would now be 44 cases of DCI treated at DDHM per annum based on incidence per 10,000 dives: or over 500 DCI cases per annum based on tonnage of fish. The industry has become more efficient regarding fish production and less diver-dependent for some tasks, such as net cleaning. The Tasmanian aquaculture industry is rapidly evolving, and with this evolution there are further changes in diving practices, and calls for greater flexibility. There have been requests to combine square dive profiles before or after bounce diving. We have received requests to assess deeper bounce-dive series, and also to complete the data collection on bounce diving in pens less than 9 msw. In addition the impact of nitrox diving, exercise post diving and ascents to altitude (very relevant in Tasmania) on decompression stress have yet to be tested.

Conclusions

This study has permitted significant improvements in productivity for the Tasmanian aquaculture industry between depths of 13 to 21 msw whilst maintaining a good safety record. Our data suggest that bounce diving was not a major factor causing DCI in Tasmania's aquaculture divers in the late 1980s and early 1990s. The industry is to be congratulated for embracing multiple improvements to diving procedures and improving diver training. In this research we have come full circle. A safety problem was detected and, with industry cooperation, controls were implemented, which were successful in reducing risk. Finally, we have been able to tailor some solutions to meet industry needs. It is a process of continual cooperation and evolution, and further study is ongoing.

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Retrospective review of grommet procedures under general versus local anaesthesia among patients undergoing hyperbaric oxygen therapy

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Abstract

(Lamprell L, Young D, Vangaveti V, Orton J, Suruliraj A. Retrospective review of grommet procedures under general versus local anaesthesia among patients undergoing hyperbaric oxygen therapy. *Diving and Hyperbaric Medicine*. 2014 September;44(3):137-140.)

Introduction: One significant side effect of hyperbaric oxygen treatment (HBOT) is middle ear barotrauma (MEBT) may require tympanostomy tube (grommet) insertion by the Ear, Nose and Throat service. Where timely HBOT is needed, routine insertion of grommets under local anaesthesia (LA) is becoming common.

Aims: To investigate the differences between patients receiving HBOT and concurrent grommets under LA versus general anesthesia (GA) at The Townsville Hospital (TTH).

Methods: A retrospective chart analysis of patients receiving HBOT between 2008 and 2012 and requiring grommets was undertaken.

Results: Thirty-one (5%) out of 685 patients treated with HBOT from 2008 to 2012 received grommets. Twelve cases received grommets under LA, and 19 under GA. Twenty out of the 31 cases had grommets following MEBT and the remainder prophylactically. Complications of grommet insertion comprised two cases with blocked grommets. There was a significant difference ($P = 0.005$) in the time in days from ENT referral to HBOT between the LA group (median 1 day, range 0–13 days) and the GA group (median 8 days, range 0–98 days).

Conclusion: A greater number of hyperbaric patients received grommets under GA than LA at the TTH. Insertion of grommets under LA was safe, offering advantages to both the patient and the treating team in the setting of HBOT-associated otic barotrauma.

Key words

Barotrauma, ear barotrauma, ENT, hyperbaric oxygen, hyperbaric oxygen therapy

Introduction

The Townsville Hospital Hyperbaric Medicine Unit (TTH HMU) is home to a state-of-the-art hyperbaric chamber and is the only facility in Queensland outside of Brisbane, servicing North and West Queensland, the Great Barrier Reef and South Pacific regions. One commonly encountered side effect of hyperbaric oxygen treatment (HBOT) is middle ear barotrauma (MEBT), which may require myringotomy and tympanostomy tube insertion by the Ear, Nose and Throat (ENT) service.[1] The practice of cannula insertion into the tympanic membrane (TM)as a rapid temporary tympanostomy is not used at TTH. Large elective surgery waiting lists coupled with the need for timely treatment for new HBOT referrals means the routine insertion of grommets under local anaesthesia (LA) is becoming more commonplace.[2,3]

The aim of this study was to investigate the differences between patients receiving HBOT and concurrent grommets under either general anaesthesia (GA) or LA in North Queensland.

Methods

Approval by the Townsville Hospital Human Research Ethics Committee was obtained (HREC/13/QTHS/58). A retrospective chart review was undertaken. Patients included in the study were required to have been treated by TTH HMU during the five-year period 2008 to 2012 and have received grommets in association with their hyperbaric treatment. Cases were identified from the unit's patient database, and the following data were collected from the patient charts and the database: indication for HBOT; demographic data; date of grommet; time between ENT referral for grommets insertion and recommencement (or commencement) of HBOT; use of LA or GA; grommet type; indication for grommet insertion; grade of MEBT at the time of grommet insertion; number of HBOT sessions before and after grommet insertion; grommet-related complications. Details of the initial HBOT consult were also recorded, particularly where this led to early ENT referral for consideration for prophylactic grommet insertion (i.e., otoscopy findings; observations of the patient's ability to clear their ears or communicate physical distress). Where a single patient had more than one course of HBOT, they were counted as a new case if a new grommet was inserted for the subsequent HBOT course.

STATISTICAL ANALYSIS

The IBM SPSS 22 (IBM, New York) software was employed to identify any significant differences between LA cases versus GA cases based on comparisons between the

Table 1
Summary of data comparing cases treated with grommets under local (LA) or general (GA) anaesthesia; * $P = 0.005$

	LA cases	GA cases
No. of patients	12	19
Male/female ratio	8/4	13/6
Age (median (range))	75 (43–88)	66 (43–78)
No. prophylactic grommets	5	6
No. post-barotrauma grommets	7	13
Days from referral to HBOT* (median (range))	1 (0–13)	8(0–98)
Post-grommet complications	0	2
No. pre-grommet HBOT (median (range))	2 (0–30)	0.5 (0–39)
No. post-grommet HBOT (median (range))	25 (2–40)	25 (0–58)

above-mentioned data. Data were checked for normality of distribution using the Kolmogorov-Smirnov test. The Mann-Whitney U Test was used to compare delays with re/commencing treatment between the two groups. The Wilcoxon signed ranks test was employed for comparing the number of treatments pre- and post-grommet insertion between the two groups. A P-value of < 0.05 was considered statistically significant.

Results

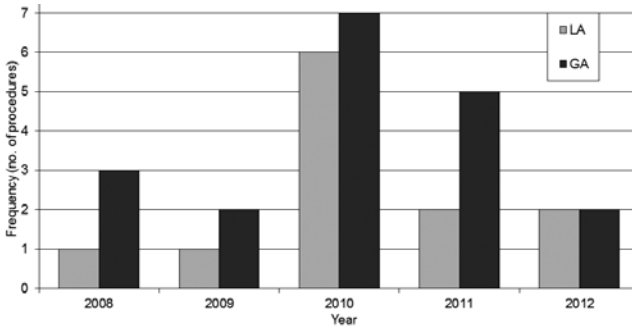
Thirty-one patients (5%) out of a total of 685 treated with HBOT from 2008 to 2012 received grommets to enable HBOT. Males outnumbered females in keeping with more males than females receiving HBOT at TTH HMU in a recent audit (147 males versus 70 females for 2010–2011).

There were some differences between those patients receiving grommets using GA compared to LA (Table 1). The median age of patients needing grommets was 75 (range 43–88) years for patients who received grommets under LA versus 66 (range 43–78) years for patients who received grommets under GA, although this was not statistically significant. Among these, two men underwent HBOT twice (counted as four male cases in total) with grommets inserted under GA for each. One of these patients had grommets inserted prophylactically prior to both HBOT courses, whereas the other had grommets prophylactically prior to their first HBOT course, but post MEBT for their second HBOT course.

Indications for HBOT included osteoradionecrosis (eight cases), problem wounds (six cases) and radiation proctitis (two cases). The less common indications were osteomyelitis, air embolism, tracheoesophageal fistula, Fournier's gangrene and necrotizing fasciitis.

GA grommet cases outnumbered LA cases (Table 1 and Figure 1). Types of LA used included liquid phenol applied to the tympanic membrane with an applicator, EMLA® cream (a eutectic mixture of lignocaine and prilocaine) and cophenylcaine spray. Three cases received Shah flanged grommets,17 had Shepard short-stay grommets and in 11 cases the type of grommet was undocumented.

Figure 1
Frequency of grommet procedures under GA versus LA between 2008 and 2012



The median time from ENT referral for grommets to the date of re/commencement of HBOT was eight days (range 0–98) for cases receiving GA grommets compared to 1 day (range 0–13) for cases receiving grommets under LA ($P = 0.005$).

Furthermore, 20 out of 31 cases had grommets post MEBT. Among the post-MEBT grommet cases, pre-treatment assessment of Eustachian tube function included documented history-taking (seven patients), success with a Valsalva manoeuvre (five patients), formal audiology (10 patients) and otoscopy (six patients). The difference in the number of post-MEBT cases treated with GA versus LA was not statistically significant.

The median number of HBOT prior to the insertion of grommets was one (range 0–39) indicating a generally short trial period of HBOT prior to the insertion in most cases. Overall, the median number of HBOT post insertion was 25 (range 0–58) treatments ($P = 0.001$ for the difference between the number of HBOT sessions pre-grommet versus post-grommet). Issues with HBOT prior to grommet insertion were dominated by MEBT (20 out of 31 cases) and slowed descents (three out of 31 cases). Issues arising with HBOT after grommet insertion included blocked grommets in two cases, cardiac issues in two cases, poor compliance and incompatibility with comorbid acute sinusitis and cellulitis respectively.

Discussion

Anecdotally, the ENT department in Townsville reports a few incidences of primary attempts at grommet procedures under LA that were later referred for a GA where the patient (often young) does not tolerate the procedure. Grommets will

also be inserted simultaneously under GA where the patient has another procedure planned near to the time of the ENT referral, such as a wound dressing change or washout and these cases were classified as a GA grommet for this study. The practice of cannula insertion as a rapid, temporary tympanostomy is not utilised at this facility, as the demand for tympanostomy in the setting of HBOT is satisfactorily met with conventional grommet insertion. The potential for prolonged HBOT is also locally viewed as optimally managed by grommet insertion.

The study found LA to be a safe and effective alternative to GA grommets with complications confined to two blocked grommets able to be cleared with conservative strategies. No grommets required removal and nil required re-insertion. However, the length of follow up, which was 6 months for patients treated in 2012, could be considered brief. As patient discomfort was not formally documented, an accurate measure of pain as a consequence of either LA or GA grommet insertion could not be studied.

MEBT AND HBOT

In a prospective study at TTH HMU of 106 patients using multivariate logistic regression, the local cumulative risk of MEBT was 35.8% in the first five HBOTs and 10.3% for needing tympanostomy tubes; 13.2% of the patients required tubes at any time during their HBOT course.[4] The predominant risk factors include Eustachian tube dysfunction, presence of an artificial airway, reduced level of consciousness, head and neck radionecrosis, nasal and paranasal disease, age over 55 years, female gender and previous middle ear surgery.[5] The practice of the TTH HMU is to assess patient risk of MEBT prior to treatment as described above, including history and otoscopic examination followed by a trial of otic equalization techniques. However, not all cases that demonstrated risk factors for MEBT on the initial assessment received grommets in the prophylactic setting. Reasons for this may include: the need for urgent HBOT to proceed; barriers to early access to ENT services; the ease of an initial trial of HBOT versus referring the patient for grommets. This may be influenced by the findings in the aforementioned TTH HMU study.[4] It was concluded that among this local population, it was not possible to accurately predict which patients needed tympanostomy tubes during their HBOT to substantiate a recommendation to place grommets prophylactically in any selected patients; a conclusion shared by others.[4,5]

MEBT is common in HBOT, with the potential for inner ear barotrauma in severe, but rare cases. The diagnosis is based on history and a confirmatory otoscopic examination with Edmond's classification of MEBT utilised at TTH HMU.[6] The only intervention to date is prevention of further MEBT by the cancellation of HBOT or by the insertion of grommets.[7]

The rate of insertion of grommets at TTH HMU reflects the degree of consideration being made for the risks of grommet insertion (cholesteatoma, otorrhoea, persistent TM perforation requiring myringoplasty, early extrusion, tympanosclerosis, retraction pockets, infection, ossicular damage) versus the benefit of not aborting further HBOTs.[8–10] It may also reflect the local tolerance by patients of MEBT in the setting of HBOT, or the efficacy of early education provided by the unit staff regarding MEBT preventative techniques during treatment.

LOCAL ANAESTHETICS AND GROMMETS

Local anaesthesia of the TM using iontophoresis was revived in the 1970s.[11] In 1988 the histologic changes in the TM in guinea pig models after application of different LA preparations was studied.[12] An observed loss of epithelium and mucosal cells with tetracaine recovered after three months, whereas hyperplastic connective tissue was seen with Bonain's solution (equal amounts of cocaine hydrochloride, menthol and phenol).[12] A more recent study failed to demonstrate any significant histologic difference in the healing TM among phenol, tetracaine or EMLA®.[13] A double-blind, controlled trial has compared injected anaesthesia versus EMLA concluding that EMLA, was equally effective with a lesser degree of invasiveness for the patient.[14] A 1991 study of the EMLA technique undertook pure tone audiometry before and after each procedure noting no evidence of ototoxicity with EMLA.[15]

More recently safe alternatives to EMLA have been sought, and phenol has been reported as a safe LA in 71 procedures.[16] Visual analogue measures of pain and overall satisfaction with the treatment experience in a double-blind, randomized trial of 41 patients found no statistically significant differences between tetracaine and EMLA.[3]

RESEARCH LIMITATIONS AND FUTURE DIRECTIONS

The main limitation of this study is the variability in the adequacy of documentation regarding grades of MEBT, types of LA being used, patient discomfort and the pre-HBOT assessment of Eustachian tube dysfunction. Future research should examine the rate of audiology complications between patients receiving grommets in association with their HBOT versus patients with MEBT who do not receive grommets. Also a review of cost differences between grommet procedures under LA and GA may lead to the increased usage of LA techniques.

CONCLUSION

Grommet insertion under LA was associated with shorter timeframes to HBOT. In this study, more patients received grommets under GA. Factors influencing a higher local rate of GA grommets may be the convenience of simultaneous grommet insertion with an upcoming GA procedure, a

surgeon's preference for GA insertion or the weight given to minimising patient discomfort during grommet insertion.

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The database of randomised controlled trials in hyperbaric medicine maintained by Michael Bennett and his colleagues at the Prince of Wales Hospital Diving and Hyperbaric Medicine Unit, Sydney is at:
<http://hboevidence.unsw.wikispaces.net/>

Assistance from interested physicians in preparing critical appraisals is welcomed, indeed needed, as there is a considerable backlog. Guidance on completing a CAT is provided. Contact Associate Professor Michael Bennett: <M.Bennett@unsw.edu.au>

The cardiac effects of hyperbaric oxygen at 243 kPa using in-chamber echocardiography

Ian C Gawthrope, David A Playford, Benjamin King, Kathryn Brown, Catherine Wilson and Barry McKeown

Abstract

(Gawthrope IC, Playford DA, King B, Brown K, Wilson C, McKeown B. The cardiac effects of hyperbaric oxygen at 243 kPa using in-chamber echocardiography. *Diving and Hyperbaric Medicine*. 2014 September;44(3):141-145.)

Introduction: The adverse effects of hyperbaric oxygen (HBO) on cardiac physiology are considered a potential hazard during the treatment of some patients. The haemodynamic effects of HBO are poorly understood and the incompatibility of electrical equipment inside the chamber has made assessment difficult. At Fremantle Hyperbaric Unit, we have modified an ultrasound machine (Logiq™ e) for safe use within the hyperbaric environment. The aim of this study was to evaluate the cardiac changes that occur during HBO using in-chamber transthoracic echocardiography (TTE) in subjects without evidence of active cardiac disease.

Methods: Eleven patients and nine members of staff underwent comprehensive TTE examinations before and during HBO administered at a pressure of 243 kPa. The TTE examinations were reported by two independent cardiologists and statistically evaluated using paired Student's *t*-tests.

Results: There was a significant decrease in heart rate during HBO (65 vs. 70 bpm on air at atmospheric pressure, $P = 0.002$) resulting in a decrease in cardiac output (5.3 vs. 5.9 $L \cdot min^{-1}$, $P = 0.003$). Left ventricular outflow tract (LVOT) dimension was larger during HBO than baseline imaging (2.30 vs. 2.23 cm, $P = 0.0003$). LVOT velocity time integrals (VTI) decreased (19.9 vs. 21.5 cm, $P = 0.009$) and therefore a similar stroke volume was maintained (61 vs. 65 ml, $P = 0.5$). Ventricular and atrial volumes, intracardiac flows and minor valvular abnormalities were not significantly affected by HBO. No adverse cardiac effects were observed.

Conclusions: TTE can be safely performed within a hyperbaric chamber. Cardiac physiology is not adversely affected by HBO in individuals without active cardiac disease.

Key words

Physiology, cardiovascular, hyperbaric oxygen, echocardiography, hyperbaric research

Introduction

A number of potentially adverse changes occur in the cardiovascular system in response to hyperbaric oxygen (HBO), and these remain relatively little studied, in part owing to the incompatibility of electronic equipment inside the chamber.[1–5] At the Fremantle Hyperbaric Unit we have become the first, to our knowledge, to develop an ultrasound machine capable for use inside the chamber.[6]

HBO treatment is used in a wide range of patients for a variety of conditions including wound healing, delayed radiation tissue damage, necrotising infections and diving-related indications. Many of these patients are elderly with significant co-morbidities and the risk factors for the development of their primary complaints are similar to the potential risks for underlying cardiac disease. Chamber attendants are also subject to the physiological effects of breathing HBO.

Echocardiography continues to develop as an important tool in the recognition of cardiac disease and assessment of cardiac function. Previous literature has documented transthoracic echocardiography (TTE) findings before and after HBO.[7] Limited TTE studies have also been performed in hyperbaric conditions with the machine external to the chamber using the subject or an individual separate to the machine to acquire the images.[8,9] Both these studies highlighted some difficulties of imaging with the machine external to the chamber. Actual in-chamber 2-D TTE of subjects has never been performed.

The aim of this study was to evaluate the cardiac changes that occur during HBO using in-chamber TTE in subjects without evidence of active cardiac disease.

Methods

The study was approved by the Western Australian South Metropolitan Area Health Service Human Research Ethics Committee (approval no: 10/478), and conducted according to the principles of the Helsinki Declaration (revised 2008). Informed written consent was obtained from all subjects.

As previously described, with the assistance of Fremantle Hospital Biomedical Services and using available guidelines and recommendations, an ultrasound machine (Logiq™ e, GE Healthcare) was modified for safe use within the chamber.[6] The ultrasound machine had a cardiac software package and images were acquired with a 3 MHz cardiac probe. The cardiac software available did not have tissue Doppler capability.

Table 1
Acronyms used for physiological terms in this paper

LV – left vetricle
RV – right ventricle
LA – left atrium
RA – right atrium
LVOT –left ventricular outflow tract
RVOT – right ventricular outflow tract
VTI – velocity time integral
TR – tricuspid regurgitation
EDV –end diastolic volume
ESV – end systolic volume
EF – ejection fraction

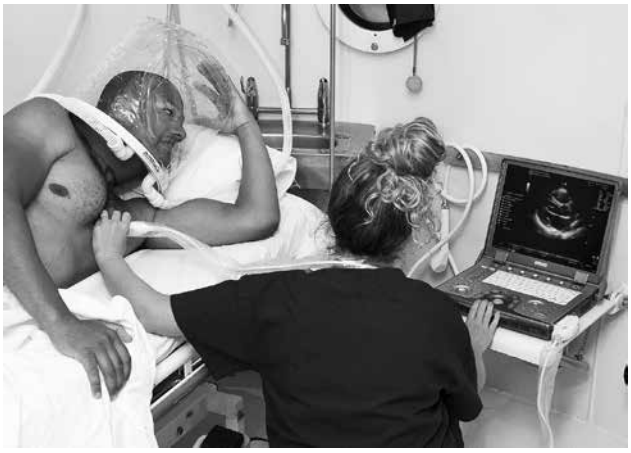
The subjects were a convenience sample of volunteers either being treated or working at Fremantle Hyperbaric Unit during the years 2011 and 2012. Patients and staff were recruited when there was both available space in the chamber and an available sonographer to conduct the examination. Examinations took place singularly within the chamber so privacy was not an issue. Patients undergoing HBO treatment for a range of conditions and available members of staff underwent TTE immediately before and during HBO. The patients were examined during their routine treatment and staff examined under the exact same conditions following 30 minutes of HBO. The chamber was pressurised to 243 kPa and 100% oxygen given through an Amron™ head hood at 30 $L \cdot min^{-1}$. The subjects were imaged on a trolley within the chamber in the left lateral position (parasternal long and short axis and apical views) and supine (subcostal views) as per a routine TTE examination (Figure 1).

A certified cardiac sonographer performed a comprehensive TTE examination. Assessments were made at room pressure breathing air before pressurisation and at pressure breathing 100% oxygen of cardiac chamber volumes and function; valve function; inflow velocities and outflow velocity time integrals and heart rate (see Table 1 for list of acronyms used). LV stroke volumes were calculated from LVOT VTI and LVOT diameter data. Cardiac outputs were derived from stroke volume and heart rate.

The TTE examinations were reported by two independent cardiologists blinded to the pressure, and subsequently reviewed if there was a discrepancy between the reported results. A consensus decision was then made on the findings.

The data were statistically evaluated using SPSS version 20. Paired Student's *t*-tests compared surface air and HBO measurements. Parameters were tested for normality of distribution before comparisons were made. Agreements between tests were measured using Kappa tests and correlations using Pearson tests. Significance was accepted as a *P*-value of 0.05 or less.

Figure 1
Subject undergoing echocardiography at 243 kPa pressure whilst breathing 100% oxygen from a head hood



Results

Eleven patients and nine members of staff were recruited. The 20 subjects (13 male, 7 female) were aged 48.8 (SD 15.7) years, their mean weight was 77.8 (SD 15.7) kg and body mass index 25.7 (SD 4.8) $kg \cdot m^{-2}$.

There were no significant differences between the patient and staff groups when compared using non-parametric testing. Therefore, the data were pooled for analysis. The TTE recorded measurements are shown in Table 2. Where possible, data were obtained under both surface air and HBO conditions, excepting that there was insufficient TR to estimate the right ventricular systolic pressure as paired data in 15 subjects (i.e., insufficient TR at atmospheric pressure, during HBO or both).

VALVULAR REGURGITATION AND STENOSIS

On surface imaging, there were 12 subjects with no TR, and five subjects with trace TR. There was one subject each with mild and moderate TR. During HBO imaging, TR was absent in 13 subjects and mild in four subjects. The apparent difference in severity of TR between surface air and HBO could not be compared statistically because of the small sample size. There was a sufficient envelope for estimation of right ventricular systolic pressure in four subjects during atmospheric imaging and two subjects during HBO conditions. HBO did not appear to have an important effect on the degree of TR. At atmospheric pressure on air, one subject had moderate aortic regurgitation, five subjects had mild pulmonary regurgitation, four had mild mitral regurgitation and another had moderate mitral regurgitation. No subjects had significant valvular stenosis. HBO conditions did not change the severity of valvular regurgitation identified at atmospheric pressure in any of the subjects.

Table 2

Haemodynamic data from surface air and hyperbaric oxygen (HBO) at 243 kPa derived from trans-thoracic echocardiographic imaging; * sample size too small for statistical comparison; † no correlation between measurements during atmospheric and HBO imaging (data presented for completeness)

Variable	*n*	Surface air Mean (SD)	HBO at 243 kPa Mean (SD)	*P*-value
LV diastolic diameter (cm)	16	4.8 (0.5)	4.7 (0.4)	0.3
LV diastolic volume (mL)	19	107.2 (22.9)	102.7 (25.8)	0.2
LV systolic volume (mL)	19	41.4 (17.5)	42.5 (18.8)	0.8
LV stroke volume (mL)	18	64.5 (14.6)	61.4 (15.2)	0.5
LV ejection fraction (%)	19	62.3 (9.6)	60.6 (10.5)	0.5
RA area (cm^2)	14	14.6 (3.4)	15.6 (2.6)	0.2
LA volume index ($mL \cdot m^{-2}$)	19	39.5 (11.4)	39.3 (14.0)	0.8
Aortic valve mean gradient (mmHg)	14	4.2 (1.6)	3.7 (1.2)	0.09
Aortic valve VTI (cm)	14	29.5 (5.8)	29.9 (5.7)	0.99
LV outflow tract (OT) diameter (cm)	20	2.23 (0.25)	2.30 (0.25)	<0.001
LVOT VTI (cm)	19	21.5 (6.0)	19.9 (6.3)	0.009
Heart rate (bpm)	20	69.7 (11.8)	64.9 (11.3)	0.002
Cardiac output ($L \cdot min^{-1}$)	19	5.9 (2.4)	5.3 (2.2)	0.003
Mitral inflow E wave ($cm \cdot s^{-1}$)	20	71.5 (22.5)	74.7 (25.7)	0.2
Mitral inflow A wave ($cm \cdot s^{-1}$)	18	59.1 (16.9)	55.3 (15.0)	0.2
Mitral inflow E:A ratio	18	1.4 (0.5)	1.5 (0.6)	0.06
E wave deceleration time (ms)†	19	219.0 (48.0)	227.0 (54.6)	0.6
Pulmonary vein S wave ($cm \cdot s^{-1}$)	12	43.9 (13.0)	44.1 (16.7)	0.9
Pulmonary vein D wave ($cm \cdot s^{-1}$)	12	44.0 (15.6)	45 6 (14.9)	0.9
TR peak velocity ($m \cdot s^{-1}$)*	4	2.6 (0.2)	2.2 (0.0)	n/a
RVOT VTI (cm)	18	16.0 (4.7)	14.4 (4.0)	0.07
Estimated pulmonary artery systolic pressure (mmHg)	5	32.5 (9.1)	30.7 (8.5)	0.3

CHAMBER DIMENSIONS AND VOLUMES

The LV dimensions using the standard, parasternal long-axis basal dimension, or apical LV volumes using Simpson's method,[10] were not different during atmospheric or HBO imaging (Table 2). However, the LVOT dimension was larger during HBO imaging than at atmospheric pressure (2.30 cm vs. 2.23 cm, $P = 0.0003$). Both the LA and RA sizes (volume and area, respectively) were no different between atmospheric and HBO imaging.

CARDIAC OUTPUT

LV stroke volume was measured using both LVOT (LVOT VTI and LVOT dimension) and Simpson's method (LVEDV – LVESV). The LV stroke volume and LV ejection fraction were no different between surface and HBO imaging by either of the two methods for their measurement. Along with the increase in the LVOT dimension under HBO conditions, the LVOT VTI decreased (21.5 cm vs. 19.9 cm, $P = 0.009$), thus maintaining a similar stroke volume.

There was a significant decrease in heart rate during HBO conditions (65 bpm during HBO vs 70 bpm at atmospheric pressure, $P = 0.002$). As a result, there was a significant decrease in cardiac output during HBO conditions (mean 5.3 $L \cdot min^{-1}$ vs. 5.9 $L \cdot min^{-1}$ at on surface air, $P = 0.003$; Table 2).

INTRA-CARDIAC FLOWS

There was no statistical difference between surface air and HBO conditions for mitral inflow E wave, A wave, mitral deceleration time, or pulmonary vein flows (Table 2). There was a trend toward a lower mitral inflow E:A ratio at ambient pressure vs. HBO (1.4 vs. 1.5 $m \cdot s^{-1}$, $P = 0.06$), consistent with the higher (but non-significant) early trans-mitral flows. Right-sided flows, reflected in the RVOT VTI measurements, trended toward lower values during HBO (14.4 vs. 15.8 cm, $P = 0.07$). The RVOT dimension was not measured during the study because of variable image quality of the region of interest.

INTERNAL CONSISTENCY OF DATA

To confirm the internal consistency of the data, correlational analysis was performed between the two conditions for each measurement described. Strong correlations ($r > 0.9$, $P < 0.001$) were found between surface air and HBO measurements for most variables, and similarly strong agreements were found using Kappa tests. Lesser degrees of agreement were found between Simpson's-derived cardiac output and ejection fraction ($r = 0.5$, $P = 0.02$ and $r = 0.6$, $P = 0.005$, respectively) hence LVOT-derived cardiac output and PLAX-derived LV EF were presented in Table 2 ($r = 0.96$, $P < 0.001$; $r = 0.9$, $P < 0.001$, respectively). No correlation

was observed between mitral deceleration time data pairs.

Discussion

Our study describes the cardiac physiology in response to HBO administered at 243 kPa. We describe that TTE is feasible and safe to perform inside a hyperbaric chamber. No adverse cardiac responses were observed in our group of individuals without evidence of active cardiac disease. Our findings provide a basis by which future studies on the cardiovascular effects of HBO could be considered in patients with cardiac disease. The observed fall in cardiac output during HBO in our study is a result of a decrease in heart rate. There was no significant change in stroke volume despite an increase in LVOT dimension and a decrease in LVOT flow. Ventricular and atrial volumes, intracardiac flows and minor valvular abnormalities were not affected importantly by HBO conditions.

It has been well documented that during HBO there is a decrease in cardiac output, primarily owing to bradycardia and increased afterload.[1–4] This decrease has previously been attributed to hyperoxia alone since, in animal models, cardiac output and heart rate do not significantly change under normoxic hyperbaric conditions.[3] However, other mechanisms may also play a role: animal models have demonstrated discrepancies between myocardial oxygen supply and demand, and the direct effect of hydrostatic pressure on cardiac pacemaker function may cause bradycardia.[2,11] There is no clear effect of HBO on myocardial contractility in either animal or human studies.[12–14] Our data show a reduction in heart rate, which appears to be the primary driver for the decrease in cardiac output, LVEF and stroke volume, both indirect measures of LV contractility, did not change.

Acute pulmonary oedema is considered a potential hazard during the treatment of patients with HBO. Case reports estimate the incidence of pulmonary oedema to be approximately 1 in 1,000 patients treated.[5,15] A postulated explanation for this was a disturbance in ventricular balance in patients with congestive cardiac failure.[5] Congestive cardiac failure remains a relative contra-indication to HBO treatment. In our study, we were unable to demonstrate any change in intracardiac flows or measures of left ventricular function during HBO. From our data, in a small patient cohort with no evidence of active heart disease, it appears that HBO does not predispose an individual to pulmonary oedema due to abnormal left ventricular systolic and/or diastolic function.

STUDY LIMITATIONS

Because of the relatively small number of subjects (20), we may have been unable to identify minor cardiac physiological effects of HBO. This includes trends observed in decreases in mitral inflow E:A ratio or RVOT VTI. We did not measure the effect of normoxic hyperbaric conditions, so are unable to exclude an effect of hyperbaric conditions specifically in the absence of hyperoxia.

We considered the possibility of variability in the echo imaging between HBO and surface conditions as an explanation for the results obtained. However point-to-point variation (test–retest variability) was extremely small between sonographer and independent observer. The overall agreement between atmospheric and HBO parameters was also good. Measurements were performed only on images felt to be of good quality.

Imaging of subjects at atmospheric and HBO conditions was slightly different in that a small positive pressure must be attained within the oxygen hood (maximum pressure < 1 cm H_2O) in order to prevent its collapse. Although we cannot exclude a minor effect from the positive pressure on cardiac physiology, we did not feel this to be an important factor.

An increase in the LVOT dimension under HBO conditions was not expected. There are no published data on the behaviour of the LVOT under HBO-loading conditions, so we are unable to verify our results from other studies. At atmospheric pressure, the LVOT does not vary significantly on repeated studies. However in our study, the increase in LVOT dimension was found by the sonographer performing the study, and by two independent cardiologists reviewing the study and blinded to the other analyses. Further, the increase in LVOT dimension offset the observed decrease in the LVOT VTI, preserving the stroke volume. The decrease in cardiac output we observed was driven by the decrease in heart rate under HBO conditions, rather than by a change in the LVOT dimension. Based on these observations, we consider this small increase to be a real phenomenon.

Conclusions

We have demonstrated that TTE is feasible within a hyperbaric chamber, and that cardiac physiology is not adversely affected by HBO conditions in patients and volunteers without evidence of active cardiac disease. The decrease in heart rate observed with HBO appears to drive the decrease in cardiac output, with no evidence for adverse effects of HBO on intracardiac flows or chamber volumes. Further study of the effects of HBO is required in individuals with significant cardiac disease.

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Conflicts of interest: nil

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Transcutaneous oximetry: normal values for the lower limb

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Abstract

(Blake DF, Young DA, Brown LH. Transcutaneous oximetry: normal values for the lower limb. *Diving and Hyperbaric Medicine*. 2014 September;44(3):146-153.)

Introduction: Current guidelines for transcutaneous oximetry measurement (TCOM) for the lower limb define tissue hypoxia as a transcutaneous oxygen partial pressure < 40 mmHg. Values obtained with some newer machines and current research bring these reference values into question.

Aim: To determine 'normal' TCOM values for the lower limb in healthy, non-smoking adults using the TCM400 oximeter with tc Sensor E5250.

Method: Thirty-two healthy, non-smoking volunteers had TCOM performed at six positions on the lower leg and foot. Measurements were taken with subjects lying supine breathing air, then with leg elevated and whilst breathing 100% oxygen.

Results: Room-air TCOM values (mean mmHg, 95% confidence interval (CI)) were: lateral leg 41.3, CI 37.8 to 44.7; lateral malleolus 38.6, CI 34.1 to 43.1; medial malleolus 43.9, CI 40.2 to 47.6; dorsum, between first and second toe 39.3, CI 35.9 to 42.7; dorsum, proximal to fifth metatarsal-phalangeal joint 46.4, CI 43.4 to 49.3; plantar 52.3, CI 49.6 to 55.1. Using the currently accepted value of less than 40 mmHg for tissue hypoxia, 24 of our 32 'healthy' subjects had at least one air sensor reading that would have been classified as hypoxic. Seventeen subjects had TCOM values less than 100 mmHg when breathing 100% normobaric oxygen.

Conclusion: Normal lower limb TCOM readings using the TCOM400 with tc Sensor E5250 may be lower than 40 mmHg, used to define tissue hypoxia, but consistent with the wide range of values found in the literature. Because of the wide variability in TCOM at the different sensor sites we cannot recommend one TCOM value as indicative of tissue hypoxia. A thorough clinical assessment of the patient is essential to establish appropriateness for hyperbaric oxygen treatment, with TCOM used as an aid to help guide this decision, but not as an absolute diagnostic tool.

Key words

Transcutaneous oximetry, hyperbaric oxygen therapy, wounds, patient monitoring, standards

Introduction

Transcutaneous oximetry measurement (TCOM) is the process of measuring the tissue partial pressure of oxygen through the skin. The technique was originally used in neonatology but has now become an essential component of wound assessment in hyperbaric medicine.[1] TCOM estimates tissue oxygenation non-invasively by measuring the diffusion of extracellular oxygen into a heated sensor on the skin. Confirmation of tissue hypoxia and demonstrated responsiveness of the tissue to oxygen in the area surrounding a wound allows selection of patients most likely to benefit from hyperbaric oxygen treatment (HBOT).[2] TCOM also provides useful information for patients requiring further vascular assessment and assists in determining amputation levels.[3]

Previous studies of TCOM in healthy individuals found values in the lower limb varied from 48 to 79 mmHg.[4–7] Values obtained with some newer machines and sensors bring these values into question. Reviews have defined lower limb hypoxia as a transcutaneous oxygen partial pressure ($P_{tc}O_2$) of less than 40 mmHg.[2,8,9] However, this single reference value may not be an accurate normal value for all points on the lower limb. A recent study found different 'normal' TCOM values for different areas of the upper limb.[10] As TCOM values are currently considered fundamental in determining suitability of patients for HBOT, it is essential to know normal reference values. The aim of this study was to establish normal TCOM values in various areas of the lower limb in healthy, non-smoking adult subjects.

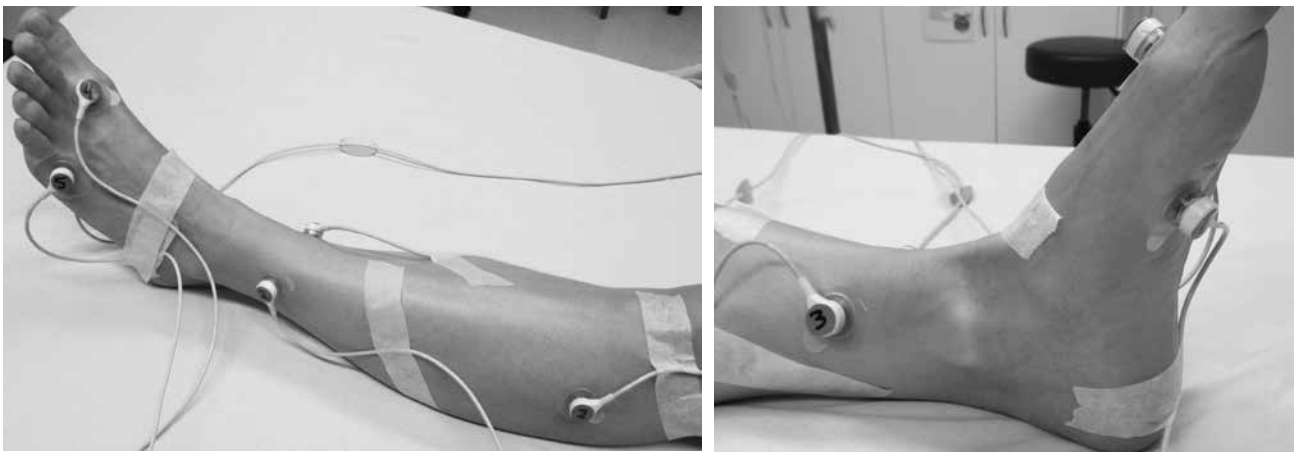
Methods

Ethics approval for this study was granted by the Human Research Ethics Committee of the Townsville Hospital and Health Service (HREC/12/QTHS/209). Thirty-two (16 male, 16 female) healthy volunteers were recruited from the hospital staff and general population to participate in the study. Exclusion criteria included subjects younger than 18 years old; current or former smoker; known cardiovascular disease including treated or untreated hypertension; significant respiratory disease or any other significant medical condition. Subjects with only one leg, significant scarring, or a skin condition on the lower limb, were also excluded. As subjects were required to have a plastic hood placed over their head to receive oxygen during part of the study, severe claustrophobia was a further exclusion criterion.

All participants were given a study information sheet and informed consent was obtained. Subjects refrained from consuming food or caffeine or performing heavy exercise

Figure 1

Transcutaneous oximetry measurement: placement of the six sensors on the lower limb
Sensor 1 – 10 cm distal to the lateral femoral epicondyle
Sensor 2 – 5 cm proximal to the anterior aspect of the lateral malleolus
Sensor 3 – 5 cm proximal to the centre of the medial malleolus
Sensor 4 – Dorsum of the foot between the 1st and 2nd metatarsal heads away from any obvious veins
Sensor 5 – Dorsum of the foot proximal to the head of the 5th metatarsal
Sensor 6 – Plantar 1st metatarsal area (proximal to the fat pad at the base of the great toe)



for two hours prior to participating in the study. The study was performed at sea level. Subjects were placed in a supine position on a hospital bed with their head slightly raised on one pillow for the duration of the study. They were offered a blanket for comfort and to limit any vasoconstrictive effects of being cold. The room temperature was maintained between 22.0 and 22.5^{O}C (the ambient temperature recommended by the TCOM manufacturer). The participants rested quietly while the sensors were placed.

Basic demographic data were collected including height and weight. Oxygen saturation and blood pressure were measured on both arms. Dorsalis pedis and posterior tibial pulses were recorded for both legs. Ankle brachial index (ABI) and toe pressures were also measured.

Participants were randomized to have six sensors placed on either their right or left leg (Figure 1). The sensor sites were prepared by shaving hair if necessary, wiping clean, rubbing with an alcohol swab and drying with gauze. One sensor was positioned 10 cm distal to the lateral femoral epicondyle and two sensors were each placed 5 cm proximal to the lateral and medial malleoli. Two sensors were placed on the dorsum of the foot attempting to avoid large superficial vessels, one between the first and second metatarsal heads and the second proximal to the fifth metatarsal-phalangeal (MTP) joint. The final sensor was placed on the plantar aspect of the foot proximal to the first metatarsal-phalangeal joint. The leads were secured in place with tape to prevent pull on the sensors. Subjects were requested to keep talking to a minimum during the study.

All TCOM assessments were performed by the same technician using the TCM400 Transcutaneous (tc) pO_2 Monitoring System with tc Sensor E5250 (Radiometer Medical ApS, Bronshoj, Denmark) which can record $P_{tc}O_2$ data from six tc E5250 sensors simultaneously. The electrode temperatures were pre-set to 44^{O}C and atmospheric and zero-point electrode calibrations were performed as per the manufacturer's recommendations. A humidity correction factor was calculated from the room temperature, saturated water vapour pressure and relative humidity, and input into the machine according to the TCM400 operator's manual.[11] The TCM400 displays $P_{tc}O_2$ values in units of mmHg.

We used the TCOM protocol described by Sheffield, which has been used historically in hyperbaric medicine to identify tissue hypoxia and responsiveness to hyperoxia.[12,13] Initial normobaric, room-air readings from all sensors were recorded after a minimum 20-minute equilibration period that allowed all sensors to stabilize.[4] The leg was then elevated 45° above its resting level and placed on a foam wedge, with sensor readings recorded after 5 minutes. The leg was returned to the horizontal position for a minimum 5-minute period allowing all sensor readings to re-stabilize, and another set of readings were recorded to ensure TCOM had returned to baseline. The subjects then breathed 100% oxygen at a flow rate of 15 L·min^{-1} for 10 minutes via a clear plastic hood with a soft neck seal, with sensor readings recorded at the end of the 10-minute period, once stabilized (a pilot study demonstrated that 10 minutes was sufficient to reach stable levels). All sites were inspected for thermal injury. All collected data were de-identified and entered

Table 1
Demographic and baseline characteristics of the 32 subjects; means and SD or number (n) shown;
* $P < 0.05$ for difference between male and female

Variable	Males (n = 16)		Females (n = 16)		All (n = 32)	
Age (years) *	45.1	(10.6)	53.8	(12.3)	49.4	(12.1)
< 50 years old (n)	5		10		15	
Body mass index (kg·m^{-2})	27.2	(2.9)	27.2	(3.8)	27.2	(3.3)
Normal weight (BMI = 20–25) (n)	3		4		7	
Overweight (BMI > 25–30) (n)	11		9		20	
Obese (BMI > 30) (n)	2		3		5	
Systolic BP (L) (mmHg) *	124	(7)	117	(9)	121	(9)
Systolic BP (R) (mmHg)	124	(8)	120	(11)	122	(10)
Diastolic BP (L) (mmHg) *	80	(9)	72	(6)	76	(9)
Diastolic BP (R) (mmHg)	76	(6)	73	(6)	74	(6)
SpO_2 (L) (%) *	97.4	(1.0)	98.4	(1.1)	97.9	(1.2)
SpO_2 (R) (%)	97.7	(1.1)	98.3	(1.0)	98.0	(1.1)
Heart rate (beats·min^{-1})	68	(9)	65	(14)	66	(12)
Ankle brachial index	1.09	(0.07)	1.08	(0.07)	1.09	(0.07)
Toe brachial index	0.78	(0.11)	0.78	(0.15)	0.78	(0.13)
Toe systolic BP (mmHg)	98.5	(15.4)	94.6	(17.7)	96.5	(16.5)

into a pre-formatted Excel spreadsheet. These data were then exported into Stata Statistical Software: Release 11 (StataCorp LP, College Station, TX, USA) for analysis.

ANALYSIS

The primary outcome of this study was a determination of the normal range of TCOM readings when measured at various places on the leg of healthy, volunteer subjects. Based on previous reports of mean normal TCOM readings ranging from 52 to 70 mmHg with a standard deviation of approximately 10 mmHg,[4–7] our sample size of 32 subjects was intended to allow us to estimate mean TCOM readings with a 95% CI of ± 3.5 mmHg. Having 16 male and 16 female subjects also provided 80% power (with $\alpha = 0.05$) to detect a 10 mmHg difference in mean TCOM readings of males versus females using Student's t-test.

Demographic characteristics of male and female subjects were compared using Fisher's Exact Test or Student's t-test as appropriate. Descriptive statistics are reported for TCOM readings at each of the six sensor sites: mean and 95% confidence interval (CI) for the mean are reported when data are normally distributed; median, inter-quartile range and approximate 95% CI for the median are reported for non-parametric data. Differences between mean TCOM measurements for males and females were compared using t-tests when data were normally distributed, and using the Wilcoxon rank sum test for non-parametric data. Correlations between baseline perfusion measures of systolic blood pressure (SBP), diastolic blood pressure (DBP), oxygen saturation (SpO_2) and toe SBP in the randomized limb and the observed room-air and on-oxygen TCOM readings at each sensor site were evaluated using linear regression or Spearman's rank correlation for normal and non-parametric data respectively, with Bonferroni correction for multiple comparisons.

Results

Demographic and baseline data are shown in Table 1. The subjects ranged in age from 22 to 80 years. Female subjects were older than male subjects (mean age, 53.8 vs. 45.1 years); mean left-sided systolic blood pressure, diastolic blood pressure and oxygen saturation also differed statistically between female and male subjects, but these differences were clinically irrelevant. Baseline measures of perfusion were clinically unremarkable in all subjects.

The surface-air TCOM readings for each sensor site were normally distributed, both in the aggregate and for males and females separately. The leg-elevated and on-oxygen TCOM readings were not normally distributed. The mean, 95% CI and minimum and maximum values for the room-air sensor readings are shown in Table 2. Female subjects had higher room-air TCOM readings at the lateral leg sensor (44.8 versus 33.7 mmHg, $P = 0.04$), otherwise there were no differences in the mean room-air TCOM readings for female and male subjects. The median, inter-quartile range, 95% CI for the median, and minimum and maximum values for the leg-elevated and on-oxygen sensor readings are shown in Table 3. Female subjects had higher leg-elevated TCOM readings than male subjects at the lateral leg sensor site (median 39.5 vs. 32.0, Wilcoxon Rank Sum test, $P = 0.04$); there were no significant differences in the leg-elevated and on-oxygen TCOM readings for female and male subjects at any of the sensor sites (Wilcoxon Rank Sum test, all $P \geq 0.05$, data not shown). No evidence of skin injury was found.

Table 2
Mean and 95% confidence interval (95% CI) for TCOM readings for each sensor breathing room air (mmHg); * $P = 0.04$

Sensor	Male (n = 16)	Female (n = 16)	All (n = 32)
Lateral leg*			
Mean (95% CI)	37.7 (32.6–42.8)	44.8 (40.6–49.0)	41.3 (37.8–44.7)
Range	13–51	29–59	13–59
$n < 40$ mmHg	9	3	12
Lateral ankle			
Mean (SD)	41.0 (33.5–48.5)	36.2 (31.3–41.0)	38.6 (34.1–43.1)
Range	13–61	12–48	12–61
$n < 40$ mmHg	8	10	18
Medial ankle			
Mean (SD)	43.9 (37.8–50.1)	43.8 (39.4–48.2)	43.9 (40.2–47.6)
Range	13–65	29–58	13–65
$n < 40$ mmHg	5	4	9
Dorsum, 1st & 2nd toe			
Mean (SD)	41.0 (36.8–45.2)	37.6 (32.2–42.9)	39.3 (35.9–42.7)
Range	24–53	21–59	21–59
$n < 40$ mmHg	8	8	16
Dorsum, 5th toe			
Mean (SD)	45.8 (41.4–50.1)	47.0 (42.9–51.1)	46.4 (43.4–49.3)
Range	21–60	34–59	21–60
$n < 40$ mmHg	4	3	7
Plantar, 1st MTP			
Mean (SD)	53.3 (48.8–57.8)	51.4 (48.2–54.6)	52.3 (49.6–55.1)
Range	39–70	37–63	37–70
$n < 40$ mmHg	2	1	3

Figure 2 displays graphically the room-air TCOM readings for all study subjects at all sensor sites, showing several TCOM readings below 40 mmHg, particularly for the proximal sensors. Twenty-four of the 32 subjects had at least one room-air TCOM reading below 40 mmHg. Sixteen subjects had at least one on-oxygen sensor reading less than 100 mmHg (Table 3). Eleven had multiple readings less than 100 mmHg: eight with two sensors, two with three sensors and one with four sensors. Of the 31 on-oxygen sensor readings less than 100 mmHg, all but four of these same sensors (in three subjects) had also exhibited decreases in TCOM of at least 10 mmHg with leg elevation. None of the sensors recorded very low (i.e., TCOM < 30 mmHg) on-oxygen readings. The average change with leg elevation in those sensors with on-oxygen TCOM < 100 mmHg was -13.5 mmHg, with the biggest change being -24 mmHg and the smallest being -5 mmHg. We were unable to discern a pattern to either the decrease with leg elevation and initial values or to the response to oxygen and initial values.

Figure 2
Distribution of TCOM readings at each sensor site on the lower limb on room air (< 40 mmHg is currently regarded as hypoxic)

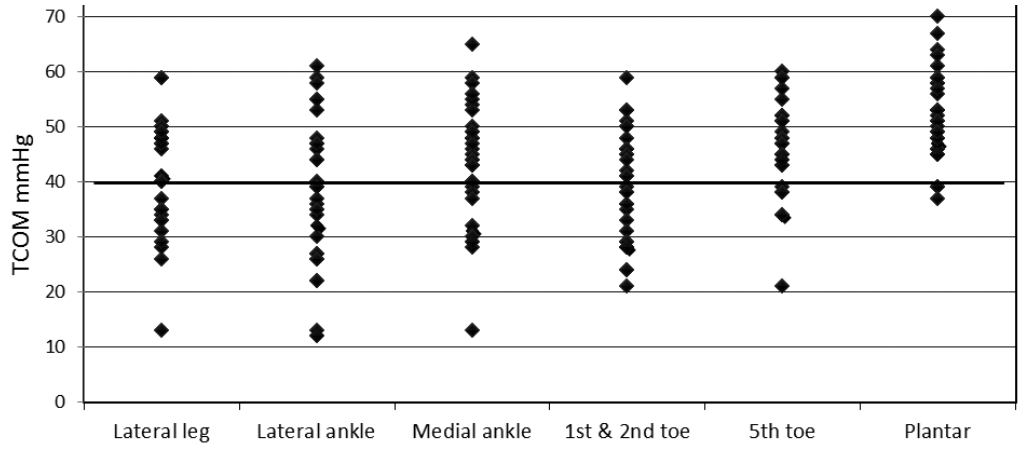


Table 3
Median (inter-quartile range) and 95% confidence interval (95% CI) for TCOM readings for each sensor; subject breathing room air with leg elevated and subject breathing 100% oxygen with leg level (mmHg).

Sensor	Room air, leg elevated	100% oxygen, leg level
Lateral leg		
Median (IQR)	34.5 (27.5–40.0)	241.5 (203.5–307.5)
95% CI	31.0–40.0	207.0–279.5
Range	4–55	130–366
$n \geq 10$ mmHg drop	8	n/a
$n < 100$ mmHg, oxygen	n/a	0
Lateral ankle		
Median (IQR)	29.0 (12.5–34.5)	200.0 (158.0–279.0)
95% CI	14.0–32.5	164.0–241.0
Range	1–44	53–337
$n \geq 10$ mmHg drop	23	n/a
$n < 100$ mmHg, oxygen	n/a	2
Medial ankle		
Median (IQR)	31.5 (25.5–36.5)	213.5 (158.5–275.0)
95% CI	27.0–36.0	176.0–269.5
Range	6–60	55–389
$n \geq 10$ mmHg drop	20	n/a
$n < 100$ mmHg, oxygen	n/a	1
Dorsum, 1st and 2nd toe		
Median (IQR)	27.5 (16.0–34.5)	137.5 (72.5–195.5)
95% CI	17.5–30.0	101.0–180.5
Range	1–47	45–384
$n \geq 10$ mmHg drop	27	n/a
$n < 100$ mmHg, oxygen	n/a	10
Dorsum, 5th toe		
Median (IQR)	33.0 (24.0–40.0)	132.0 (86.5–179.5)
95% CI	25.5–38.0	94.5–168.5
Range	4–55	51–307
$n \geq 10$ mmHg drop	26	n/a
$n < 100$ mmHg, oxygen	n/a	11
Plantar, 1st MTP		
Median (IQR)	43.5 (38.0–50.0)	162.0 (113.0–210.5)
95% CI	40.5–50.0	124.0–190.5
Range	26–62	69–246
$n \geq 10$ mmHg drop	12	n/a
$n < 100$ mmHg, oxygen	n/a	7

TCOM levels were not explained by other measures of perfusion: there was a small but statistically significant ($\beta = 0.25$, r-square = 0.308) positive correlation between toe SBP and room-air TCOM at the sensor on the dorsum of the foot proximal to the fifth MTP joint, otherwise there were no significant associations between any of the perfusion measures and the observed TCOM at any sensor, whether on room air, with the leg elevated or breathing 100% oxygen.

Discussion

TCOM is a non-invasive method of estimating tissue oxygenation and the results are used to assist selection of appropriate patients for HBOT. The current normal reference value of 40 mmHg in non-diabetic patients may not be an accurate reference by which to define hypoxia for all locations on the lower limb. Using this value to define hypoxia, about half of the readings between the first and second toes and those of the lateral ankle would have been classified as hypoxic. There was no sensor site for which all of our subjects had values above 40 mmHg, and 24 of the 32 recorded a room-air TCOM below 40 mmHg for at least one sensor site. TCOM values on room air were less than 20 mmHg at four sites in three subjects and, therefore, could have been misclassified as evidence of critical limb ischaemia.[8] However, all four sites responded to oxygen with values above 100 mmHg, suggesting the possibility of

a diffusion barrier contributing to their low room-air values rather than critical ischaemia. Further, the use of multiple electrodes ensures that data from a single electrode is not used in isolation.

Incorrectly classifying patients as having hypoxic tissue may lead to some patients receiving HBOT unnecessarilly. Unfortunately, a more conservative reference value is not a complete solution. A reference value of 34 mmHg (one SD below the mean recorded in our study) would still lead to classification of 9% of our room-air sensor readings as hypoxic. Clinical practice guidelines for TCOM have been developed to assist the clinician;[8] however, our results reaffirm that clinical history and physical examination remain mandatory in selecting appropriate patients for HBOT.

Our study recorded no room-air TCOM values greater than 70 mmHg, although higher values have been reported in earlier studies.[5,6,14,15] A possible explanation for the difference in our results compared to previous studies is that the TCOM400 monitoring system may measure tissue oxygenation differently, as newer sensors have different technical specifications.[16] As discussed with Radiometer, the TCM400 electrode temperature is controlled by two thermistors in the electrode head. These must be in agreement with each other to within less than 0.6^{O}C. If they are not, then a temperature error is flagged and it is not possible to use that electrode. The specifications for the TCM400 state that temperature accuracy is described as better than ± 0.1^{O}C.

Recent lower limb studies using the TCM400 continue to be guided by earlier normal values.[17–20] Also, previous studies have focused on patients with vascular disease or diabetes, with no healthy control arm to define normal values. One previous study used a standardized sensor position, the first inter-metatarsal space, and found mean values of 55 (± 9.92) mmHg in a group of diabetic patients and mean values of 56 (± 8.8) mmHg in non-diabetic patients.[17] These values are again somewhat higher than those we observed at the same sensor site in healthy, non-smoking subjects, mean 39 (± 9.8) mmHg. We are unaware of any studies evaluating measurement validity for different TCOM machines measuring at the same anatomical site.

It has been common practice to place a sensor on the anterior chest wall as a central reference that is reported to provide information regarding the cardio-respiratory status of the patient. In an earlier TCOM study, we found that the chest sensor reading was below that of at least one arm/hand sensor reading in more than three-quarters of our healthy subjects, with one subject's room-air chest sensor value being 13 mmHg, with arm/hand sensor readings ranging between 38 and 63 mmHg.[10] The same has been found in other studies and a recent expert consensus statement confirms that a percentage of patients have an abnormally low chest TCOM reading and the value of this site as a central reference is questionable.[8,17] Given the unreliability of the chest sensor as a reference site, we did not use it in this study and chose to focus all sensors on the lower limb.

Historically as part of routine TCOM assessment, the leg is elevated for five minutes.[3,21,22] A drop of 10 mmHg is considered indicative of significant vascular disease and decreased healing in amputations.[23,24] Two recent vascular studies have examined this using the TCM400. One study found a drop of less than 10 mmHg in diabetic and non-diabetic patients with severe limb ischaemia; however, their starting values were in the low teens and these patients would have been identified as having severe disease without the added leg elevation.[18] The other study used the 10 mmHg drop with elevation to stratify their patients. Ninety-two per cent of patients in the equivocal TCOM range for healing of 20–40 mmHg, with a drop on elevation of > 10 mmHg, failed to heal whereas 80% of patients who had ≤ 10 mmHg drop on elevation healed.[20] However, a drop of 10 mmHg has also been found in healthy subjects.[7] In our study, the response to elevation varied by sensor site with the distal sites more responsive to leg elevation (Table 3). In total, all except one of our subjects had TCOM decrease ≥ 10 mmHg for at least one sensor site when their leg was elevated; this brings into question the use of this manoeuvre in assessing patients during TCOM and, therefore, it is no longer used in our unit.

Expert consensus is that in normal subjects breathing 100% oxygen at normobaric pressure, TCOM on the leg should always increase to a value ≥ 100 mmHg.[8] In this study, on-oxygen TCOMs below 100 mmHg were recorded at every sensor site except the most proximal site (Table 3). This lack of response to normobaric oxygen was most pronounced at the most distal sensor sites. With oxygen administration, TCOM increased by as little as 25 mmHg at the lateral ankle and medial ankle sensor sites, and by as little as 11 mmHg at the site between the first and second toe. There was one subject whose on-oxygen TCOM increased only 6 mmHg at the fifth toe site, and another subject whose on-oxygen TCOM did not increase at all at the plantar site.

While some of these observations might represent random measurement errors, they are too persistent throughout our data. The lateral and medial ankle sites and the dorsum of the foot are not straight-forward measurement sites. Suitable sensor sites were dictated by the availability of flat surface areas where a fixation ring could be applied, but the sites we used are clinically relevant. These sites are dominated by bones and superficial blood vessels. It is feasible that our low values and lack of response to 100% normobaric oxygen could be explained by the influence of de-oxygenated blood in the surrounding vessels.

The normal procedure in Australia is an oxygen challenge using a head hood not a non-rebreather (NRB) mask. We

have just completed another study comparing oxygen flow rates using the hood and the mask (Blake DF, unpublished observations). The hood at 15 L·min^{-1} performed better than 15 L·min^{-1} with a NRB mask. The maximum oxygen concentration in the hood is 98%, reached within approximately six minutes. Of note, the TCOM values with the hood were 50 to 90 mmHg higher than with the NRB mask.

Our study has limitations. The conventional view is that the sole of the foot is not a good measurement site because of the thickened skin and low TCOM values not being representative of the tissue below the keratin layer.[9,25] Neuropathic ulcers are common in this area, and results from our previous study, showing that the palmar surfaces of the hand have high values and low dispersion, led us to include the plantar site in this study.[10] Only three subjects had room-air TCOM values lower than 40 mmHg at this site, although it had a poorer response to oxygen. Including this site in clinical practice and undertaking further studies may be worthwhile.

Our study was also limited in that we used only one TCOM machine. Further studies comparing normal values on different machines and sensors may help elucidate the differences and variability in our values from those quoted in the literature. Finally, our study speaks only to the specificity of lower limb TCOM values in healthy, disease-free non-smokers; we cannot comment on the sensitivity and specificity of TCOM in other patient groups.

Conclusions

Normal lower limb TCOM readings using the TCOM400 oximeter with tc E5250 sensors may be lower than 40 mmHg, the currently accepted definition of hypoxia, but consistent with the wide range, 10 to 40 mmHg, found in the literature. Because of the wide variability in TCOM at the different sensor sites we cannot recommend a single TCOM value as indicative of tissue hypoxia. Using comparative TCOM on the contralateral leg might be better for identifying 'abnormal' tissue and the expected effect of an oxygen challenge; however, many patients may have bilateral disease. A thorough clinical assessment of the patient is essential to establish appropriateness for HBOT, with TCOM results used to help guide this decision and not as an absolute until normal baseline values have been fully validated.

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Systematic review of the effectiveness of hyperbaric oxygenation therapy in the management of chronic diabetic foot ulcers

Liu R, Li L, Yang M, Boden G, Yang G

Objective: To assess the efficacy and safety of hyperbaric oxygenation (HBO) therapy as adjunctive treatment for diabetic foot ulcers with a systematic review and meta-analysis of the literature.
Methods: MEDLINE, EMBASE, and the Cochrane Library were searched to find relevant articles published up to April 20, 2012, without restriction as to language or publication status. All controlled trials that evaluated adjunctive treatment with HBO therapy compared with treatment without HBO for chronic diabetic foot ulcers were selected. A meta-analysis was performed to assess the efficacy and safety of hyperbaric oxygen in managing foot ulcers.
Results: Thirteen trials (a total of 624 patients), including 7 prospective randomized trials, performed between 01 January 1966, and 20 April 2012, were identified as eligible for inclusion in the study. Pooling analysis revealed that, compared with treatment without HBO, adjunctive treatment with HBO resulted in a significantly higher proportion of healed diabetic ulcers (relative risk, 2.33; 95% CI, 1.51–3.60). The analysis also revealed that treatment with HBO was associated with a significant reduction in the risk of major amputations (relative risk, 0.29; 95% CI, 0.19–0.44); however, the rate of minor amputations was not affected ($P = 0.30$). Adverse events associated with HBO treatment were rare and reversible and not more frequent than those occurring without HBO treatment ($P = 0.37$).
Conclusions: This meta-analysis reveals that treatment with HBO improved the rate of healing and reduced the risk of major amputations in patients with diabetic foot ulcers. On the basis of these effects, we believe that quality of life could be improved in selected patients treated with HBO.

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Effect of splenectomy on platelet activation and decompression sickness outcome in a rat model of decompression

Kate Lambrechts, Jean-Michel Pontier, Aleksandra Mazur, Peter Buzzacott, Christelle Goanvec, Qiong Wang, Michaël Theron, Marc Belhomme and François Guerrero

Abstract

(Lambrechts K, Pontier J-M, Mazur A, Buzzacott P, Goanvec C, Wang Q, Theron M, Belhomme M, Guerrero F. Effect of splenectomy on platelet activation and decompression sickness outcome in a rat model of decompression. *Diving and Hyperbaric Medicine*. 2014 September;44(3):154-157.)

Introduction: Splenic platelets have been recognized to have a greater prothrombotic potential than others platelets. We studied whether platelets released by splenic contraction could influence the severity and outcome of decompression sickness (DCS) and bubble-induced platelet activation.

Methods: Sixteen, male Sprague-Dawley rats were randomly assigned to either a control or a splenectomized group. Both groups were compressed to 1,000 kPa (90 metres' sea water) for 45 min while breathing air before staged decompression (5 min at 200 kPa, 5 min at 160 kPa and 10 min at 130 kPa). The onset time of DCS symptoms and of death were recorded during a 60-min observation period post dive. Parameters measured were platelet factor 4 (PF4) for platelet activation, thiobarbituric acid reactive substances (TBARS) for oxidative stress status and Von Willebrand factor (VWf) for endothelial activation.

Results: There were no differences between the groups in DCS outcome or in PF4, TBARS and VWf concentrations.

Conclusion: These results do not support that the spleen and its exchangeable platelet pool is involved in DCS pathogenesis in a rat model, invalidating the hypothesis that increased decompression-induced platelet aggregation could be influenced by splenic contraction and then play a role in DCS outcome.

Key words

Pharmacology, platelets, physiology, treatment, decompression sickness, animal model

Introduction

Decompression from a scuba dive may result in the production of both intra- and extra-vascular bubbles. These cause a complex pathophysiological cascade that includes vascular dysfunction, microcirculatory alterations, inflammatory processes with leucocyte adhesion, pro-coagulant activity and oxidative stress, leading to decompression sickness (DCS).[1–9] Circulating bubbles are thought to affect the clotting system both through activation of the coagulation cascade and the induction of platelet aggregation. In a rat model, the post-dive decrease in platelet count (PC) correlated with severity of DCS, indicating that platelet activation and aggregation are associated with the pathogenesis of DCS.[10,11] Moreover, clopidogrel, an inhibitor of the P2Y12-receptor, reducing ADP-induced platelet activation, has been shown to reduce both DCS severity and platelet count after decompression.[12,13]

Activated endothelial cells are known to inhibit anti-coagulant mechanisms while stimulating pro-coagulant ones, by releasing substances such as tumor necrosis factor (TNF) which will induce tissue factor (TF) production, a pro-coagulant factor. Conversely, activated platelets following diving have been shown to release platelet factor 4 (PF4) in the rat and microparticles (MPs) in divers.[6,8,14] MPs generated by decompression stresses precipitate neutrophil activation, vascular damage and thus endothelial activation.[7]

In physiological conditions, the spleen contains an important number of erythrocytes, leukocytes and platelets.[15,16] About 30% of the total number of platelets are stored in an exchangeable splenic pool, with a mean platelet volume (MPV) 20% higher than the MPV of circulating platelets, owing to increased concentrations of procoagulant surface proteins (P-selectin, GP IIb/IIa).[17] Platelet size correlates positively with platelet reactivity demonstrating that platelets with higher MPV are more active haemostatically.[18]

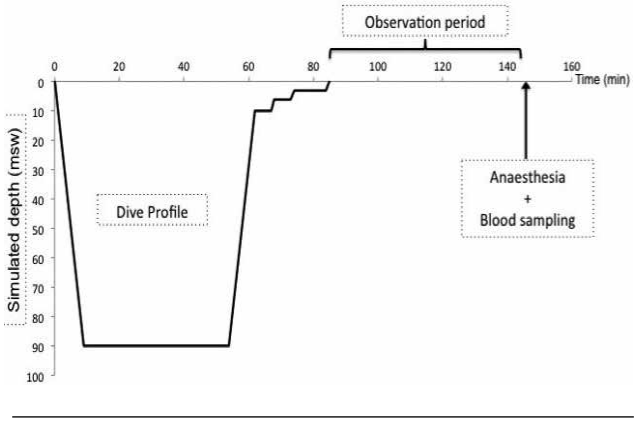
The aim of the present study was to assess whether the spleen might be involved in DCS severity, DCS outcome and platelet activation following an air-breathing compression/decompression protocol known to provoke a predictable proportion of DCS in a rat model.

Materials and methods

ANIMALS

Male Sprague-Dawley rats ($n = 16$; Janvier SAS, Le Genest St Isle, France), aged 11 weeks and weighing 376 ± 27 g (mean ± SD) were used in the study. Animals were housed two per cage, under controlled temperature (21 ± 1°C) and lighting (12 h of light, 0800–2000 h; 12 h of dark, 2000–0800 h) with access to standard rat food and water ad libitum. Rats were studied ≥ 7 days after arrival. Animal experiments were conducted in accordance with the *Guide*

Figure 1
Timeline including hyperbaric exposure profile, observation period and blood sampling



for the Care and Use of Laboratory Animals (US National Institutes of Health; NIH Publication No. 85–23, revised 1996) and with the approval of the local ethics committee for animal experimentation (approval No. 1462.01). This study accords with recognised ethical standards and national/international laws.

SURGICAL PROCEDURE FOR SPLENECTOMY

The rats were randomly assigned to one of two groups: a splenectomy group (SP, n = 8) underwent a splenectomy while a sham group underwent sham surgery (SHAM, n = 8). The rats were anaesthetized by intraperitoneal injection of ketamine (80 mg·kg^{-1}) and xylazin (15 mg·kg^{-1}), shaved and placed on an operating board and secured with tape. Midline laparotomy (3 cm) was performed under full sterile conditions. In the SP group, the spleen was identified and resected after ligature of the splenic vessels. In the SHAM group, the spleen was lifted out of the abdomen and then put directly back into the peritoneal cavity. The peritoneal cavity was irrigated with warm normal saline and the wound closed in two layers.

Individual rats were placed in separate cages postoperatively. Postoperative pain was treated with buprenorphine (Bupracare, Animalcare, Dunnington, UK) 0.3 mg·kg^{-1} injected intraperitoneally twice daily for three days. The rats were allowed to recover for two weeks after surgery before hyperbaric exposure.

DIVE PROFILE AND DECOMPRESSION PROTOCOL

Each rat was positioned in a 130-L steel hyperbaric chamber, always at the same time of day and then compressed with air at a rate of 100 kPa·min^{-1} up to 1,000 kPa absolute pressure (90 metres' sea water equivalent) and remained at this pressure for 45 min. Decompression was performed at a rate of 100 kPa·min^{-1} with three decompression stops: 5 min at 200 kPa, 5 min at 160 kPa and 10 min at 130 kPa. Total dive time was 83 min. This dive profile has previously been described and is known to reliably induce DCS in approximately 70% of rats.[6,19] For one hour after the exposure, the rats were passively observed for the appearance of signs of DCS such as unusual fatigue, ambulatory deficit, abnormal breathing, convulsions or death. The rats were classified into three categories: dead, alive with obvious symptoms within 60 minutes post dive or no symptoms of DCS (Figure 1).

BLOOD SAMPLING AND ELISA

Following the observation period, surviving rats were anaesthetized with pentobarbital (50 mg·kg^{-1}) by intraperitoneal injection. Intracardiac blood collection was performed immediately following anaesthesia or death into a BD Vacutainer® citrate tube (0.11 M) and into 2 mL Eppendorf® tubes with 30 µl 7.5% EDTA as an anticoagulant. Afterwards, surviving rats were euthanized whilst still anaesthetized by a lethal intraperitoneal injection of pentobarbital.

Blood was centrifuged at 1000 g and 4^{O}C for 10 min. Collected plasma was aliquoted and stored at -80^{O}C until assayed. The concentrations of markers of platelet activation: platelet factor 4 (PF4), endothelial activation (Von Willebrand factor, VWf), and oxidative stress status (thiobarbituric acid reactive substances, TBARS) were determined using commercially available ELISA kits for PF4 (Uscn Life Science Inc., Houston, USA), for VWf (Cusabio Biotech., Wuhan, China) and for TBARS (Cayman Chemical, Michigan, USA). Assay procedures were performed according to provider's instructions.

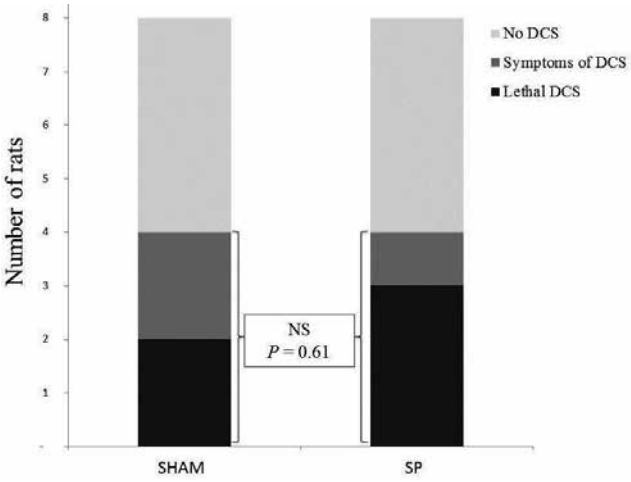
STATISTICAL ANALYSIS

Data are expressed as mean ± SD; n indicates the number of subjects in each group. For statistical analysis of blood marker concentrations post dive we used the Statistica 10 programme (Tulsa, Oklahoma, USA). Student's t-tests were used to compare groups of paired data (PF4, vWF and TBARS) after Kolmogorov-Smirnov tests for normality. Where data were not normally distributed, Wilcoxon signed rank sum tests were performed. Finally, the influence of splenectomy upon DCS was tested for significance with a Fisher's exact chi-square test. Significance in all cases was accepted at $P \leq 0.05$.

Results

The mean weights for the two groups of animals did not differ significantly (P = 0.85). There was no difference between the groups in the incidence of DCS symptoms (SHAM: n = 2, SP: n = 1). Five of the rats died within the 60-min observation period post dive. For DCS prevalence, including symptomatic and dead rats, there was no

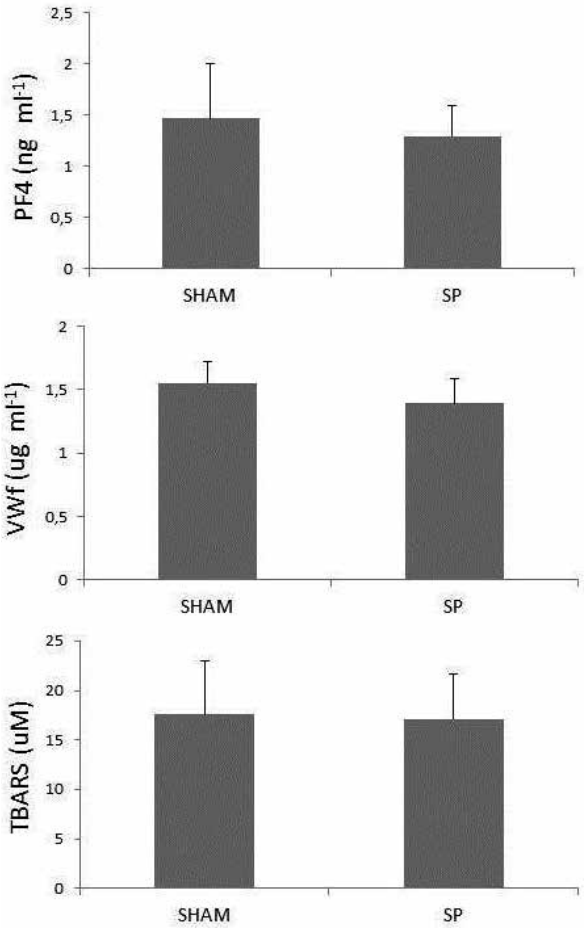
Figure 2
Number of lethal decompression sickness (DCS) (within 60 min of decompression), symptoms of DCS (paralysis or dyspnoea) or no symptoms following decompression in sham or splenectomised rats, n/s = not significant



significant difference in DCS outcome between the group ($n = 4$, $P = 0.61$). There were two deaths from DCS in the SHAM group and three in the SP ground (chi-square test $P = 0.27$; Figure 2).

Comparing plasma markers, no significant differences between the two groups were detected in any of the tests (Figure 3). Following decompression, the PF4 plasma concentration was 1.47 ± 0.54 ng·ml^{-1} in the SHAM group vs. 1.29 ± 0.31 ng·ml^{-1} in the SP group; VWf was 1.55 ± 0.17 µg·ml^{-1} in the SHAM group vs. 1.39 ± 0.19 µg·ml^{-1} in the SP group. The post-dive TBARS concentration was 7.61 ± 5.38 uM in the SHAM group compared to 17.12 ± 4.89 uM in the SP group.

Figure 3
PF4 – platelet factor (ng·ml^{-1}), VWf – Von Willebrand factor (µg·ml^{-1}) and TBARS – thiobarbituic acid reactive substances (uM) concentrations after decompression in sham rats (SHAM) and splenectomised rats (SP)



Discussion

The aim of the present study was to investigate whether the spleen and its exchangeable platelets could influence DCS outcome, using splenectomized and intact rats. We found no differences in either DCS outcome or platelet activity between the control and splenectomized groups. This suggests that the spleen does not play an important role in the pathogenesis of DCS in this model following a DCS-provoking, deep air-dive profile.

During exercise, or in aquatic mammals during diving, the spleen serves as a dynamic red cell blood reservoir.[20] Splenic contraction increasing haematocrit and haemoglobin content has been reported in diving mammals, such as the Weddell seal.[21] In humans, splenic contraction has been shown to prolong apnea dives.[20] A rapid, sustained increase of MPV in systemic venous blood, but without any changes in total platelet count, has been reported after repetitive breath-hold dives.[22] These results suggest that splenic contraction and the release of larger platelets are part of the diving response during breath-hold diving.

The results of a human breath-hold study suggest that there must be splenic capture of smaller platelets in addition to ejection of the larger ones since platelet counts were unchanged.[22] Our results demonstrate that PF4 concentrations correlate with DCS outcome, and do not change if the DCS outcome remains unchanged. Besides this, the equivalent level of oxidative stress (TBARS) and endothelial activation (VWf) between both groups is consistent with the equivalent concentration of PF4. As these three factors interact, a difference in platelet activation would have influenced the status of free radicals and endothelial cells. In the case of increased decompression stress and platelet activation we should have observed higher concentration of TBARS owing to NAD(P)H oxidase–dependent O_2 release and significant levels of VWf due to endothelial activation by stimulation of platelet MPs.[7,23] The similar levels of PF4, TBARS and VWf between groups are consistent with an equal platelet concentration and an unchanged DCS outcome.

Conclusion

This study suggests that splenic contraction, normally considered to be a physiological response to breath-hold diving, is not involved in platelet activation or DCS incidence after a DCS-provoking air dive in a rat model. However, we have not established whether this lack of effect is the result of a non-influence of large platelets released by the spleen because of decompression stress or if it is, at least partly, a result of the nonexistence of spleen contraction during diving. Further research should aim to demonstrate whether or not scuba diving induces splenic contraction in man.

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Editorial note: Assistant Professor Guerrero states that all nine authors meet the guidelines for authorship of the International Committee of Medical Journal Editors.

Short communication

The effect of air scuba dives up to a depth of 30 metres on serum cortisol in male divers

Rasool Zarezadeh and Mohammad Ali Azarbayjani

Abstract

(Zarezadeh R, Azarbayjani MA. The effect of air scuba dives up to a depth of 30 metres on serum cortisol in male divers. *Diving and Hyperbaric Medicine*. 2014 September;44(3):158-160.)

Introduction: Environmental pressure changes with depth may lead to changes in various hormone levels in the body. Of interest are the so-called stress hormones, such as cortisol. Other factors altering cortisol levels are anxiety, exercise and cold. We investigated serum cortisol changes after air scuba dives in 24–27^{O}C open water up to a depth of 30 metres.

Method: Ten, experienced, male divers participated in the study. Four dives, to depths of 1, 10, 20 and 30 metres' sea water (msw) for 20 minutes bottom time, at rest, were conducted at about 1000 h on four consecutive days in the Persian Gulf. Before diving and soon after surfacing, approximately 5 ml blood was drawn from a right antecubital vein for serum cortisol assay, using a radioactive immunoassay technique. Repeated measures was used to analyse cortisol changes with depth.

Results: There were significant differences in the pre-dive cortisol levels ($df = 1$, $F = 5.978$, $P < 0.037$) and post-dive levels ($df = 1$, $F = 34.567$, $P < 0.001$). Cortisol levels increased with immersion irrespective of depth compared to pre-dive levels, whilst they were further significantly raised after dives to 10 m (mean 312.6 nmol·L^{-1}), 20 m (mean 299.1 nmol·L^{-1}) and 30 m (mean 406.7 nmol·L^{-1}) depth compared to levels after the 1 m dive (mean 189 nmol.L^{-1}).

Conclusion: The observed changes in serum cortisol were considered to be the result of the physiological effects of immersion combined with increased environmental pressure, rather than resulting from anxiety, heavy exercise or cold stress.

Key words

Scuba diving, physiology, endocrinology, diving research

Introduction

Many individuals, whether for recreational or professional reasons, are involved with the underwater world and its challenges. Environmental pressure changes with depth, and such changes may lead to changes in various hormone and enzyme levels in the body. Of particular interest are the so-called stress hormones, such as cortisol, regarded as the most important glucocorticoid in humans.[1] Cortisol levels demonstrate diurnal variation, highest levels occurring at about 0600 to 0800 h and lowest levels at about midnight. Normal values for a blood sample taken at 0800 h vary between 165.5 and 634.5 nmol·L^{-1}.[2] Reports suggest that plasma or salivary cortisol levels fall during dry-chamber pressure exposures but rise after open-water dives.[3,4] Given the physiological importance of cortisol, we measured serum cortisol levels before and after open-water air dives to depths up to 30 metres' sea water (msw).

Methods

SUBJECTS

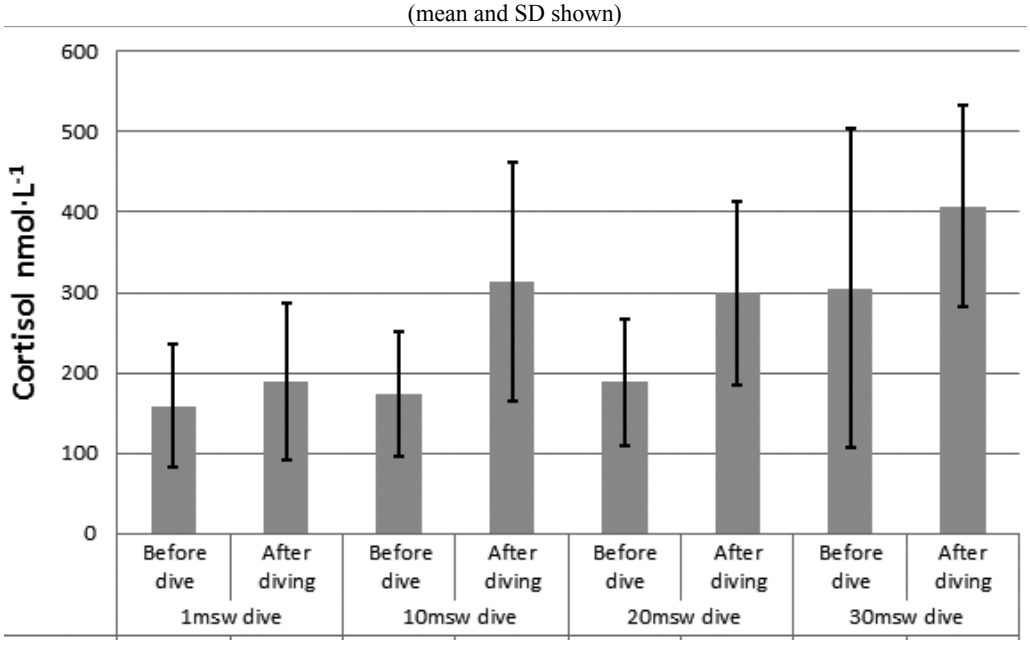
The study was approved by the Department of Physical Education, Science and Research Branch, Islamic Azad University, Fars, Iran. Twelve, experienced, male divers certified to dive up to 30 msw depth were recruited for the study. Two subjects were subsequently excluded because of illness during the project. The divers gave informed consent and completed a medical questionnaire. Age, height and weight were measured, and body mass index (BMI) calculated.

PROCEDURE

Four dives were conducted on four consecutive days off Qeshm Island in the Persian Gulf, with a water temperature of 24 +/- 2^{O}C and on sunny days with an air temperature of 27 +/-2^{O}C. The divers wore 3-mm-thick wetsuits. All dives were performed at about 1000 h. The four dives were to depths of 1, 10, 20 and 30 msw for 20 minutes bottom time. The divers remained at rest, kneeling on the sea floor throughout the time at depth. The dive order was not randomised.

Before diving, approximately 5 ml of blood was drawn from a right antecubital fossa vein into a plain sample tube. Upon surfacing at the end of the dive, a second blood sample was drawn. All pre- and post-dive blood samples were taken to a modern laboratory in Shiraz and centrifuged at 3,200 rpm and the serum separated and stored at -25 ^{O}C for measurement the following day. Cortisol was measured using Cortisol Radioimmunoassay Kit IM 1841, (Czech Beckman Coulter Company).

Figure 1
Changes in serum cortisol (nmol·L^{-1}) before and after 20-minute dives to depths of 1, 10, 20 and 30 metres' sea water (msw) (mean and SD shown)



STATISTICAL METHODS

A repeated measures ANOVA was used to analyse cortisol changes with depth. If sphericity was not assumed, a Greenhouse-Geisser correction was applied. If a significant difference was observed, paired Student's *t*-tests were used to analyse specific differences. All data are reported as the mean and standard deviation (SD). Significance was assumed at the $P \leq 0.025$ level after applying a Bonferroni correction for multiple comparisons. The software package SPSS 22 was used.

Results

The 10 divers studied had a mean age of 28 years (range 19–39), mean height 178 cm (range 173–184), mean weight 84 kg (range 72–96) and BMI 27 kg·m^{-2} (range 23–30). Pre- and post-dive cortisol levels are shown in Figure 1. The differences in the pre-dive cortisol levels (1 msw, mean 158.36 (76.6) nmol·L^{-1}; 10 msw, mean 173.8 (78.5) nmol·L^{-1}; 20 msw, mean 188.2 (78.4) nmol·L^{-1}, and at 30 msw, mean 305.4 (199.1) nmol·L^{-1}) did not reach statistical significance after a Bonferroni correction (df = 1, F = 5.978, $P < 0.037$). There were significant differences between the pre-dive cortisol levels (df = 1, F = 5.978, $P < 0.037$) and post-dive levels (df :1 , F = 34.6, $P < 0.001$) at each depth. Cortisol levels increased with immersion, irrespective of depth, compared to pre-dive levels, whilst they were raised further significantly after dives to 10 msw (mean 312.6 (SD 148.8) nmol·L^{-1}; 20 msw (mean 299.1 (114.5) nmol·L^{-1} and 30 msw (mean 406.7 (125.4) nmol·L^{-1} compared to 1 msw (mean 189 (97.8) nmol·L^{-1}).

Discussion

In a study of the effects of increased pressure, variations in inspired gases and the use of a mask during dry chamber dives on salivary cortisol in professional divers, levels decreased from a mean of 16.0 mmol·L^{-1} pre-dive to 10.3 mmol·L^{-1} post-dive ($P < 0.01$).[3] Cortisol values did not relate to the anthropometric and physical fitness characteristics of the divers or to increased pressure, variation in inspired gases or the use of a mask. The individual variation in cortisol values was large. In another dry chamber study, eight professional divers were exposed to air or 100% oxygen at 253 kPa for 60 min. As in the first study, cortisol levels decreased significantly ($P = 0.001$) during the dry dives.[5] During a hyperbaric saturation dive to 4.1 MPa in six subjects, salivary cortisol concentration did not change throughout the dive.[6]

In a 1972 study, plasma cortisol levels in dry-suited divers before and after 1 or 30 msw dives in cold (12^{O}C) water were significantly elevated compared to control values.[7] The rise was twice as great with the 30 msw dives (+42%) compared to 1 msw (+23%). Levels before 30-metre dives were 25% higher than before 1-metre dives, and all values were approximately twice those seen in the present study. These changes were attributed to anxiety over deep open-water diving in a 'stressful' diving situation.

In other studies, salivary cortisol levels in trainee scuba divers showed significant increases before a swimming-pool training session and before an open-water dive compared to control values.[4] During prolonged whole-body immersion in cold water, cortisol demonstrated a marked diurnal variation,

with large increases occurring after 2200 h.[8] Increased levels of cortisol have also been shown in divers exposed to an underwater navigation stress.[9]

In the present study, serum cortisol increased progressively with dives of increasing depth over the depth range of 1 msw to 30 msw. Psychological stress and anxiety are known to increase serum cortisol. There is evidence that individuals who are characterised by elevated levels of trait anxiety are more likely to have greater state anxiety responses when exposed to a stressor, and hence, this sub-group of the diving population is at an increased level of risk.[10] Divers with an elevated level of anxiety and poor coping are at higher risk of developing panic reactions than those possessing more adequate stress-coping mechanisms.[11] However, we did not assess our divers for their trait anxiety levels. Since the divers who participated were experienced, professional divers, it does not seem likely that anxiety was the main factor behind these increases.

Exercise is a factor changing cortisol. Variations in free cortisol concentrations associated with a treadmill test to exhaustion and high-level competition have been studied in top-level swimmers.[12] Salivary cortisol was measured 30 minutes before and 15 minutes after competition and was compared with concentrations obtained at the same times of the day before and after the treadmill tests and during a rest day. Cortisol levels were significantly higher before and after competition than before and after a treadmill test. In endurance athletes, cortisol is increased significantly in both serum and saliva in response to high-intensity exercise.[13] Since the divers in the present study were at rest during the dives, it is unlikely that physical activity was the main factor behind the increases that we observed.

Cold is a well-recognised factor changing cortisol. Prolonged whole-body immersion in cold water results in elevated plasma cortisol levels.[8] The temperature of the water in the present study was warm, but slightly less (24 ± 2^{O}C) at 30 msw depth than at the surface (27 ± 2^{O}C). As all divers were wearing 3-mm-thick wetsuits and none showed any signs of coldness, it does not seem that cold was the main factor behind these changes. However, body core temperature was not measured.

We conclude that the changes in serum cortisol observed were predominantly the result of the physiological effects of immersion combined with increased environmental pressure.

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Case report

Hyperbaric oxygen treatment for unilateral testicular torsion in a child

Bilal Firat Alp, Gamze Cebi, Adem Özdemir, Hasan Cem Irkilata and Günalp Uzun

Abstract

(Alp BF, Cebi G, Özdemir A, Irkilata HC, Uzun G. Hyperbaric oxygen therapy for unilateral testicular torsion in a child. *Diving and Hyperbaric Medicine*. 2014 September;44(3):161-162.)

Torsion of the testis is a urological emergency most commonly occurring in adolescent boys. Hyperbaric oxygen treatment (HBOT) has been shown to alleviate reperfusion injury in experimental ischaemia of the testis. We report a 13-year-old boy who had prolonged right testicular ischaemia. Despite surgery, the colour of the testis remained poor. He underwent a post-operative course of 10 HBOT over 8 days, with restoration of blood flow on colour Doppler and reduction of oedema. At four-month followup, the testis appeared normal on ultrasonography. To the best of our knowledge, this is the first published case of torsion of the testis treated with HBOT.

Key words

Genitourinary tract, children, hyperbaric oxygenation therapy, reperfusion injury, case report

Introduction

Torsion of the testis and spermatic cord is a urological emergency most commonly occurring in adolescent boys.[1] In 5–6% of patients torsion is secondary to trauma.[2] Torsion interrupts the blood supply leading to ischaemic damage to the testis. In addition, re-establishing blood flow via surgical intervention (de-torsion) may result in a reperfusion injury. Oxidative stress and inflammation associated with reperfusion contributes to germ cell injury.[3] Therefore, additional treatments other than re-establishing the blood supply are needed to prevent the development of germ cell injury in these patients.[4]

Hyperbaric oxygen treatment (HBOT) has been shown to alleviate reperfusion injury in a number of organs including the testis.[3,5] To the best of our knowledge, the use of HBOT has not been reported for testicular torsion. Herein, we report a patient who had prolonged testicular ischaemia from torsion, only partially relieved by surgery and successfully treated with the addition of HBOT.

Case Report

A 13-year-old boy presented to another emergency department complaining of severe scrotal pain and swelling, which was first noticed at 0700 h. Treatment with an antibiotic and an anti-inflammatory drug was started on the basis of the diagnosis of orchitis. Later the same day (1630 h), he was admitted to our hospital's emergency department because of worsening pain. Urological assessment revealed a history of an accident while riding his bicycle the day before. On examination, scrotal oedema and mild hyperaemia were observed. His right testicle was very painful to palpation and there was no pain relief with testicular elevation (negative Phren's sign), indicating testicular torsion. The left testis was normal in size and not painful to palpation. Scrotal colour Doppler ultrasonography (CDU) showed a right testis measuring 31 x 21 x 17 mm and left 31.2 x 18.8 x 17.7 mm. No right epididymal or testicular arterial blood flow could be detected on CDU.

Emergency surgery was performed at 1830 h (11–12 h after the onset of symptoms). At surgery, a 360^{O} torsion of the right testicle was found, and the testis was completely purple in colour. Despite testicular de-torsion and warm serum application for 45 minutes, the colour of the testis did not improve. However, some bleeding was observed in a small incision made in the right testis. The surgery was completed with bilateral testicular fixation. Considering the prolonged duration of ischaemia and complete arterial obstruction, the boy was referred for HBOT following informed parental consent.

The first HBOT commenced about 5 h post-operatively (about 18 h from the onset of symptoms). HBOT was carried out at 243 kPa for 90 minutes, interspersed with two 5-minute air breaks. Two further HBOT were given 15 and 26 hours post-operatively followed by daily sessions to a total of 10 sessions, without incident.

CDU on the fifth post-operative day showed normal right epididymal and testicular arterial blood flow. The right testis was significantly increased in size compared to the left. The boy was discharged without any complaints after eight days. At a four-month follow up, Doppler ultrasonography showed the right testis size to be 21.6 x 17.7 x 15.9 mm and the left 31.4 x 18.7 x 17.8 mm.

Discussion

Early diagnosis and surgical treatment is important to achieve the best outcome in the treatment of testicular torsion. Testicular necrosis develops if testicular torsion is not corrected within 4 to 6 hours in case of complete arterial occlusion.[6,7] Testicular atrophy together with subfertility develops in two-thirds of patients in the long term.[6] The two most important factors determining outcome after testicular torsion are the duration and the degree of testicular torsion. The success rate is 100% if the patient is treated within 6 hours after the onset of symptoms, 70% if treated between 6 and 12 hours and only 20% if treated between 12 and 24 hours.[8] Beyond 10 h of torsion, most patients will have significant atrophy, unless a spontaneous reduction had occurred or the torsion was limited to 180^{O}–360^{O}. With a torsion of > 360^{O} that lasts more than 24 hours, all patients will have complete or severe atrophy.[9] In our case, the patient had a 360^{O} torsion and this was not corrected for approximately 12 hours after the onset of symptoms. Therefore, he has a high risk for testicular necrosis or atrophy.

HBO is a safe treatment modality, widely used for various indications. HBOT given during ischaemia or reperfusion reduces germ cell injury in an animal model of testicular torsion.[5] Its beneficial effects are related to reduced neutrophil recruitment, inhibition of inflammatory cytokine secretion, antioxidant enzyme activation and blockade of lipid peroxidation in rats.[3] Testis weight and daily sperm production at one month improved only in the HBO-treated rats in this study.[3] To the best of our knowledge, the use of HBOT for testicular torsion in a child has not been reported previously. Considering the prolonged duration of ischaemia and complete arterial obstruction in our patient, we wished to use HBOT to prevent reperfusion injury. Long-term follow up would be needed to know whether testicular atrophy and hypofertility were prevented in this case.

In conclusion, we successfully used HBOT in a boy with prolonged testicular torsion and ischaemia. However, we cannot endorse routine use of HBOT for such patients until results from clinical trials are forthcoming.

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The world as it is

Survey of referral patterns and attitudes toward hyperbaric oxygen treatment among Danish oncologists, ear, nose and throat surgeons and oral and maxillofacial surgeons

Lone Forner, Anne Lee and Erik Christian Jansen

Abstract

(Forner L, Lee A, Jansen EC. Survey of referral patterns and attitudes toward hyperbaric oxygen treatment among Danish oncologists, ear, nose and throat surgeons and oral and maxillofacial surgeons. *Diving and Hyperbaric Medicine*. 2014 September;44(3):163-166.)

In head and neck cancer patients with late radiation injury, hyperbaric oxygen (HBO) is used for therapeutic or prophylactic reasons against soft-tissue and osteoradionecrosis (ORN). Twenty-nine departments of oncology, ENT, oral and maxillofacial (OMF) surgery were surveyed using the Enalyzer tool <www.enalyzer.com>, of whom 21 responded. Data were incomplete in four returns. Within the previous year, 14 departments had referred at least one patient for hyperbaric oxygen therapy (HBOT). There appears to be a generally positive attitude in Danish OMF, ENT and oncology departments towards referral of patients with ORN for HBOT. However, there is an increasing desire for better evidence for its role in head and neck cancer in the prevention and treatment of soft-tissue injury and osteonecrosis following radiotherapy.

Key words

Osteoradionecrosis, bone necrosis, hyperbaric oxygen therapy, radiotherapy, questionnaire, survey

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Introduction

In head and neck cancer patients with late radiation injury, hyperbaric oxygen treatment (HBOT) is used for therapeutic or prophylactic reasons against soft-tissue injury and osteoradionecrosis (ORN). There is some evidence for a clinical effect of HBO on ORN; however, further research within this field is desirable in order to strengthen the evidence as few studies – randomized trials in particular – have been conducted for this purpose.[1] The existing level of evidence for HBOT may give rise to differences in referral patterns because attitudes rather than facts may be decisive for the choice of treatment. Thus, the aim of this survey was to evaluate referral patterns and attitudes toward HBOT in Denmark.

In Denmark, HBOT is organized by the public health care system. There is a seven-seat, multiplace chamber in Copenhagen University Hospital, while Aarhus University Hospital and Odense University Hospital have one monoplace chamber each. The standard treatment is 30 hyperbaric exposures at 243 kPa for 90 minutes with 5 minutes of compression and 5 minutes of decompression. At the time of this survey, the chamber in Odense had not been installed. All three chambers are available to the general public. At referral, the general practitioner or a hospital department refer the patient to the HBO unit. Funding is provided without need for individual application. Generally, the indications on the UHMS website are considered 'approved indications'.

Patients and methods

In January 2010, the official Danish online healthcare system <www.sundhed.dk> was searched for hospitals with departments of oncology and ENT and oral and maxillofacial (OMF) surgery. Twenty-nine departments were invited to participate in the survey. The survey was conducted using the Enalyzer tool <www.enalyzer.com>.

Results

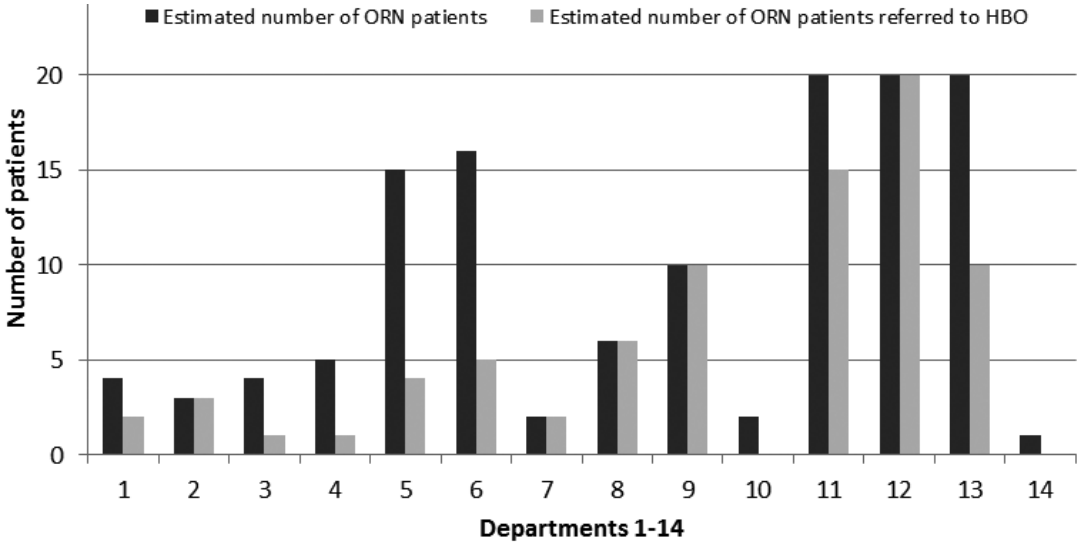
RESPONSE RATES

Twenty-two of the 29 departments responded; nine out of 10 oncologists, seven out of twelve ENT surgeons and six out of seven OMF surgeons. One of the 22 responding did not wish to participate. Of the 21 contributions, four of the answers were incomplete. Fourteen respondents/departments reported to have referred for HBOT at least one patient with ORN within the latest year. Copenhagen University Hospital is a national centre for treatment of ORN and has consequently a large number of patients compared to other centres (Figure 1). Also, the population around Copenhagen is the largest, which explains the higher number of patients at this hospital.

REFERRAL PATTERNS

Of the 14 respondents who had referred at least one case of ORN, 13 responded that they routinely referred patients for

Figure 1



HBOT; no-one reported having stopped referring patients for HBOT having done so in the past. Three respondents reported use of dietary counselling to ORN patients. One of these commented that well-nourished patients appeared to have better ORN recovery. One reported "*no other treatment*". Four reported "*other treatment*". This was further specified as "*antibiotics*" by two respondents, purification and antibiotics by one respondent and an oncologist reported onward "*referral to Department of Oral and Maxillofacial Surgery*".

Eight respondents referred patients to Copenhagen University Hospital; for six, this was the nearest chamber. The other five referred patients to the monoplace chamber in Aarhus University Hospital, whilst two referred to both centres, depending on the patient's wishes, although they were from the region nearest to the monoplace chamber in Aarhus. Reasons for choice of referral centre were mainly 'geographical', whilst one gave "*facilities for the patients*" as the reason, and in another case, the reconstruction surgery to follow HBOT was planned to be in Aarhus. Three did not give any reason.

ATTITUDES

Of the 14 responders, 10 (three oncologists, two ENT surgeons and five OMF surgeons) answered that they believed that "*the treatment is helpful to the patients*". Five believed that the "*effect was questionable*". Nine stated that "*the treatment was generally well accepted among the patients*". Of the responses indicated in our survey to question 8 (see Table 1), six indicated that "*there are patients who do not want this treatment*", while only one stated that "*patients generally do not want this treatment*". This last mentioned respondent (oncologist) was the same one who did not refer patients to HBOT. Additional comments to the question were "*it is difficult to estimate the effect of HBO, since the patients generally also undergo surgery*" (oncologist) and "*the patients feel welcomed in the hyperbaric facility and treated by kind staff*" (OMF surgeon).

Seven respondents reported that one or two patients annually would refuse HBOT for a variety of reasons including travel distances, various physical and psychosocial factors, "*anxiety of the unknown*", "*flash back to radiation treatment*" and a "*lack of guarantee for clinical effect*".

Several respondents reported distance and lack of evidence as barriers for using HBOT. Eight reported "*lack of evidence*". One respondent (oncologist) said that some cases of enhanced tumour growth of recurrent cancer, probably in a hypoxic area, had been observed in their department.

There was a tendency for oncology respondents to be more sceptical than surgeons towards HBOT. Eleven respondents indicated that improved evidence for the beneficial effect of HBOT would influence them to use it more often. There was consistency between what the respondents viewed as barriers to treatment and what they considered as the necessary changes that would result in greater use of this treatment.

Discussion

This survey shows that most referring physicians in Denmark generally consider HBOT helpful to patients with ORN, although they are also critical about the existing level of evidence, seeing this as a major barrier for HBOT. In this respect HBO treatment may no longer be offered to ORN patients if more convincing evidence is not provided. For this purpose, Danish and Dutch research groups have initiated RCTs with participation from other European countries (information available at <www.clinicaltrials.gov>).

A study by Marx showed a therapeutic effect of HBOT on osteo-radionecrosis. Among the 268 included patients, 100% resolved within the three stages of the Marx protocol, 38 in stage I and 48 in stage 2 while 182 progressed to stage 3 before disease resolution.[2] A French randomised study reported a statistically significant better outcome in the placebo arm (32%) than in the HBO arm (19%).[3] However, this study has been widely criticized for its design including issues such as treatment compliance, statistical power, lack of well-defined diagnostic criteria, lack of stratification according to disease severity and potentially leading to bias, since more severely affected cases could have been assigned to one arm or the other.[4] The quality of this study highlights the need for well-designed randomised trials within this field.

In general, departments refer to the nearest regional hyperbaric centre for economic reasons and because of clinical collaborative agreements. However, one department in Northern Jutland, which was nearest to the monoplace chamber, responded that they referred their patients to the multiplace chamber in Copenhagen because of the facilities for the patients. Respondents from the two other departments in Northern Jutland let the patients choose the hyperbaric facility they preferred, despite the geographical relation to Aarhus. This indicates that a culture may develop in one institution which potentially affects clinical decisions. Also, it shows that surroundings and facilities are of great importance to the patients, which seems logical considering the amount of time they spend in the department during their treatment course.

HBOT is well accepted among patients as only one respondent stated that the patients generally do not want this treatment. This respondent was one of the four questioning the effectiveness of HBOT. This indicates that the attitude of the physician may affect the attitude of patients towards the treatment. Apart from this, the barriers for the patients appear mostly to be either geographical, health-related or psychological.

Enhanced tumour growth by HBOT in patients with recurrent cancer is a commonly raised concern. The known effects of HBOT on angiogenesis and cellular regeneration have led to suspicion of a similar stimulation of tumour growth. A Cochrane review has concluded that there is some evidence that HBOT improves local tumour control and mortality as well as local tumour recurrence for head and neck cancer. Other reviews support this conclusion stating that the published literature within this field provides little basis for the opinion that hyperbaric oxygen enhances malignant growth or metastases.[5–7]

In conclusion, further randomized trials are required in order to better determine the role of HBOT for the prevention and treatment of soft-tissue injury and osteo-radionecrosis in head and neck oncology. The importance of a strong multidisciplinary approach between OMF/ENT surgery, oncology and hyperbaric medicine cannot be emphasised enough as this is vital for the success of the treatment. This would be even more successful if the focus was increased on developing better staging systems and international treatment guidelines.

Table 1

Questionnaire sent to the participants; HBOT – hyperbaric oxygen treatment; ORN – osteoradionecrosis

1. Are you an:
   a) Oncologist
   b) Oto-rhino-laryngology surgeon
   c) Oral and maxillofacial surgeon
2. How many ORN patients are diagnosed/treated in your department annually?
3. How many of these were (would you assess) referred to HBOT?
4. What treatment(s), apart from surgery, does your department offer for ORN?
   a) Referral for HBOT
   b) Used to refer for HBOT, but have now ceased
   c) Dietary counselling
   d) No other treatment
   e) Other treatments than above
5. In case of 'other treatment', what treatment(s) do your department offer?
6. If you refer for HBOT, which hyperbaric facilities do you refer to?
   a) Copenhagen
   b) Aarhus
7. What is the reason for referring patients to the chosen facility?
8. What is your departmental experience with HBO-treated patients? (several answers may be chosen)
   a) We think that it is helpful to the patients
   b) We question the effect
   c) The treatment is generally well accepted among the patients
   d) There are patients who do not want this treatment
   e) The patients generally do not want this treatment
   f) Other experiences
9. If other experiences, please describe these:
10. How many patients decline HBOT each year?
11. What do you think is the reason that patients decline HBOT?
12. What barriers are there for HBOT of ORN patients?
   a) Lack of evidence
   b) Distance to nearest HBO unit
   c) Other
13. If other, please specify:
14. What factors could increase the use of HBOT?
   a) Better evidence for the treatment
   b) Distance to nearest hyperbaric unit
   c) Other
15. If other, please specify.

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Influence of repeated daily diving on decompression stress

Zanchi J, Ljubkovic M, Denoble PJ, Dujic Z, Ranapurwala SI and Pollock NW

Abstract

Acclimatization (an adaptive change in response to repeated environmental exposure) to diving could reduce decompression stress. A decrease in post-dive circulating venous gas emboli (VGE or bubbles) would represent positive acclimatization. The purpose of this study was to determine whether four days of daily diving alter post-dive bubble grades. Sixteen male divers performed identical no-decompression air dives on four consecutive days to 18 meters of sea water for 47-minute bottom times. VGE monitoring was performed with transthoracic echocardiography every 20 minutes for 120 minutes post dive. Completion of identical daily dives resulted in progressively decreasing odds (or logit risk) of having relatively higher grade bubbles on consecutive days. The odds on Day 4 were half that of Day 1 (OR 0.50, 95% CI: 0.34, 0.73). The odds ratio for a > III bubble grade on Day 4 was 0.37 (95% CI: 0.20, 0.70) when compared to Day 1. The current study indicates that repetitive daily diving may reduce bubble formation, representing a positive (protective) acclimatization to diving. Further work is required to evaluate the impact of additional days of diving and multiple dive days and to determine if the effect is sufficient to alter the absolute risk of decompression sickness.

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The silent witness: using dive computer records in diving fatality investigations

Martin DJ Sayer and Elaine Azzopardi

Abstract

(Sayer MDJ, Azzopardi E. The silent witness: using dive computer records in diving fatality investigations. *Diving and Hyperbaric Medicine*. 2014 September;44(3):167-169.)
Downloaded data from diving computers can offer invaluable insights into diving incidents resulting in fatalities. Such data form an essential part of subsequent investigations or in legal actions related to the diving incident. It is often tempting to accept the information being displayed from a computer download without question. However, there is a large variability between the makes and models of dive computer in how the data are recorded, stored and re-displayed and caution must be employed in the interpretation of the evidence. In reporting on downloaded data, investigators should be fully aware of the limitations in the data retrieved. They should also know exactly how to interpret parameters such as: the accuracy of the dive profile; the effects of different mode settings; the precision of displayed water temperatures; the potential for misrepresenting breathing rates where there are data from integrated monitoring systems, and be able to challenge some forms of displayed information either through re-modelling based on the pressure/time profiles or by testing the computers in standardised conditions.

Key words
Diving deaths, scuba accidents, investigations, computers – diving

Introduction

In their review of recent changes in diving fatality investigations, Edmonds and Caruso highlight the types of possible information that can assist inquiries through downloading dive computers.[1] We agree fully with the authors that data retrieved from dive computers can generate an indicative recording of the incident dive and, in most cases, the preceding dive history. We also agree that these records can be of extreme value in incidents where the diver dies alone and/or where there are conflicting reports of the incident dive. However, we would urge caution in how these data are accepted and interpreted. In particular, we should like to highlight areas where the accuracy of the information produced on download requires additional analysis. Whereas some of the points we raise could be considered pedantic, they are all relevant to interrogations that have taken place during Fatal Accident Inquiries (Scotland), Coroner's Inquests (England/Wales) or related legal cases.

Handling the victim's computer

On receipt of the dive computer, it is standard practice to make a full photographic record of the unit. Where there has been a delay, new batteries may be needed in order to begin any investigation. This may affect the quality of the downloaded information and needs to be referred to in any report produced; it may also result in a total loss of data. If the computer displays time, then a comparison is made between what is displayed and the actual time. Many computers will store some dive information that can be accessed without downloading the unit. However, it is mainly with downloaded data that difficulties with interpretation could occur. In general, most downloads provide a summary logbook of the diving history and more detailed information on the actual dives performed. The dive computer's logbook information can be relied on to give a good overview of the dive history preceding the incident. Invariably, most computers will store at least the basic parameters of the dives (date, maximum depth, duration, start and finish times); some store much more detailed information, including full dive profiles, although the volume of stored information does vary between models.[2] The initial checks of the computer's time clock against actual time will give baseline information on any differences that could be expected on download. In a small number of instances, the times on the dive computer were altered to the time of the computer onto which it had been downloaded; variations caused by daylight saving changes should also be checked for. It is obviously important that each computer is assigned its own logged file, but overwriting other files, to produce ones contaminated with data from multiple downloads, is possible using some of the proprietary software programmes.

Influence of mode settings on data

Most dive computers have several mode settings that can be adjusted before diving to show: whether seawater or freshwater is being dived in; the gas mixture being breathed; the level of conservatism being applied and the altitude of the dive.[2] All of these user settings have the ability to significantly alter the relevance of the data displayed to the actual incident.[3] It is not always straightforward to locate the user settings from a download; there is considerable variation between models and manufacturers and the information may either be with the individual dive information or in the summary logbook.

The dive profile, displayed as a simple depth/time profile, often attracts the most attention in any investigation as it presents an understandable visualisation of the incident dive. There are a number of issues related to the accuracy of the profile information recorded and displayed, but one of the main points to address is the accuracy of the depth recordings themselves. Nearly all dive computers measure only pressure and time. Usually the pressure sensor is temperature-compensated and highly accurate. However, calculating accurate depths of water from pressure recordings is not a simple task and is influenced primarily by the density of the water being dived in (temperature will also have an effect but this is much less than that of density). Dive computers can only convert the pressure measured to a depth estimate based on whatever water density the computer has been calibrated to. The calibration range and so the estimated depth cannot always be relied on to present an accurate record of the actual dive depths.[4] Whereas the variation in estimated depths will not affect the decompression information displayed by the computers (decompression obligations are calculated using the pressures recorded), caution should be employed when using computer depth to make decompression table-based comparisons or when using the integrated profiles to calculate relative decompression stresses.

Variations in recording and display of dive profiles

There is considerable variation in how different dive computers record and display the profile information.[2] This may cause difficulties in deducing an accurate profile of an incident dive. Central to these difficulties is understanding how the data are being displayed. Where the display is based on the maximum depth reached during a recording period, it is likely to give a relatively accurate record of the descent (until the descent is arrested) but a time-delayed record of the ascent (Figure 1). Ascent and descent rates will be more accurately displayed by computers that record the depth at the end of the recording period if no opposing changes in depth occur during that time; computers that record average depth values for each period provide little accurate profile information. The accuracies will also be affected by the length of the recording period. With relatively long recording intervals, it is possible that a significant depth excursion upwards in the water column could be missed entirely by computers recording only maximum or final depths, and the expanse of the excursion would be under-represented by computers measuring average values. However, it might be possible that an unrecorded depth excursion could still register an ascent rate warning and it is not uncommon to see ascent warnings on near horizontal profiles. But care should still be taken in interpreting the warning as an unrecorded depth excursion; ascent warnings can also be generated simply by the diver lifting their dive computer up to study.

In comparing more than one dive profile from the same incident dive, it will always be difficult to state the positions of the divers relative to each other with certainty. Examples of this are divers in a group ascending a shot-line together. When the dive profiles are compared, it will appear as though the divers were separated because of the different depths the divers were at when the recordings were made. These differences are then magnified if different models of computer are used that record and display depth differently, or convert pressure to depth differently. An opposite example is of two divers swimming around the hull of a wreck or a relatively level seabed but in different directions. The profiles could suggest they were together for some of the dive because of the similarity in the depths recorded.

Water temperature estimation

There is a high probability that the water temperatures displayed on dive computer downloads are linked in some way to the temperature-compensated pressure recorders. There is no evidence to support that temperature is being measured directly by computers and no information on how the measurements displayed are being derived. As a result, there is considerable variation across dive computers in the accuracy of the temperatures recorded in downloads.[4]

Air-integrated computers

Breathing rates can be calculated from downloads by using some measure of volume of gas breathed, corrected for ambient pressure derived through an integration of the dive profile. Sometimes, there will need to be an assumption, with confidence limits, of the volumes of gas consumed based on simple pre- and post-dive contents. There is likely to be a more accurate assessment from downloads that display information from dive computers that are integrated with a cylinder pressure sensor. The first 1–2 minutes of a dive profile will most likely yield erroneous breathing rates. This can be because of the delay in some computers in starting to register a dive; some computers undertake start-up checks for up to the first 80 seconds of a dive. Where the temperature of the water is less than air temperature there will likely be a concomitant drop in cylinder pressure that could suggest higher than actual breathing rates. In all cases, breathing rates should be presented at body temperature and pressure, saturated (BTPS) and not at ambient temperature and pressure (ATP). Thus breathing rates implied from pressure-corrected loss of cylinder content against time should be multiplied by:

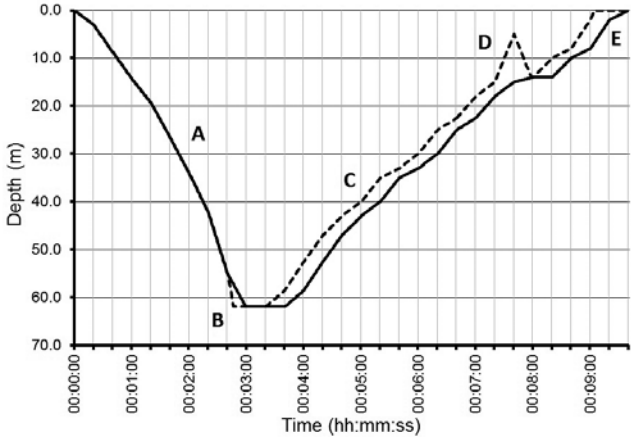
$$(273 + 37)/(273 + \text{ambient water temperature } {}^{O}C) \quad (1)$$

Where cylinder pressure recordings are more frequent than breathing rate, some form of rolling average will have to be employed to generate meaningful results.

Display of 'physiological' data

Dive computer downloads often present differing forms of 'physiological' data such as the diver's temperature,

Figure 1
A downloaded dive profile constructed from a real diving incident recorded and re-displayed by an UWATEC Aladin Ultra dive computer (solid line). The hashed line gives a different interpretation of what the actual dive profile could have been, based on the known fact that this model of computer displays profile information made up of the deepest depth estimate recorded during every 20-second period. With this recording format, it is unlikely that the displayed and actual descent profiles will differ if uninterrupted (A). One interpretation of the diver's actions in reaching the maximum depth is that they were in control and slowed their descent (B); however, they could have been in free-fall with the maximum depth reached much earlier during the 20-second recording period (B). In this type of computer there will be a near 20-second delay in the profile displayed during the ascent (C). Rapid ascents and descents in the water column lasting as long as 39 seconds in this case (D) could be missed off a displayed dive profile even though an ascent alarm may be indicated. Surfacing times on a display may be nearly 40 seconds later than the actual time of breaking surface (E).



breathing rate, microbubble formation, and the saturation levels in the tissue compartments. It is never clear how relevant these data are to the diver or how the levels are being calculated. A report of a computer download often has to discuss these data as they may appear pictorially on many of the figures being presented. However, it is often safer to dismiss these as indeterminate data and instead recalculate using probabilistic DCS modelling or some form of cumulative analysis (nitrogen loading or pressure root time) based on corrected integrated dive profiles.[5,6]

Laboratory and re-enactment testing

Dive computers can be tested in the laboratory or in incident re-enactments.[1] Bench testing can be relatively straightforward: e.g., comparing the accuracy of the unit's internal clock, or calibrated pressure exposures to validate the relative accuracy of depth estimation. Using the incident computer in a re-enactment helps to evaluate whether downloaded information accords with the information that was available to the diver at the time. However, with some models, it is important to realise that subsequent test dives may put the stored incident dive data at risk of loss.

Conclusions

Downloaded data from dive computers may seem to display incident dives accurately. However, the data are open to different levels of interpretation that can be challenged in the legal setting. Anyone using such downloads in fatal inquiries and/or related legal cases should be acquainted fully with the operational limits of the model under investigation.

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British Sub-Aqua Club (BSAC) diving incidents report 2013

Compiled by Brian Cummings, Diving Incidents Advisor

<http://www.bsac.com/core/core_picker/download.asp?id=24722&filetitle=Diving+Incident+Report+2013>

Summary of the 2013 report prepared by Colin Wilson

The BSAC started collecting and reporting on diving incidents in 1980, all of which data continue to be available.[1,2] Over the years, they have improved the amount and quality of the information collected, auditing the incidents reported to them and producing an annual report. Although these data are mainly from reports made by club members, other sources are also used. Information from these reports has allowed the BSAC to identify errors and mistakes and the audit cycle has led to changes in training methods to reduce these errors. The reports have been summarised annually in this journal since that of the 2006 incidents and fatalities.[3]

The 2013 report, as in previous years, covers the United Kingdom (UK) with a few reports from BSAC divers of overseas incidents. With 263 incidents reported, the decline in numbers continues, being 29% lower than in recent years and the lowest since 1994. This is thought to be owing to less diving in the UK rather than to safer diving or underreporting. Decompression incident reports were slightly higher than last year at 91, with 101 cases of decompression illness (DCI). A caveat, as in previous years, is made that a large number of the cases reported in the "*diver injury/illness*" category are probable DCI cases. The total in this group is 28, considerably lower than in recent years which may account for a higher number of reported DCI *c*ases. Previous reports have identified the ascent as being a potentially dangerous phase of the dive. This year's data seem to support the benefit of training directed to preventing ascent errors, with a continued fall in these to the smallest number (43) recorded since 1999. The involvement of the Coastguard, the Royal National Lifeboat Institute (RNLI) and Search and Rescue (SAR) helicopters were all less but as a percentage of the total reports they remain about the same.

Fatalities

Following last year's increase in recorded fatalities,[4] this year records 14 cases, close to the 10-year average of 15. BSAC members accounted for five of these, lower than the recent average. Unlike in other reports of fatalities, the quality and depth of information does not always allow clarity as to the root cause of these, though in most cases an educated assessment is made.[5] It is also clear that more than one cause may be at play when things go wrong. The causal factors associated with these fatalities are similar to previous years, with the analysis of the facts showing;

- Three cases suffered a non-diving-related medical incident (e.g., heart attack) while in the water. This also appears to be likely in one further case.
- Four cases involved separation; three of these were unintentional and as a result of problems experienced by the divers.
- Three cases involved divers who were unable to maintain positive buoyancy and who sank as a result.
- Three cases involved divers diving in a group of three, though it is not clear to what extent this action contributed to these fatalities.
- Two cases involved diving alone. In an additional case, it is not clear whether the diver was diving alone or became separated whilst underwater, having been found on the seabed entangled in rope.
- One case involved a diver using a rebreather. In the past, the numbers of rebreather fatalities have been higher.
- One case involved a diver reaching a maximum depth of 54 metres' sea water (msw).
- One case involved a diver who entered the water with his gas supply turned off.
- One case involved a double fatality. The bodies were retrieved nine days later from the seabed with little known currently as to the surrounding circumstances.

Five of these fatalities occurred in May. With only 43 incidents reported in that month, 12% of these were fatalities.

As in previous years, there is an increasing age in the reported fatalities. Eight of the 15 were aged 50 or over with an overall average age of over 52 years. A recent analysis of BSAC members reported that their average age had increased from 36 to 44 years since 1998. The average age of fatalities is increasing at twice the rate of the average age of the diving population. Although health and fitness does decline with increasing age, these numbers indicate that greater attention should be paid to the aging diving population. Accurate and honest medical reporting in the medical declaration form and subsequent follow up should be the acceptable, minimum approach.

From the fatalities section:

Case 1

"*A diver and his buddy had descended to a maximum depth of 19 msw. The pair remained together and the buddy monitored the diver closely for any problems during the early stages of the dive but none were apparent. Approximately 11 min into the dive the buddy lost sight of the diver and, having conducted a 360 degree turn to look for him, saw the diver a short distance away and approached him. The diver seemed to be drifting upwards and was moving his arms as if to push himself back down and then he stopped moving and sank to the seabed. The buddy made contact and found the diver's eyes closed and he was unresponsive. The buddy raised the diver up to the surface using a controlled buoyant lift and immediately commenced rescue breaths and summoned help. The diver was recovered into one of the two RHIBs covering the diving group, CPR was commenced and*

the alarm raised. Oxygen was used for enhanced rescue breaths. An RNLI RHIB on a training exercise in the area responded and took the diver and one of the diving crew aboard to assist with CPR efforts. The diver was returned to shore to be met by a paramedic and then transferred to hospital where he was pronounced deceased. The diver's death was confirmed as being due to natural causes because of heart disease."

Case 2

"*A group of divers were preparing to dive an offshore reef from a charter boat. One pair intended to carry out a negative entry and descend direct to the reef without resurfacing. The first diver entered the water whilst the second was still adjusting his kit and he commenced his dive. The second diver entered the water and it was quickly apparent that his gas was not switched on and he surfaced briefly. The boat skipper noticed this and shouted at other divers to jump in and assist the diver. One diver already on the stern lift of the boat jumped in and grabbed the diver but was unable to keep hold as he was being dragged down quite quickly. A second diver descended and located the unconscious diver on the bottom but was unable to lift him immediately and had to remove his BCD in order to bring him to the surface. The rescuer and casualty ascended from a depth of 36 msw to the surface in approximately 40 secs. The BCD was recovered by the first diver to enter the water to assist. On surfacing the casualty was recovered onto the charter boat and given CPR by the skipper and others on board for 30–40 min. Another charter boat in close vicinity spotted what was going on and contacted the Coastguard and co-ordinated communications. A rescue helicopter was tasked and airlifted the casualty to hospital where he was declared deceased. The diver who recovered the casualty was not taken by the helicopter but shortly after started to display symptoms of DCI following his fast ascent. A further emergency call was made and the diver was airlifted to a recompression facility for treatment.*"

Decompression incidents (DCI)

Ninety-one decompression incidents were reported, involving 101 cases of DCI. As in previous reports, identifying the cause(s) was difficult in a number of cases but where identified, and again some may involve a number of causes, these are similar to previous reports:

- 38 involved repetitive diving;
- 15 involved rapid ascents;
- 13 involved diving to depths greater that 30 msw;
- 13 involved missed decompression stops.

This list is virtually identical to past years. Several "*diver injury/illness*" reports are also probably DCI, though the rate of reporting is less than that of previous years.

From the DCI section:

Case 3

"*A diver had been diving for six consecutive days on a hardboat dive trip. Two dives a day were carried out using air and were within computer limits with no alarms showing on downloaded dive profiles. Decompression stops were in excess of the minimum required by the computer. Of the twelve dives carried out the deepest was to 40 msw on the third day and six of the dives were deeper than 30 msw but most were shelving reefs or wrecks allowing for gradual ascents. Surfacing from the last dive nothing was apparent and the diver helped in unloading the boat along with the rest of the group. Back at her accommodation the diver noticed a rash on her upper right arm and shoulder and suspected it was a skin DCI but, with no other symptoms present and with the rash fading, she did not want to worry her non-diving partner but, did inform the rest of the diving party. The following day, the diver had swelling to her right upper arm and shoulder together with some pain. The other divers insisted she tell her partner and they telephoned a decompression help line for assistance. The advice was that the diver should attend the local hospital where she was examined by the duty doctor in consultation with the decompression help line doctor. The diver was evacuated by helicopter to a recompression chamber and diagnosed with a rare lymphatic tissue and neurological DCI and underwent eight sessions of recompression treatment. The diver was advised not to dive for three months and to be medically assessed before diving again.*"

As in the 2012 report, there appears to be less diving being done in the UK. Yet again, avoidable incidents were documented. Thanks go to Brian Cummings and the BSAC team for collating this report, but we must also acknowledge those who have honestly reported their failures and misdemeanours. We are invited to browse these details and learn from others' mistakes.

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Key words
Recreational diving, accidents, diving deaths, abstracts, case reports

Letters to the Editor

The effect of vinegar on discharged nematocysts of *Chironex fleckeri*

We are writing because we have serious concerns about the statistical analyses and data interpretation reported by Welfare, Little, Pereira, and Seymour.[1] The authors state in the Abstract, "*Part 1: There was a 69 +/- 32% (F = 77, P < 0.001) increase in venom discharge after vinegar was applied compared to post electrical stimulation*." The recovery of venom protein from a membrane after the application of vinegar subsequent to electrically stimulating tentacle cnidae to discharge, W4, was compared with protein recovered post stimulation in a saline wash, W3. Figure 2 shows W3 to be approximately 23 +/- 20%. While the authors imply the statistical difference between "*venom discharge after vinegar was applied compared to post electrical stimulation*", or W4 vs W3, only the overall ANOVA significance comparing all four treatments was quoted ($F = 77$, $P < 0.001$) and no statistical significance was provided for this specific W4 vs W3 comparison. If we assume that standard errors of the means (SEMs) were used in Figure 2, a simple *t*-test will provide a *P*-value of only 0.11, comparing W4 and W3, an insignificant finding. The comparison of W4, post electrical stimulation to W1 the pre-stimulation control would yield a significant value of $P < 0.001$ but this is hardly surprising and intuitively obvious.

For this and the following reasons, it seems that the data may not have been properly analysed and not properly presented:

- The same three samples seem to have been used for W1–W4, resulting in internally matched samples, but the data were analysed using ANOVA, assuming samples in different treatments are all independent.
- It is not clear what the value after the "+/-" represents, CI, SEM, or SD, as this is not stated in the caption.
- It is not clear what the 3 x 82 subscript means for the reported F = 77.12 (page 32, right column, line 2 below Table 1).

We respectfully recommend that the editors engage a third-party statistician to run an independent analysis of the primary data. If these statistical errors exist, we suggest that the publication be retracted.

It is troubling that this small study reporting recovery of cytolytic activity from a placental membrane proxy of envenomation has been used to launch wildly extrapolative press releases despite over 40 years of using vinegar as a first-response treatment without a clear case of death resulting from its use. Statements such as "*For decades experts have recommended vinegar to treat box jellyfish stings. Now, Queensland researchers have discovered the cure can kill*"[2] are simply not true; there was no death or killing in the Welfare et al study.

Claims that "*Vinegar may kill rather than cure victims of box jellyfish stings ... The remedy, used for decades to treat stings, causes up to 60 per cent more venom from the lethal jellyfish to be discharged into the victim*"[3] are also not supported by these data. There were no 'victims' and the slight elevation in the amount of protein recovered in W4 vs W3 was not statistically significant. The authors also report that "*vinegar promotes further discharge of venom from already discharged nematocysts*" but data show only modest enhanced recovery of cytolytic activity from the membrane, not the degree of cnidae discharge. Finally, the authors do not consider alternative potential causes of enhanced cytolytic recovery, e.g., vinegar improves recovery of certain venom component activities. Thus, these findings may suggest the diametric opposite to the authors' conclusion – that is, vinegar may enhance venom extraction from a sting site and thus increase survival. However, without validation of this membrane model in an authentic animal model, there is no clear way to interpret these data let alone extrapolate to make emergency medical care recommendations.

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Key words
Jellyfish, marine animals, envenomation, statistics, research, letters (to the Editor)

Reply:

We thank Yanagihara and Chen for their comments and for the opportunity to further the discussion.[1] Our statistician has re-examined (and reanalysed) these data, and we have supplied our data to an independent statistician (who supported our subsequent re-analysis) and are more than willing to supply these data to the journal editors should they feel this is necessary. Furthermore, the manuscript was independently reviewed by two reviewers who expressed no concern over our analysis. We are confident of our results.

Yanagihara and Chen have incorrectly assumed that the errors displayed in Figure 2 are SEM. These errors represent the 95% confidence limits (CL) and as such their arguments are invalid. Furthermore, they outline that no statistical significance was provided for the specific W4 vs. W3 comparison. Although no specific statistics were displayed in the article, we do outline that LSD post hoc analysis was conducted and the means and 95% CL (as signified in Figure 2) that were significantly different were listed. This analysis showed that the percentage of venom discharged after the application of voltage (W2) and after the application of vinegar (W4) were significantly different from one another and both were significantly higher than either the initial before-voltage percentage (W1) or after the third washing (W3) after voltage application.

It is further suggested that the samples used (W1 to W4) are internally matched samples and hence ANOVA is inappropriate. They suggest that a simple *t*-test would give different results. To alleviate their concerns, we have re-analysed the data using a paired *t*-test, comparing the level of protein present after the third washing (W3) since voltage was applied to the tentacle and the amount of protein present after vinegar was applied (W4). We paired each sample with itself (which effectively removes the issues surrounding analysis of internally matched samples). This analysis showed that the difference between the matched pairs was significant ($t = 8.938$, df = 2, $P = 0.012$). We further re-analysed these data comparing the mean protein expression after vinegar application (W4) to a standard value (23.2%) which was the mean percentage found after three washings (W3) post voltage application. Once again, the difference was found to be significant ($t = 6.012$, df = 2, $P = 0.027$).

We would argue, however, that the use of *t*-tests, as suggested by Yanagihara and Chen, is inappropriate owing to a possible non-normal distribution of the data. To address this, we further analysed these data using a non-parametric median test to a binomial distribution for data collected after the third washing (W3) post voltage application and data collected after the application of vinegar (W4). We used a one-sample median test to a binomial.[2] This statistical test is non-parametric as no assumption is made about the form of the population distribution except that it is continuous. This analysis once again revealed a significant difference between the treatments ($Z_b = 1.73$, $P = 0.04$) and, as such, the amount of protein after vinegar application is greater than after the washing protocol; that is, the application of vinegar increases the amount of venom expressed. Finally, we have reanalysed our data using a Friedman's test (as suggested by another independent biostatistician consulted by the Editor) and once more found that the application of vinegar increased the presence of toxin ($\chi^2 = 9.0$, df = 3, $P = 0.029$).

We thank Yanagihara and Chen for pointing out an issue of the degrees of freedom listed. We realize there was a transcription error that was not identified by the authors within the proofs. Where it reads ($F = 77.12_{3x82}$), it should read ($F = 77.12_{3x8}$).

Yanagihara and Chen have also expressed concerns about the press releases associated with this paper. We were contacted by the media as a result of the article's abstract release and the cover page of this journal for March 2014 with the heading "*Does vinegar make box jellyfish stings worse?*" Our sole press release (in response to the above) stated our findings and suggested a review of the current guidelines, as we do in the article. We do not have control over what the media publishes. We would point out that in every interview conducted by the authors, it was explicitly stated that first aid for cubozoan envenomings in Australia should continue to follow the ARC guidelines unless these guidelines are changed.

We remind Yanagihara and Chen that the scientific evidence supporting the use of vinegar as first aid is poor and we have seen an increased use of opioid analgesia in patients with Irukandji syndrome, who received vinegar (compared to those who did not).[1,3] Yanagihara and Chen suggest vinegar may enhance venom extraction from a sting site and thus increase survival. This is interesting speculation at best, with no data or evidence to support such an assumption.

In summary, our data show that vinegar promotes further release of venom from *Chironex fleckeri* tentacles that have been electrically discharged. We reiterate that we believe our findings are sufficiently significant for consideration in the development of first-aid guidelines, particularly in the face of a clear absence of any previous evidence supporting vinegar, which has always been assumed to be efficacious and safe.

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Key words
Jellyfish, marine animals, envenomation, statistics, research, letters (to the Editor)

Measuring aerobic fitness in divers

The editorial by Bosco, Paoli and Camporesi in the last issue of this journal provides an interesting overview of some of the factors that are either known or suspected to be important in the physiological health of divers.[1] The part pertinent to our paper concerns the meaning and use of metabolic equivalents (MET).[2] Our goal was to estimate the metabolic effort required for a substantial sample of recreational dives. Computing MET values based on an assumed resting oxygen consumption rate of 3.5 millilitres of oxygen per kilogram body mass per minute is well established. Most pointedly, MET is used in the Recreational Scuba Training Council (RSTC) Guidelines for Recreational Scuba Diver's Physical Examination found in the Medical Statement documentation.[3] Given the increasingly widespread use of the RSTC assessment, it makes the most sense to be consistent. Concerns over whether or not a more appropriate index value could be used are moot. Anyone wishing to compute a different base for 1.0 MET can simply cross-multiply and divide.

The question to be answered is not what level of aerobic capacity is desirable for divers, the answer to that is the higher the better. The critical question is what constitutes a reasonable minimum threshold aerobic capacity consistent with operational safety. The authors mention the often invoked 13 MET capacity identified as a threshold for US Navy divers. What is typically ignored, however, is the fact that the Navy has far more applicants for dive school than posts to be filled, making very stringent selection standards feasible even if not truly operationally necessary. It is not at all clear that this is a reasonable threshold for the broader diving community. Despite this, the RSTC documentation adheres to the traditional position. "*Formalized stress testing is encouraged if there is any doubt regarding physical performance capability. The suggested minimum criteria for stress testing in such cases is at least 13 METS [sic]. Failure to meet the exercise criteria would be of significant concern*." This is contrary to the available data. A review of 14 studies in which the aerobic capacity of divers was measured found that mean aerobic fitness ranged from 37–57 $mL \cdot kg^{-1} \cdot min^{-1}$ (10.6–16.3 MET).[4] The lowest individual scores were below 5.0 MET. The threshold of 13 MET was exceeded by the group mean in only six of the 14 studies described. This certainly does not support 13 MET as a meaningful threshold for participation.

Our current work was intended as a simple effort to begin to assess the aerobic demands of recreational diving. It is our hope to promote discussion that is willing to risk the heresy of challenging conventional wisdom and to stimulate additional research.

We certainly agree with the authors and feel strongly that enhanced in-water evaluation of physical fitness is desirable to establish diver readiness. We would not, however, refer to this as a "medical examination" since it is likely that it will largely be dive professionals and not clinicians that conduct the evaluations.

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Key words
Fitness to dive, oxygen consumption, medicals – diving, letters (to the Editor)

Diving and hyperbaric medicine: an undergraduate's experience

As part of my undergraduate medical degree and as a keen scuba diver, I undertook my clinical elective at the Hyperbaric Medical Centre, Sharm el Sheikh, Egypt (Figure 1). The Centre first opened in 1993, and its hyperbaric chamber quickly became one of the busiest in the world. This was mainly owing to the popularity of Sharm with both scuba and free divers for the pristine reefs and rich underwater wildlife. The Centre offers consultations and diving medical examinations, as well as a 24 h emergency service. In recent years the number of divers has been affected, with diving eligibility examinations and injuries halved to around 1,200 each year, owing to the country's political climate.

During my elective, I learnt about diving physiology and hyperbaric chamber use, how to diagnose and treat common and severe diving injuries, and become proficient in diving medical eligibility assessment. Diving medicine and hyperbaric oxygen therapy are not covered in the core medical curriculum, despite doctors frequently certifying divers. Most days' work involved carrying out several diving medicals for instructors and training course candidates, or those who declared a pre-existing medical condition on their medical statement.[1] After observing the diving medical specialists, I was able to conduct my own consultations, which involved taking a focused history, a physical examination and, if necessary, cardiopulmonary exercise testing. The most useful skill I gained was confidence counselling divers on how to manage and prevent further injuries. Certain conditions (such as sinus congestion,

asthma, diabetes and certain prescription medications) are known to increase the risk of diving injuries, and these are not always obvious. Finally, I observed and participated in the diagnosis and treatment of a wide range of diving injuries, from middle ear barotrauma, pulmonary barotrauma and animal stings to decompression illness (DCI).

Later, I reported a case of *cutis marmorata*.[2] Interestingly within days of being published online, this case report and the accompanying image appeared on a popular diving forum, with divers commenting on the usefulness of seeing first-hand such a common clinical sign of DCI.

On one of my general practice placement visits, I saw a patient who had developed a middle ear barotrauma. Following appropriate advice, she wanted to discuss her daughter's diving problems and I referred her to the UK Sports Diving Medical Committee website for contact details of local, approved diving medical referees.[3] Potential divers requiring a medical clearance often present to their own doctor (general practitioner), who may not be aware of the diving regulations and contra-indications so they can counsel patients appropriately.[4] With this in mind, I have set out to raise awareness amongst general practitioners (Modell MM, Glew S, Sornalingam S, Cooper M, unpublished work) on how to provide onward referral to diving medical specialists.

I would highly recommend such an elective to both medical students and qualified doctors interested in emergency medicine or sports medicine. With dive trips to remote locations easily accessible, both divers and doctors should be aware of severe diving-injuries.

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Key words
Diving, hyperbaric medicine, scuba, medical education, tourism, fitness to dive, letters (to the Editor)

Figure 1
Fourth-year UK medical student Michael Modell at the Hyperbaric Medical Centre, Sharm el Sheikh, Egypt

Immersion pulmonary oedema and diving fatalities

The report by Smart et al is very interesting.[1] They note that "*forensic pathologists should be properly trained in and have guidelines for the conduct of post-immersion and post-diving autopsies*." In the medical curriculum, there is little on diving medicine and many pathologists have little knowledge on this issue.[2,3] For example, in coastal Thailand, a very popular region for scuba diving, there are no pathologists with a specific training in diving medicine, and the issue here is how to improve their knowledge. The investigation of diving fatalities is well summarised by Busuttil and Obafunwa: "*a multi-disciplinary approach that involves co-divers and instructors, the rescue team, the police, forensic scientists, diving equipment suppliers, underwater physiologists and physicians, decompression chamber personnel, general practitioners, relatives and the forensic pathologist*" is required for any investigation of diving deaths.[3]

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Key words
Immersion, pulmonary oedema, scuba diving, deaths, letters (to the Editor)

Continuing professional development

Oxygen in wound healing

Christian Fabricius

Accreditation statement

INTENDED AUDIENCE

The intended audience consists of all physicians subscribing to *Diving and Hyperbaric Medicine* (DHM), including anaesthetists and other specialists who are members of the Australia and New Zealand College of Anaesthetists (ANZCA) Diving and Hyperbaric Medicine Special Interest Group (DHM SIG). However, all subscribers to DHM may apply to their respective CPD programme coordinator or specialty college for approval of participation.

This activity, published in association with DHM, is accredited by the ANZCA Continuing Professional Development Programme for members of the ANZCA DHM SIG under Learning Projects: Category 2 / Level 2: 2 credits per hour.

OBJECTIVES

The questions are designed to affirm the takers' knowledge of the topics covered, and participants should be able to evaluate the appropriateness of the clinical information as it applies to the provision of patient care.

FACULTY DISCLOSURE

Authors of these activities are required to disclose activities and relationships that, if known to others, might be viewed as a conflict of interest. Any such author disclosures will be published with each relevant CPD activity.

DO I HAVE TO PAY?

All activities are free to subscribers.

Key words
Oxygen, wounds, transcutaneous oximetry, MOPS (maintenance of professional standards)

Recommended background reading

Practitioners are referred to the following background references and reading.

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How to answer the questions

Please answer all responses (A to E) as True or False.
Answers should be posted by email to the nominated CPD coordinator.
EUBS members should send their answers to Lesley Blogg.
E-mail: <lesley.blogg@eubs.org>.
ANZCA DHM SIG and other SPUMS members should send their answers to Neil Banham.
E-mail: <Neil.Banham@health.wa.gov.au>.
If you would like to discuss any aspects with the author, contact him at**:** <christian.fabricius@mecon1.se>.

On submission of your answers, you will receive a set of correct answers with a brief explanation of why each response is correct or incorrect. A correct response rate of 80% or more is required to successfully undertake the activity. Each task will expire within 24 months of its publication to ensure that additional, more recent data has not superseded the activity.

Question 1. Chronic wounds are hypoxic. The oxygen partial pressure in a chronic wound is believed to be:

A. 0–10 mm Hg;
B. 5–20 mm Hg;
C. 10–30 mm Hg;
D. 20–40 mm Hg.

Question 2. During the inflammatory phase of wound healing, leukocyte oxygenase produces large amounts of oxidants. To work at 50% and 90% of maximum enzymatic speed a wound oxygen partial pressure of between what values is needed?

A. 10 and 50 mm Hg, respectively;
B. 20 and 100 mm Hg, respectively;
C. 25 and 250 mm Hg, respectively;
D. 50 and 400 mm Hg, respectively.

Question 3. The production of collagen in a wound is proportional to an oxygen partial pressure between:

A. 0 and 100 mm Hg;
B. 20 and 120 mm Hg;
C. 50 and 150 mm Hg;
D. 0 and 400 mm Hg.

Question 4. When using a Clarke electrode for transcutaneous oxygen monitoring (TCOM), the following must be checked to ensure patient safety:

A. that the measuring time is kept below 20 minutes;
B. signs of thermal injury at the measuring site;
C. the electrical isolation of the measuring electrode;
D. that there is air between the skin and the measuring electrode.

Question 5. For healthy persons, chest (pre-sternal) TCOM as compared with arm or hand TCOM show that:

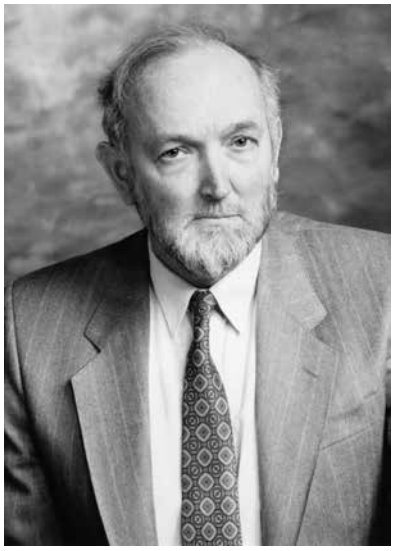
A. chest pO_2 = arm and hand pO_2;
B. chest pO_2 > arm and hand pO_2;
C. chest pO_2 < arm and hand pO_2;
D. none of the above.

Question 6. TCOM at the level of the malleoli of healthy subjects, 22–80 years of age, show an oxygen partial pressure of:

A. 42–85 mmHg when the subject is breathing air and 78–468 mmHg when the subject is breathing 100% oxygen;
B. 1–60 mmHg when the subject is breathing air and 55–389 mmHg when the subject is breathing 100% oxygen;
C. 23–48 mmHg when the subject is breathing air and 98–256 mmHg when the subject is breathing 100% oxygen.

Obituary

Thomas Richard Hennessy, MSc, PhD, CMTH, FIMA, FSUT, 1940–2014



Tom Hennessy began diving in 1964 with the Cambridge University Underwater Exploration Group, while researching theoretical hydrodynamics at Churchill College. However, diving physiology took a hold and in 1973, as a Senior Lecturer in Applied Mathematics, he completed a PhD in the biophysics of inert gas exchange in decompression at the University of Cape Town.

During 1968-69, as Diving Officer of the Atlantic Underwater Club in Cape Town, Tom introduced a diver and instructor training programme based on that of the British Sub-Aqua Club (BSAC), later adopted as the national standard in South Africa, and as President of the South African Society for Underwater Science 1975–76 he represented the special needs of scientific divers in new professional diving legislation.

The opportunity to do real decompression research lured Tom back to the UK in 1976 to work with Val Hempleman at the Royal Naval Physiological Laboratory (RNPL). He analysed the RNPL deep heliox saturation dives conducted between 1969 and 1979 and successfully calibrated a mathematical model of saturation/'big-drop, long-stop' decompression rules for safe saturation-excursion dives. As a Principal Research Fellow, he co-developed with Tom Shields new RN trimix tables to 80 m depth. Thereafter, as Project Leader, he developed new excursion nitrox tables to 60 m for the Department of Energy, released in 1985 by the Construction Industry Research and Information Association Underwater Engineering Group.

In 1984, concerned by the withdrawal of MoD support for diving research, Tom transferred to the Admiralty Research Establishment as Head of Section in Non-Acoustic Detection and Wake Hydrodynamics, managing a large research programme in ship and submarine wake detection for five years. In 1987 in his spare time, Tom developed the BSAC '88 Tables which became the standard for decompression training of BSAC divers throughout the world, and in 1996 he introduced the BSAC Nitrox Tables. He was awarded the premier BSAC Colin McLeod Medal in 1991 for developing the air decompression procedures, judged to be a significant improvement in diver safety and the understanding of decompression. Also for the BSAC in 2009, he produced new, accelerated oxy-nitrogen decompression tables (the Ox-Stop Tables) which use high oxygen mixtures during the ascent phase to shorten the ascent time.

As a Consultant to Wharton Williams Underwater Engineering Contractors, he developed special long-exposure air tables and rules for planning multi-level heliox saturation/excursion dives in commercial diving operations, and was awarded the Houlder Cup in 2000 by the Society for Underwater Technology for his contributions to North Sea diving operations.

Tom later held the post of Senior Lecturer in Medical Informatics in the Centre for Measurement and Information in Medicine at City University. In 1991, he established jointly with Guy's, King's & St Thomas' Medical Schools the first MSc in Medical Informatics course in Europe and was its first Course Director until 2001. In 1990, he was a member of the European planning group that successfully launched an Erasmus MSc course in Health Informatics at the University of Athens, and he subsequently taught a module on the Athens course over the next decade.

In 2011, Tom was elected an Emeritus Member of the Undersea and Hyperbaric Medical Society of which he had been a member since 1972. He published about 50 papers in diving physiology and decompression modelling and co-authored a book on nanoscale fluid dynamics in physiological processes.

Tom had heart disease for 30 years and died after undergoing his third major heart surgery. He is survived by his wife, two daughters, five grandchildren and a sister, his only sibling.

Deborah Hennessy

Key words
Obituary, decompression tables, diving tables, diving research

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Editor's note: Dr Hennessy's research and other documents will probably be archived under the supervision of the Imperial College Archivist whilst a suitable repository is determined. Meantime, enquiries should be addressed to Dr Deborah Hennessy at <deb.hennessy@btinternet.com>.

SPUMS Notices and news

SPUMS notices and news are now on the website <www.spums.org.au>

SPUMS 44th ASM 2015

Venue: Palau Royal Resort, Malakai, Palau, Micronesia
Dates: 16–23 May 2015

Guest Speaker: Neal Pollock, Duke University and Director of Research DAN International

Topics: Diabetes and diving; the older diver; breath-hold diving

Convenor: Dr Catherine Meehan, Cairns

Preferred travel from Australia will be with China Airlines ex Brisbane.
This avoids lengthy layovers and awkward connections.
Several packages with significant cost savings are likely to be available.
The link to the conference booking site will be available at the end of August on <spums.org.au>

For further information e-mail: <cmeehan@mcleodstmed.com.au>

Douglas Walker, Life Member of SPUMS

Dr Douglas Walker first learnt to dive with the British Sub-Aqua Club. His involvement in diving medicine stemmed from the time he attended the Royal Navy Diving Medical Officers' course in Alverstoke, UK, even before the course was advertised as being available to civilians. For many years after immigrating to Australia, he was a general practitioner on the northern beaches of the Sydney region, and was a foundation member of the South Pacific Underwater Medicine Society along with Dr Deal, Surgeon Lt Cmdr Edmonds, Sgn Cmdr Gray, Sgn Lt Thomas, and Dr Unsworth. The inaugural meeting of SPUMS was held in the Wardroom of HMAS Penguin on 03 May 1971. The initial subscription for membership was set at $2! Douglas was Editor of the *SPUMS Journal* from 1974 to 1990, with John Knight assisting him from 1979. He again sat on the SPUMS executive committee between 1999 and 2004.

Douglas first reported on the subject of diving accidents and deaths at the first SPUMS ASM held on Heron Island in 1972. However, he had begun to collect data on Australian diving deaths as early as the late 1960s. He determinedly and singlehandedly searched for and followed up leads from newspapers and elsewhere and painstakingly collected information from coronial offices throughout all Australian States and Territories – a project entitled *Project Stickybeak*. The results of his labours have been published in the *Journal of the South Pacific Underwater Medicine Society* since 1972 and these annual reports have provided a solid foundation and leading light for the investigation and reporting of diving fatalities internationally. He received a Master of Medicine from the University of Sydney in 1994 for his dissertation based on this work.

Carl Edmonds commented recently that "*Douglas is one of the most generous of researchers. When I was reviewing the causes of diving deaths, Doug not only supplied his fatality reports but he also literally gave me all his original documentation and clinical material – not a précis, but boxes and boxes of original data. This never happened to me any other time in my professional life. It demonstrated how honest and helpful he was.*"
In a recent phone conversation with Karen Richardson, Doug commented that he had just turned 90 and had given up diving a few years ago as he was no longer physically capable and felt he would prefer to be in a position to write the coronial reports rather than be a subject in one of them! Douglas Walker has made an invaluable contribution to diving medicine and safety, and his name is presented to you today [*at the 44th Annual General Meeting of SPUMS*] to acknowledge him as a Life Member of SPUMS.

SPUMS and Facebook

Remember to 'like' SPUMS at:
<http://www.facebook.com/pages/SPUMS-South-Pacific-Underwater-Medicine-Society/221855494509119>

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

In order for the Diploma of Diving and Hyperbaric Medicine to be awarded by the Society, the candidate must comply with the following conditions:

1 (S)he must be medically qualified, and remain a current financial member of the Society at least until they have completed all requirements of the Diploma.
2 (S)he must supply evidence of satisfactory completion of an examined two-week full-time course in diving and hyperbaric medicine at an approved facility. The list of such approved facilities may be found on the SPUMS website.
3 (S)he must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
4 (S)he must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval before commencing their research project.
5 (S)he must produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.

In the absence of other documentation, it will be assumed that the paper is to be submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the 'Instructions to Authors' available on the SPUMS website <www.spums.org.au> or at <www.dhmjournal.com>.

The paper may be submitted to journals other than *Diving and Hyperbaric Medicine*; however, even if published in another journal, the completed paper must be submitted to the Education Officer for assessment as a diploma paper. If the paper has been accepted for publication or published in another journal, then evidence of this should be provided.

The diploma paper will be assessed, and changes may be requested, before it is regarded to be of the standard required for award of the Diploma. Once completed to the reviewers' satisfaction, papers not already submitted to, or accepted by, other journals should be forwarded to the Editor of *Diving and Hyperbaric Medicine* for consideration. At this point the Diploma will be awarded, provided all other requirements are satisfied. Diploma projects submitted to *Diving and Hyperbaric Medicine* for consideration of publication will be subject to the Journal's own peer review process.

Additional information – prospective approval of projects is required

The candidate must contact the Education Officer in writing (or email) to advise of their intended candidacy and to discuss the proposed topic of their research. A written research proposal must be submitted before commencement of the research project.

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis and if the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered. It is expected that the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author where there are more than one.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: <www.nhmrc.gov.au/_files_nhmrc/publications/attachments/r39.pdf>, or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. Human studies must comply with the Declaration of Helsinki (1975, revised 2013). Clinical trials commenced after 2011 must have been registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry <http://www.anzctr.org.au/> and details of the registration provided in the accompanying letter. Studies using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted.

The SPUMS Diploma will not be awarded until all requirements are completed. The individual components do not necessarily need to be completed in the order outlined above. However, it is mandatory that the research project is approved prior to commencing research.

As of 01 June 2014, projects will be deemed to have lapsed if

1 The project is inactive for a period of three years, or
2 The candidate fails to renew SPUMS Membership in any year after their Diploma project is registered (but not completed).

With respect to 1 above, for unforeseen delays where the project will exceed three years, candidates must advise the Education Officer in writing if they wish their diploma project to remain active, and an additional three-year extension will be granted.
With respect to 2 above, if there are extenuating circumstances that a candidate is unable to maintain financial membership, then these must be advised in writing to the Education Officer for consideration by the SPUMS Executive.

If a project has lapsed, and the candidate wishes to continue with their DipDHM, then they must submit a new application as per these guidelines.

The Academic Board reserves the right to modify any of these requirements from time to time.
As of June 2014, the SPUMS Academic Board consists of:
Dr David Wilkinson, Education Officer;
Associate Professor Simon Mitchell;
Associate Professor (retired) Mike Davis;
Dr Denise Blake.

All enquiries and applications should be addressed to:
David Wilkinson
Fax: *+61-(0)8-8232-4207*
E-mail: *<education@spums.org.au>*

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research, medical society

Notices and news

EUBS notices and news and all other EUBS information is to be found on the society website: <www.eubs.org>

41st EUBS Annual Scientific Meeting 2015

Dates: 19–22 August, 2015
Venue: Amsterdam

For prelininary information see:
<www.eubs2015.org>

The

website is at
<www.eubs.org>

Members are encouraged to log in and to keep their personal details up to date

German Society for Diving and Hyperbaric Medicine

An overview of basic and refresher courses in diving and hyperbaric medicine, accredited by the German Society for Diving and Hyperbaric Medicine (GTÜeM) according to EDTC/ECHM curricula, can be found on the website:
<http://www.gtuem.org/212/Kurse_/_Termine/Kurse.html>

DAN Europe

DAN Europe has a fresh, multilingual selection of recent news, articles and events featuring DAN and its staff.
Go to the website: <http://www.daneurope.org/web/guest/>

Capita Selecta Dive Research English Seminars 2014 University of Amsterdam, The Netherlands

29 November 2014: *Breath-hold diving*
Speakers: Rik Roskens; Erika Schagatay, environmental physiologist; Jochen Schipke, medical physiologist and diving physician

For full information contact: <www.duikresearch.org>

Scott Haldane Foundation

The Scott Haldane Foundation is dedicated to education in diving medicine, and has organized more than 150 courses over the past 19 years, both in the Netherlands and abroad. Below is a list of remaining courses for 2014.

The courses Medical Examiner of Diver (parts I and II) and the modules of the Diving Medicine Physician course comply fully with the ECHM/EDTC curriculum for Level 1 and 2d respectively and are accredited by the European College of Baromedicine.

Remaining courses for 2014
04 October: Refresher course. AMC, Amsterdam
08–15 November: Basic course (medical examination of divers) Part 1. Costa Rica
15–22 November: 22nd In-depth course Diving Medicine: case-based diving medicine. Costa Rica
22–29 November: 22nd In-depth course Diving Medicine: case-based diving medicine. Costa Rica

For further information: <www.scotthaldane.org>

Dr Kate Lambrechts

In June 2014, Kate Lambrechts successfully defended her doctoral dissertation at the Université de Bretagne Occidentale, Brest, France.

Platelet activation and vascular dysfunction after SCUBA diving: study of physiological mechanisms and their interactions

Some of this work may be accessed in this issue and at:
<http://www.ncbi.nlm.nih.gov/pubmed/23949788>
<http://www.ncbi.nlm.nih.gov/pubmed/24400144>

Certificate in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists

Eligible candidates are invited to present for the examination for the Certificate in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists.

All details are available on the ANZCA website at:
<http://anzca.edu.au/edutraining/DHM/index.htm>

Suzy Szekely, FANZCA, Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine.
E-mail: *<Suzy.Szekely@health.sa.gov.au>*

British Hyperbaric Association Annual Meeting 2014

Dates: 07–08 November 2014
Venue: The East Riding Medical Education Centre
Hull Royal Infirmary, East Yorkshire

Day 1: Oxygen and the traumatised brain
Day 2: Diving physiology / diving medicine

Keynote speakers:
Brad Sutherland, UK, Shia Efrati, Israel, Galan Rockwood, USA, Ole Hildegard, Denmark, David Doollete, USA, Martin Sayer, UK

The call for abstracts is now open. Please make submissions of 300 works or less to: <gerardladen@aol.com>

Diving and Hyperbaric Medicine Index of contents, Vol 43, 2013

The Index of contents, Volume 43, 2013, is on the journal website <www.dhmjournal.com> and also on the SPUMS and EUBS websites.

Back articles from DHM

After a one-year embargo, articles from *Diving and Hyperbaric Medicine* are placed on the Rubicon Foundation website <www. http://rubicon-foundation.org/>.
This is an open-access database, available free of charge and contains many other publications. At present, this is not fully up-to-date for DHM but articles to the September 2012 issue are now available. Rubicon seeks donations to support its work to document the hyperbaric scientific literature.

More recent articles or other enquiries about articles should be sent to: <editorialassist@dhmjournal.com>
Embargoed articles will be charged for – fee on aplication.

Royal Adelaide Hospital Hyperbaric Medicine Unit Courses 2014

Medical Officers' Course
Part 1: 01– 05 December (Lectures)
Part 2: 08–12 December

DMT Courses
Full: 06–24 October
Refresher: 22 September–03 October

All enquiries to:
Lorna Mirabelli, Course Administrator
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lorna.Mirabelli@health.sa.gov.au>

Royal Australian Navy Medical Officers' Underwater Medicine Course 2014

Dates: 06–17 October 2014
Venue: HMAS PENGUIN, Sydney

The MOUM course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Considerable emphasis is placed on the contra-indications to diving and the diving medical, together with the pathophysiology, diagnosis and management of the more common diving-related illnesses. The course includes scenario-based simulation focusing on management of diving emergencies and workshops covering the key components of the diving medical.

Costs: AUD1,355 (without accommodation)
AUD2,300 (approx. with accommodation at HMAS Penguin)

For information and application forms contact:
Rajeev Karekar, for Officer in Charge,
Submarine and Underwater Medicine Unit
HMAS PENGUIN
Middle Head Rd, Mosman
NSW 2088, Australia
Phone: +61-(0)2-9647 5572
Fax: +61-(0)2-9960 4435
E-mail: <Rajeev.Karekar@defence.gov.au>

The ANZ Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine 2015

Dates: 23 February–06 March
Venue: Prince of Wales Hospital, Sydney, Australia

Course content includes:
- History of hyperbaric oxygen
- Physics and physiology of compression
- Accepted indications of hyperbaric oxygen
- Wound assessment including transcutaneous oximetry
- Visit to HMAS Penguin
- Visit to the NSW Water Police
- Marine envenomation
- Practical sessions including assessment of fitness to dive

Approved for the ANZCA CPD programme (knowledge and skills category):
56 hours for attendance at lectures/presentations for one credit per hour.
24 hours for workshops/PBLDs/small group discussions for two credits per hour

Contact for information:
Ms Gabrielle Janik, Course Administrator
Phone: +61-(0)2-9382-3880
Fax: +61-(0)2-9382-3882
E-mail: <Gabrielle.Janik@sesiahs.health.nsw.gov.au>

UHMS Annual Scientific Meeting 2015

Dates: 04–06 June 2015 (pre-course 03 June)
Venue: Hilton Bonaventure, Montreal, Canada
More information coming soon: <www.uhms.org>

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village
Victoria, 3172, Australia
E-mail: <hdsaustraliapacific@hotmail.com.au>
Website:
<www.classicdiver.org>

18th International Congress on Hyperbaric Medicine

02–06 December 2014
Buenos Aires, Argentina

The ICHM is a worldwide organization for physicians and scientists interested in diving and hyperbaric medicine. The organization has minimal formal structure and is entirely dedicated to hosting an international scientific congress every three years.

ICHM Committee (2011–2014):
President: Prof Dr Jorge B Pisarello (Argentina)
Executive Director: Dr Alessandro Marroni (Italy)
Secretary: Assoc Prof Michael Bennett (Australia)

Registration: Online registration is now open
Website: www.ichm2014.com.ar
E-mail for further information: <info@eidosestudio.com>

Hyperbaric Oxygen, Karolinska

Welcome to: <http://www.hyperbaricoxygen.se/>.
This site, supported by the Karolinska University Hospital, Stockholm, Sweden, offers publications and free, high-quality video lectures from leading authorities and principal investigators in the field of hyperbaric medicine.

You need to register to obtain a password via e-mail. Once registered, watch the lectures online, or download them to your iPhone or computer for later viewing. We offer video lectures from:
- The 5th Karolinska PG course in clinical hyperbaric oxygen therapy, 07 May 2009.
- The European Committee for Hyperbaric Medicine "*Oxygen and infection"* Conference, 08–09 May 2009.
- The 17th International Congress on Hyperbaric Medicine, Cape Town, 17–18 March 2011.

Also available is the 2011 Stockholm County Council report: *Treatment with hyperbaric oxygen (HBO) at the Karolinska University Hospital*.

For further information contact:
Folke Lind, MD PhD
E-mail: <folke.lind@karolinska.se>
Website: <www.hyperbaricoxygen.se>

Instructions to authors

The 'short' *Instructions to Authors* will no longer be printed in each issue of the Journal. Please refer to the *Diving and Hyperbaric Medicine* website: <www.dhmjournal.com> for a downloadable pdf of the full instructions (revised June 2014).

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA
1800-088200 (in Australia, toll-free)
+61-8-8212-9242 (International)

NEW ZEALAND
0800-4DES-111 (in New Zealand, toll-free)
+64-9-445-8454 (International)

ASIA
+10-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

SOUTHERN AFRICA
0800-020111 (in South Africa, toll-free)
+27-10-209-8112 (International, call collect)

EUROPE
+39-6-4211-8685 (24-hour hotline)

UNITED KINGDOM
+44-7740-251-635

USA
+1-919-684-9111

The DES numbers (except UK) are generously supported by DAN

DAN ASIA-PACIFIC DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. All information is treated confidentially with regard to identifying details when utilised in reports on fatal and non-fatal cases. Such reports may be used by interested parties to increase diving safety through better awareness of critical factors. Information may be sent (in confidence unless otherwise agreed) to:

DAN Research
Divers Alert Network Asia Pacific
PO Box 384, Ashburton VIC 3147, Australia
Enquiries to: <research@danasiapacific.org>

DAN Asia-Pacific NON-FATAL DIVING INCIDENTS REPORTING (NFDIR)

NFDIR is an ongoing study of diving incidents, formerly known as the Diving Incident Monitoring Study (DIMS). An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report anonymously any incident occurring in your dive party. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver injury. Using this information to alter diver behaviour will make diving safer.

The NFDIR reporting form can be accessed on line at the DAN AP website:
<www.danasiapacific.org/main/accident/nfdir.php>

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