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Subclavian Doppler bubble monitoring

Australian snorkelling and diving fatalities 2012

Inner ear barotrauma – a tool for diagnosis

Which tooth restoration for divers?

HBOT for large bowel anastomosis problems

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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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DIVING AND HYPERBARIC MEDICINE

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Steve Goble admin@spums.org.au

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The Editor's offering

This year is the tenth anniversary of the joint publication of *Diving and Hyperbaric Medicine* (DHM) by SPUMS and EUBS. Both the societies and DHM have come a long way in that time, most especially this publication. Since 2008, DHM has achieved indexation on *Medline* and the *Web of Science*® (with a steadily climbing Impact Factor; see page 200), established an international Editorial Board with a wide diversity of expertise and moved with the times to become an electronic publication. This journey has not always been smooth and there remain unresolved issues between the Editor and EUBS, which hopefully can be settled soon. TRICON20018 offers a time and venue for the society executive committees to take stock of this progress and to plan for DHM's future.

Stop press news is that complete articles from DHM have now been accepted for deposit in PubMed Central (PMC), the most important medical scientific database in the public domain. This is retrospective to the March 2017 issue. PMC complies with our one-year embargo policy, so you will be able to access all articles from the March, June and September 2017 issues on PMC very soon. This markedly increases DHM's visibility to the international medical and scientific online readership and, enhancing the worth for SPUMS and EUBS members of submitting to your very own journal.

Inner ear injury from diving represents an uncommon but challenging problem of diagnosis (is it barotrauma (IEBt) or decompression sickness (DCS)?), treatment (is conservative care or surgery indicated?) and outcome (should patients return to diving?). In this issue, three medical officers from the US Navy present their ideas for a tool (for which they have allocated the acronym HOOYAH) to help with distinguishing between inner ear barotrauma and DCS.[2] It remains to be seen whether this is a useful tool in practice. A number of readily accessible articles have been published over the years in the *SPUMS Journal* and DHM on the inner ear in diving. For those interested in reviewing this topic in a painless fashion I have selected below a few articles that give a good feel for the subject and are readily available to members on the DHM website and in the GTÜeM database.[2–7] Carl Edmonds' series of 50 consecutive cases of IEBt[4] is probably the most useful clinical report, but note that his accompanying editorial[5] has a 'typo' in it in the left-hand column, 6th paragraph, line 2, which should read "*IEBt*", not "*MEBt*" (the Editor's error!). The article by Mitchell and Doolette on the likely mechanisms underlying inner ear DCS makes for fascinating reading,[6] whilst the case series by Wong and Walker addresses the difficulties of diagnosis and treatment of inner ear DCS.[7]

I first read Spencer and Johanson's report[8] on Doppler monitoring of divers in 1976 when I worked in Seattle. In the subsequent four decades, the role of Doppler in assessing decompression stress has remained a work in progress. The retrospective review[9] by scientists from Canada and France of the large Canadian Doppler database suggests that the value of subclavian monitoring has been somewhat overlooked in the past by researchers. Their results suggest that this should become a routine part of all Doppler studies in the future and that the guidelines[10] promulgated recently for the conduct of Doppler research in diving may now need revision.

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Michael Davis

Key words
Doppler; Editorials; Inner ear; Barotrauma; Medical database; General interest

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The front page photo is of technical diver Frederic Swierczynski of UnderWater eXperience monitoring his left subclavian vein with a Doppler IoT prototype (with permission).

Editorial

Decompression sickness, fatness and active hydrophobic spots

Since decompression sickness (DCS) in humans was first described,[1] mankind has embarked on an odyssey to prevent it. The demonstration that decompression releases bubbles, which mainly contain inert gas (nitrogen, helium),[2] into the circulation and that the slower the decompression rate the lesser the incidence of DCS, resulted in 1908 in the publication of the first, reasonably safe diving tables.[3]

Besides the development of proper diving tables, the selection of divers is also of importance. A relationship between body composition and DCS was observed in dogs as long ago as the nineteenth century,[2] an observation supported early in the twentieth century: "*Really fat men should never be allowed to work in compressed air, and plump men should be excluded from high pressure caissons…or in diving to more than about 10 fathoms, and at this depth the time of their exposure should be curtailed. If deep diving is to be undertaken…. skinny men should be selected.*"[4]

Alas, nothing is that simple! From my own experience it was not always the fat diver who ended up in the treatment chamber with DCS. Therefore, other factors must be at play; gender,[5,6] age,[5,7] physical fitness,[7] and the existence of a persistent foramen ovale (PFO)[8] have all been studied as possible factors for the development of vascular gas bubbles and, therefore, for DCS. However, none of these factors, alone or in combination, explain why there are intra-individual or intra-cohort differences in bubble grades (BG). In other words, why does a dive I did today led to a high BG but the same dive next week lead to a low one? Or, why is there such a difference in BG amongst divers of more or less the same age, gender, body composition and physical fitness? In a letter in this issue, a novel hypothesis is postulated that may fill in these gaps; active hydrophobic spots (AHS).[9]

These AHS can be found at the luminal side of capillary, venous and arterial walls and have an oligolamellar lining. In an *in vitro* experiment, nanobubbles developed on AHS after a 'dive' to 1,000 kPa (90 msw).[10] It appears that AHS consist of dipalmitoylphosphatidylcholine (DPPC), which is the main component of surfactant.[11] It is proposed that DPPC may leak from the alveoli into the alveolar capillary and be transported to veins and arteries where it precipitates and forms AHS.[11] Based on these ideas, it is hypothesized that AHS generate nanobubbles that can grow into microbubbles. When these microbubbles detach from the AHS they might also take along pieces of the AHS membrane making the AHS smaller or even disappear.[10] This phenomenon could explain some of the earlier findings regarding the formation of microbubbles in divers. The fact that the presence of microbubbles differs between younger and older divers, after repetitive dives, and between experienced divers and novice divers can be explained by this model,[10] and AHS may be the missing link we are looking for in our quest to understand and treat DCS.

However, some reservations must be made. Firstly, these observations are derived from *in vitro* and animal experiments and whether or not they reflect a similar process in man remains unclear. Secondly, it appears that female divers have lower bubble grades after similar dives compared to male divers, suggesting lower decompression stress.[5,6] If AHS is the main generator for microbubbles, there should be a difference in the presence of AHS between men and women. We do not know from these animal experiments whether there is a gender difference, neither does a literature search in PubMed provide us with an answer.

Thirdly, as said before, DPPC is the main component of surfactant. All alveolar surfactant phospholipids, such as DPPC, are secreted to the alveolar space via exocystosis of the lamellar bodies (LB) from alveolar type II (ATII) cells.[12] To form a functional air-blood barrier, alveolar type I and ATII cells are connected to each other by tight junctions. These tight junctions constitute the seal of the intercellular cleft and in that way form a true barrier between the alveolus and the capillary.[13] Only small molecules like oxygen, carbon dioxide, etc. can penetrate through this barrier by themselves due to passive diffusion. All other (macro) molecules, including DPPC, need intermediate processes such as ion transport proteins,[14] channels,[12] metabolic pumps,[14] etc. to gain access to the pulmonary capillary lumen. To my knowledge, no such mechanisms for DPPC or LB are known.

A theoretical explanation might be the fact that the production of DPPC and the exocytosis of DPPC-containing LBs into the alveolar space can be stimulated by stretch.[12,15] Stretch of the alveoli can switch on Ca^{2+} entry by either mechanosensitive channels, store-operated channels or second messenger-operated channels, which induces LB exocystosis.[12] Furthermore, an ATP-release mechanism might also be responsible for the pulmonary alveolar mechanotransduction of LB.[12] During diving, transpulmonary pressure changes[16] occur which might induce additional alveolar stretch and thus, theoretically, an extra release of LB. However, whether or not such exocystosis of LB is vascularly orientated remains unclear. Besides which, the leakage of DPPC from the alveolus to the pulmonary capillary might also be as simple as a malfunction of the tight junction due to epithelial membrane damage as a result of diving. Finally, it is also possible that DPPC is produced in other non-ATII cells in our body of which we are currently unaware.

To conclude, this is an interesting hypothesis regarding the origin of microbubbles. Whether or not DPPC and LB are the main reason for individual sensitivity to DCS remains

unclear. Further research will hopefully identify if DPPC and LB are indeed the missing link or just another branch on the big tree of the genesis of decompression sickness.

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Pieter Jan AM van Ooij[1,2]

[1] *Diving Medical Centre, Royal Netherlands Navy, Den Helder, The Netherlands*

[2] *Department of Respiratory Medicine, Academic Medical Centre, University of Amsterdam*

Address for correspondence: *Diving Medical Centre, Royal Netherlands Navy, PO Box 10.000, 1780 CA Den Helder, The Netherlands*

Pjam.v.ooij.01@mindef.nl

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Original articles

Reliability of venous gas embolism detection in the subclavian area for decompression stress assessment following scuba diving

Julien Hugon[1], Asya Metelkina[1], Axel Barbaud[1], Ron Nishi[2], Fethi Bouak[3], Jean-Eric Blatteau[4], Emmanuel Gempp[5]

[1] *Azothe Systems – Technopôle de la Mer, Ollioules, France*
[2] *Defence Research and Development Canada, Toronto Research Centre (retired), Toronto, ON, Canada*
[3] *Defence Research and Development Canada, Toronto Research Centre, Toronto, ON, Canada*
[4] *Institut de Recherche Biomédicale des Armées, Équipe de Recherche Subaquatique Opérationnelle, Toulon, France*
[5] *French Navy Diving School, Toulon, France*
Corresponding author: *Julien Hugon, Azoth Systems – Technopôle de la Mer, 93 Forum de la Méditerranée, 83190 Ollioules, France*
julien.hugon@azoth-systems.fr

Key words
Bubbles; Cardiovascular; Doppler; Air; Statistics; Decompression sickness; Risk

Abstract
(Hugon J, Metelkina A, Barbaud A, Nishi R, Bouak F, Blatteau J-E, Gempp E. Reliability of venous gas embolism detection in the subclavian area for decompression stress assessment following scuba diving. Diving and Hyperbaric Medicine. 2018 September;48(3):132–140. doi: 10.28920/dhm48.3.132-140. PMID: 30199887.)
Introduction: Ultrasonic detection of venous gas emboli (VGE) in the precordial (PRE) region is commonly used in evaluation of decompression stress. While subclavian (SC) VGE detection can also be used to augment and improve the evaluation, no study has rigorously compared VGE grades from both sites as decompression stress indicators.
Methods: This retrospective study examined 1,016 man-dives breathing air extracted from the Defence Research and Development Canada dataset. Data for each man-dive included dive parameters (depth, bottom time, total ascent time), PRE and SC VGE grades (Kisman-Masurel) and post-dive decompression sickness (DCS) status. Correlation between SC and PRE grades was analyzed and the association of the probability of DCS (pDCS) with dive parameters and high bubble grades (HBG III- to IV) was modelled by logistic regression for SC and PRE separately for DCS risk ratio comparisons.
Results: PRE and SC VGE grades were substantially correlated (R = 0.66) and were not statistically different ($P = 0.61$). For both sites, pDCS increased with increasing VGE grade. When adjusted for dive parameters, the DCS risk was significantly associated with HBG for both PRE ($P = 0.03$) and SC ($P < 0.001$) but the DCS risk ratio for SC HBG (RR = 6.0, 95% CI [2.7–12.3]) was significantly higher than for PRE HBG (RR = 2.6, 95% CI [1.1–6.0]).
Conclusions: The association of bubble grades with DCS occurrence is stronger for SC than PRE when exposure severity is taken into account. The usefulness of SC VGE in decompression stress evaluation has been underestimated in the past.

Introduction

To date, there have been two common ways to assess decompression-induced physiological stress for dive exposures and associated decompression procedures. The first one is the US Navy approach, which relies on statistical predictive tools calibrated with diving profile/decompression sickness (DCS) databases.[1–8] This probabilistic approach allows the construction of a DCS risk model based on gas kinetics and associated ascent criteria, linking a decompression model output to a risk. It also offers an interesting calibration possibility of the parameters for a global decompression model. The second approach is based on the detection of bubbles after diving using either Doppler ultrasound or ultrasonic echocardiographic imaging.

It is well known that the bubbles formed in the various parts of the body during a decompression can be pathogenic and may generate several forms of DCS. Even if there is no clear evidence of a causal relationship between the amount of bubbles circulating in the blood stream and DCS, numerous Doppler and ultrasonic imaging studies support the association between venous gas emboli (VGE) levels and DCS risk.[9–20] VGE is considered a relatively poor predictor of DCS (low specificity), but the absence of VGE is a good indicator of decompression safety (high sensitivity).[21,22] This is why the amount of VGE detected is believed to be a useful decompression stress indicator for comparing various decompression procedures or controlling the efficiency of a decompression procedure.[23–25] For example, the Defence and Civil Institute of Environmental Medicine (DCIEM, now Defence Research and Development Canada,

DRDC – Toronto Research Centre) has used the Doppler ultrasound method to detect VGE in order to develop various decompression tables for the Royal Canadian Navy.[26–29] The potential of bubble detection to assess the relevance of decompression procedures may have been clearly pointed out using modern statistical approaches.[22,31] This offers interesting possibilities and makes feasible – in terms of cost, time, statistical relevance and health impairment control – the validation of decompression profiles to reach a given DCS risk target.

While both statistical tools and bubble detection have proven to be useful, they remain characterized by different limitations: the probabilistic approach is an *a priori* method that does not consider inter/intra individual variability with respect to DCS susceptibility while the bubble detection approach is an *a posteriori* method that does not consider pressure profile/decompression profile to assess DCS risk. However, it is well known that both VGE formation and DCS occurrence depend primarily upon the dive exposure (depth, duration, gas breathed), the decompression procedure (ascent rate, decompression stops, oxygen during stops) and potentially upon physical characteristics of the diver (age, body mass index BMI). Nevertheless, an in-depth analysis of a large dataset using a logistic regression method showed that the association between large VGE loads and the increase in probability of DCS persists after taking into account the dive parameters, such as the depth, the bottom time and the decompression time, and the individual covariates such as age and BMI.[32]

Even with recent advances in imaging technology and image quality of 2-dimensional echocardiography, Doppler ultrasound is generally considered the most popular method in field studies due to its portability and low cost.[33] The Doppler bubble signal (in the audible frequency range) is graded using either the Kisman-Masurel (KM)[34] or Spencer[9] grading systems, with grade zero for the absence of detectable bubbles and grade four for a continuous flow of bubbles.[23]

VGE detection in the precordium is considered as the gold standard in Doppler ultrasound as it takes into account bubbles from the whole body, while additional subclavian detection has been recommended to improve the sensitivity of bubble detection.[35] Nevertheless, no large study has rigorously compared data from the precordial and subclavian sites, even if some data[17] contained cases of DCS symptoms in the upper part of the body with bubbles detected in the shoulders only and not in the chest. These data motivated our study, suggesting a more in-depth examination of the sensitivity of precordial versus subclavian bubble grades in evaluation of decompression safety. It is worth mentioning that some studies have suggested that the subclavian region, as opposed to the precordium, shows more potential for automated bubble detection due to its low noise signal.[36,37]

Our retrospective analysis compared the Doppler bubble grades from precordial and subclavian regions after a wide range of dive exposures in a dataset drawn from a large prospective cohort of divers followed by DRDC. In this paper, we aimed to confirm the usefulness of Doppler VGE grades in evaluating decompression stress in air dives drawn from this DRDC dataset. For each measurement site, we examined the association between high bubble grades and the probability of DCS taking into account the dive parameters (i.e., maximum depth, bottom time, total ascent time). This analysis was intended to compare the strength of this association for subclavian versus precordial VGE grades.

Methods

DATABASE

This study forms a retrospective analysis of a subset of the DRDC database from a number of studies conducted by DRDC over a period of about 40 years. These studies were carried out to develop and validate decompression tables and diving procedures currently used in the Canadian Armed Forces. All dives in the database were approved by the DCIEM/DRDC Human Research Ethics Committee and were done in accordance with the Helsinki Declaration. Dive participants were primarily military divers, but also included civilian divers. Over 7,000 man dives have been monitored and are included in the DCIEM/DRDC Doppler ultrasound database.

The dive subjects were monitored with continuous wave Doppler ultrasonic bubble detectors (from 1979 to 1987 – "*DUG*", Soledec S.A., Marseille, France, and from 1987 to 2013 – TSI DBM 9008, Techno Scientific Inc., Concord, ON, Canada), first at the precordium with the diver standing at rest and after movement (deep knee bend), and then at the left and right subclavian veins at rest and after a specific movement (fist clench).[29] The Doppler signals were graded using the KM code[34] where bubbles are classified on a scale from 0 to 4 based on three parameters: 1) the number of bubble signals per cardiac cycle, 2) the percentage of cardiac cycles in which bubbles are observed during the rest condition, or the number of successive cardiac cycles containing bubbles starting from the initial increase in blood flow after movement, and 3) the amplitude of the bubble signal relative to the normal background cardiac sounds. The resulting 3-digit codes are then converted to bubble grades from 0 to IV, similar to the 5-point (0 to 4) Spencer bubble grades, but with finer steps based on a 12-point scale (i.e., 0, I-, I, I+, II- .., -IV, IV).

To detect the maximum bubble activity, each dive subject was monitored several times over a given period after the dive. Typically, bubble monitoring was carried out at least three times in about two hours – the first at 20 minutes (min) after surfacing and then at 40 min intervals. If bubbles were

Table 1

Dive parameters and venous gas embolism (VGE) scores for all man-dives analysed (column 2), decompression sickness (DCS) dives (column 3) and no-DCS dives (column 4); all continuous and ordinal variables are presented as median [range]; N.B. VGE scores from III- to IV were considered high bubble grade (HBG) and encoded HBG = 1; *n* (%) of the dives with a high bubble grade detected; PRE – precordial; SC – subclavian; msw – metres' sea water

		Total included	**DCS**	**no-DCS**
Man dives (*n)*		1,016	22	994
Maximum depth *P* (msw)		44.2 [9–79.4]	45 [18–69.1]	42.4 [9–79.4]
Bottom time *t* (min)		30 [2.6–120]	30 [6.8–120]	30 [2.6–120]
Decompression duration *tat* (min)		16.2 [0.9–89.5]	55.8 [2.5 –84.6]	14.3 [0.9–85.9]
Exposure index ($Q = P\sqrt{t}$)[38]		189 [67–296]	247 [174 –285]	186 [66–295]
PRE grades		0 [0–IV]	II+ [0–IV]	0 [0–IV]
PRE HBG = 1, *n* (%)		141 (14 %)	10 (45 %)	131 (13%)
SC grades		0 [0–IV]	III– [0–IV]	0 [0–IV]
SC HBG = 1, *n* (%)		149 (15 %)	14 (63 %)	135 (14%)
PRE bubble grades	0	634 (62.4%)	2 (9.1%)	632 (63.6%)
	I-, I, I+	123 (12.1%)	3 (13.6%)	120 (12.1%)
	II-, II, II+	118 (11.6%)	7 (31.8%)	111 (11.2%)
	III-, III, III+	138 (13.3%)	9 (40.9%)	129 (13.0%)
	IV-, IV	3 (0.3%)	1 (4.5%)	2 (0.2%)
SC bubbles grades	0	616 (60.6%)	4 (18.1%)	612 (61.6%)
	I-, I, I+	154 (15.2%)	2 (9.1%)	152 (15.3%)
	II-, II, II+	97 (9.5%)	2 (9.1%)	95 (9.6%)
	III-, III, III+	136 (13.4%)	11 (50.0%)	125 (12.6%)
	IV-, IV	13 (1.3%)	3 (13.6%)	10 (1.0%)

still present at grade III or a higher level, monitoring was continued until there was a clear indication that the bubble levels were dropping. Although there were many cases where high bubble levels were observed, recompression treatment was never initiated based on bubble grades. Treatment was always based only on DCS symptoms. However, subjects with high bubble grades were kept under observation.

We examined the data from a subset of 1,041 man dives conducted on air up to 2013 extracted from the DRDC database. Repetitive dives were excluded. Each dive record contained several post-dive Doppler-detected bubble grades at rest from both precordial (PRE) and subclavian (SC) regions (both right and left); the DCS status of the diver after exposure (Type I – musculoskeletal pain; Type II – neurologic, cutaneous, marginal, no DCS), an anonymous diver identification number and the following dive parameters: maximum depth *P* in metres' sea water (msw); bottom time *t* (minutes, min); and decompression duration (total ascent time) *tat* (min).

In an earlier study that included some of these data, it was concluded that the maximum bubble grade for all conditions, rest and movement, and all sites, precordial and subclavian, showed the strongest association of bubble grades with the risk of DCS.[17] There was a considerable reduction in sensitivity in detecting VGE if only the precordial site was monitored, 47% versus 60% for all-sites monitoring.[25]

In this study, only precordial and subclavian bubble grades for VGE detected at rest were considered for analysis and bubble grades after movement were excluded. Any records with missing precordial and/or subclavian bubble grades were excluded from the analysis. Based on these rejection criteria, from 1,041 records in our dataset, 25 man-dives (including seven cases of DCS) were excluded. As a result, 1,016 man-dives (including 22 DCS cases) were analyzed. When several bubble grades were available from a given site (precordial or subclavian), only the highest bubble grade was used in the analysis. Bubble grades from III- to IV were considered high bubble grade (HBG) and encoded HBG = 1, and bubble grades from 0 to II+ were considered low bubble grades and encoded HBG = 0.

A total of 236 divers in our study completed 84 square dive profiles covering a wide range of exposures. For each dive, we computed Hempleman's stress index.[38]

$$Q = P\sqrt{t}\ (msw \cdot min^{0.5}) \quad (1)$$

This index does not incorporate any decompression information and a theoretical analysis has shown its limitations for dives requiring decompression.[39] As the dives analyzed all require decompression, we use Q as an exposure index, i.e., a measure of the severity of the exposure. It should be noted that it has been used in the past as an exposure index to limit commercial diving in the North Sea based on studies done between 1982 and 1988.[40] The main characteristics of the dive records used in our analysis are given in Table 1. Within the 22 DCS cases in Table 1, there were 15 Type I, four Type II, 1 cutaneous and two marginal cases. To increase the statistical power, we grouped all DCS types together in a dichotomized DCS variable with DCS = 1 representing all types of DCS events including marginal and cutaneous.

STATISTICAL ANALYSIS

VGE grades were treated as ordinal categorical data for statistical analysis. PRE and SC bubble grades for each diving exposure were considered as paired measures and the strength of correlation between PRE and SC grades was evaluated using polychoric correlation coefficient R,[41] similar to Pearson's correlation and appropriate for comparison of two ordinal measures (i.e., VGE grades) of unobserved continuous variable (i.e., bubble flow). In addition, we computed Spearman's correlation coefficient to confirm the polychoric correlation results. The relationship between measures was interpreted as distinct if $0.71 < R < 0.89$, substantial if $0.41 < R < 0.70$, and small if $0.21 < R < 0.40$. The correlation was significant if 95% bootstrap or normal confidence bounds did not contain zero. We tested for a systematic superiority of PRE or SC VGE grades with respect to each other using the Wilcoxon signed rank test[42] with a probability value (p) < 0.05, indicating a significant difference between two measures.

We examined the association between HBG and DCS incidence for different exposures by organizing dives into four severity groups based upon the exposure index Q: (1) low ($0 < Q < 150$); (2) low intermediate ($150 < Q < 200$); (3) high intermediate ($200 < Q < 250$); and (4) high ($Q > 250$). For each severity group, the DCS incidence for HBG = 1 versus HBG = 0 was compared and the associated DCS risk ratios were tested for statistical significance for both precordial and subclavian detections.

We used logistic regression to test the association of pDCS with VGE grades adjusted to dive parameters:

$$pDCS = p(DCS|x,HBG) = 1/[1+\exp(-a_0 - a\ x - b\ HBG)] \quad (2)$$

where x is a function of the dive parameters while a0, a, and b are logistic regression parameters. We first considered P, t, and tat as variables to lead the analysis, then we considered natural cubic splines transformations for adjustment for nonlinear effects of the dive parameters and compared the results. The adjusted odds ratios (OR HBG = 1 vs. HBG = 0) for PRE and SC and per 10-point increase in Q were computed from the adjusted logistic regression (2) with the Wald test for significance of covariates. Model comparison was done using the Akaike information criterion (AIC), which estimates the relative quality of statistical models for a given set of data, with smaller values of the criterion suggesting a better fit to the data. The reported risk ratios (RR HBG = 1 vs. HBG = 0) were computed from the odds ratios by using the following formula:[42]

$$RR_{HBG=1\ vs\ HBG=0} = OR_{HBG=1\ vs.\ HBG=0} / (1_{pDCS} + pDCS * OR_{HBG=1\ vs.\ HBG=0}) \quad (3)$$

where pDCS = 0.014 vs. pDCS = 0.009 is the DCS incidence in the database in reference groups PRE HBG = 0 vs. SC HBG = 0. All tests were two-tailed and $p < 0.05$ was considered statistically significant.

Results

AGREEMENT BETWEEN DOPPLER MEASURES

The maximum values of VGE grade at SC were not statistically different compared to the maximum values of VGE grade at PRE (Wilcoxon signed rank test: W = 45864, probability = 0.61). The polychoric correlation coefficient for VGE from both sites R = 0.66 (95% bootstrap CI [0.57–0.69]) was significant suggesting a substantial relationship between the detections. Spearman's correlation coefficient rs = 0.53 (95% normal CI [0.47–0.69]) was also significant with the same strength of relationship.

VGE AND RISK OF DCS

Table 2 shows that with an adjustment for Q in the logistic regression, both PRE HBG and SC HBG were statistically associated with DCS risk. This association was also significant when RR were adjusted directly for the dive parameters P, t, tat (linearly) with comparable RR values and model fit as measured by AIC, which suggests the usefulness of Q in assessng the impact of bubble production on diving stress (pDCS). The adjusted risk ratios were significantly higher for SC HBG compared to PRE HBG. The model fit was better when using SC HBG (AIC = 180 for SC vs. AIC = 193 for PRE with an adjustment for Q). The logistic regression with cubic splines in P, t and tat gave similar results in terms of fit and risk ratios.

Tables 3 and 4 present the contingency tables of HBG and DCS by exposure index (Q), for PRE and SC respectively. For both sites, Q was statistically associated with DCS risk (probability = 0.04 for PRE and 0.004 for SC), as

Table 2
The adjusted decompression sickness (DCS) odds (OR) and risk (RR) ratios from logistic regressions for precordial vs. subclavian bubble counts with 95% confidence intervals, [95% CIs]; the probability values are from Wald's test for significance of HBG = 1; the information criterion AIC is used in model comparison; reference groups for HBG are PRE HBG = 0 and SC HBG = 0 respectively; PRE – precordial; SC – subclavian; Q – exposure index; *P*– maximum depth (msw); *t* – bottom time (min); *tat* – decompression duration (min) (see text for explanation)

Adjustment	Covariate	OR [95% CI]	RR [95% CI]	p-value	AIC
Q	PRE HBG = 1	2.7 [1.1–6.7]	2.6 [1.1–6.2]	0.03	193
	SC HBG = 1	6.8 [2.8–17.6]	6.5 [2.8–15.2]	< 0.0001	180
P, t, tat	PRE HBG = 1	2.9 [1.2–7.4]	2.9 [1.1–6.9]	0.02	194
	SC HBG = 1	7.1 [2.9–18.4]	6.8 [2.9–16.2]	< 0.0001	181

Table 3
Decompression sickness (DCS) number, incidence and relative risk ratio for PRE HBG by exposure group (see text for explanation)

$Q = P\sqrt{t}$	DCS/no DCS	DCS (%)	DCS/no DCS	DCS (%)	RR	p-value
	PRE HBG = 1		PRE HBG = 0			
[0–150]	0/4	0	0/353	0	NA	
[150–200]	2/16	12.5	2/181	1.1	11.4	0.04
[200–250]	3/54	5.6	7/226	3.1	1.8	0.40
[250–300]	5/57	8.8	3/103	2.9	3.0	0.14

Table 4
Decompression sickness (DCS) number, incidence and relative risk for SC HBG by exposure group (see text for explanation)

$Q = P\sqrt{t}$	DCS/no DCS	DCS (%)	DCS/no DCS	DCS (%)	RR	p-value
	SC HBG = 1		SC HBG = 0			
[–150]	0/6	0	0/351	0	NA	-
[150–200]	3/28	10.7	1/169	0.6	17.8	0.01
[200–250]	6/56	10.7	4/224	1.8	6.0	0.007
[250–300]	5/45	11.1	3/115	2.6	4.3	0.05

Table 5
The adjusted decompression sickness (DCS) risk ratios from logistic regressions for precordial (PRE) vs. subclavian (SC) measurements; reference groups for HBG are PRE HBG = 0 and SC HBG = 0 respectively; for the exposure index Q, the adjusted RR are given per 10-point increase (see text for explanation)

Covariate	RR [95% CI]	p-value	AIC
***Q*, PRE**	1.18 [1.07–1.32]	0.04	193
***Q*, SC**	1.18 [1.06–1.33]	0.004	180

shown in Table 5. After an adjustment for HBG, RR was approximately 1.2 (95% CI [1.1–1.3]) per 10-point increase in *Q* for both sites.

Discussion

The relevance of bubble detection to assess decompression stress is routinely accepted. However, its use to characterize DCS risk is a controversial topic. This controversy could come from the fact that the severity of exposures has never been considered as a major discriminating factor in relating DCS to VGE. This study considers both exposure severity ranges and bubble grades to assess DCS risk. Although the exposure index used, *Q*, is based only on the depth of the dive and the time spent at that depth, i.e., the gas loading, and does not include any decompression information, there is an apparent correlation between the risk of DCS and increasing *Q*. This is a result of practical decompression tables based on supersaturation having an increasing risk of DCS as the exposure increases.

Analytical studies have shown that the risk increases considerably with longer bottom times and with increasing depth, although not nearly as much as with bottom time.[44,45] A survey of commercial air diving in the UK sector of the North Sea clearly showed that the severity of the exposure significantly increased the risk of DCS,[46] prompting the use of *Q* as a convenient means of limiting diving activities for safety.[40] It should be noted that since different decompression tables may have different risks of DCS, the relationship between *Q* and pDCS may differ between tables depending on the nature of the decompression profile and the decompression time. For this study, the majority of dives analyzed used air decompression tables developed by DCIEM.[26–29] Although *Q* is not intended to represent the 'quality' of the decompression as a stress index, nevertheless, by taking into account both depth and bottom time, it provides a valuable means for relating exposure to DCS risk.

We examined the Doppler VGE grades from the chest and shoulders and the DCS data from a large DRDC dataset of air dives. Our results seem to confirm the observations that without subclavian bubble detection, a number of DCS cases would not have been associated with bubbles based on precordial monitoring alone.[17] Thus it was important to monitor both sites. Overall, no site provided systematically higher bubble grades and both PRE and SC bubble grades were in substantial agreement. However, after taking into account the severity of exposure with the *Q* index, there was a minimum six-fold increase in the probability of DCS for high subclavian bubble grades compared to an approximately three-fold increase for high precordial bubble grades (Table 2). It was also noticeable that high SC bubble grades were particularly associated with a significant DCS risk ratio when compared to low SC bubble grades for a large spectrum of dives in terms of the severity of exposure. This was less pronounced for PRE bubble grades (see Table 3 vs. Table 4).

Until now, subclavian detection has been used as supplementary or complementary information to precordial detection to assess the physiological stress induced by decompression.[47,48] This study is the first to quantify comparison of subclavian and precordial bubble grades and suggests that the usefulness of subclavian Doppler detection in evaluating decompression stress has been underestimated in past studies. This result could be seen as unexpected as the subclavian sites can only reveal bubbles produced in the upper part of the body, while the precordial site reflects that of the whole body. However, bubbles in the precordial region can be masked by the heart (background) noises, and audio artefacts from the valves of the heart may mislead the operator and cause them to register false positive grades. This is not the case for bubbles flowing in subclavian veins where the background noises are minimal and bubble signals are relatively unambiguous.

Our study has some limitations. Firstly, we did not consider the different types of DCS or symptoms but grouped them all together, including marginal and cutaneous events. If a larger number of marginal and cutaneous events were available, they might be better treated independently.

Secondly, as the data analysed were collected over a long period (from 1979 to 2013) by several different raters, there may be some inter-rater variability in bubble detection and grading of the many divers included in the study. Assessment of the inter-rater agreement on grading bubbles, demonstrated that DRDC had effective, practical techniques to ensure comparable grades when Doppler data from several raters were combined.[49] For example, each rater was evaluated prior to any new study, and the raters often worked in pairs. In cases where there were doubts about grading difficult bubble signals, the two scorers (often including DRDC's senior Doppler rater in practically all the cases) would review these signals and reach a consensus on the correct grade. New raters would work with all the other raters and grade a number of previously graded signals until a high degree of comparability with the other raters was reached.

Thirdly, only the results for precordial and subclavian VGE for the resting condition (a steady state condition) were looked at; taking into account the movement condition that results in a transient increase in VGE levels was not considered.

Modern decompression models and algorithms developed for decompression tables or for implementation in diving computers can certainly help to reduce the risk of DCS. Nevertheless, there is a high inter- and intra-individual variability of risk and no guidelines have been provided for individualized choice of diving practices, for example, when more or less conservative procedures should be selected.

To improve diving practices and reduce DCS risks, divers need an objective measure of individual decompression

stress. VGE grades could be used to provide such a measure if an individual's history of VGE is known. In a study on no-decompression limits for compressed air, graphic methods were used to develop isopleths of equal occurrence of VGE and DCS pain and it was recommended that DCS and VGE-prone divers should dive only in shallow waters and should be rejected as candidates for occupational diving.[50] In a series of decompression-required dives tested at DCIEM, it was found that a depth-bottom time limiting line could be established beyond which high VGE grades and DCS would result, leading to the conclusion that 'high bubblers' should avoid diving at or near the limiting line and that only 'low bubblers' should dive above the limiting line.[51] More recently, it has been suggested that a modification of some of the diving practices of divers producing high VGE grades could potentially decrease their DCS risks since it was observed that divers having a history of mild DCS were more prone to VGE formation than divers without a DCS history.[52] Another suggestion made is that with a good method of interpreting VGE data, there is a possibility of long-term monitoring of an individual's susceptibility to DCS to derive individualized decompression schedules to reduce the risks of DCS.[53]

Conclusions

In general, low bubble grades are associated with lower risks of DCS. When exposure severity is taken into account, this association was stronger for bubble grades from the subclavian sites than from the precordial site. For high bubble grades, the probability of DCS with high subclavian bubble grades was much greater than that for high precordial bubble grades. These findings suggest that the usefulness of subclavian VGE detection using Doppler ultrasound in the development of safer diving has always been underestimated in the past. Doppler VGE detection could be a valuable tool in the improvement of diving practices through the reduction of bubble grades.

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Provisional report on diving-related fatalities in Australian waters in 2012

John Lippmann[1,2], Chris Lawrence[3], Andrew Fock[4], Scott Jamieson[1]

[1] *Divers Alert Network Asia-Pacific, Ashburton, Victoria, Australia*
[2] *School of Health and Social Development, Deakin University, Victoria, Australia*
[3] *Statewide Forensic Medical Services, Royal Hobart Hospital, Tasmania, Australia*
[4] *Departments of Hyperbaric Medicine and Intensive Care Medicine, The Alfred Hospital, Melbourne, Victoria, Australia*
Corresponding author: *John Lippmann, DAN Asia Pacific, P O Box 384, Ashburton, Victoria 3147, Australia*
johnl@danap.org

Key words
Diving deaths; Scuba; Breath-hold diving; Surface-supply breathing apparatus (SSBA); Diving incidents; Case reports

Abstract
(Lippmann J, Lawrence CL, Fock A, Jamieson S. Provisional report on diving-related fatalities in Australian waters 2012. Diving and Hyperbaric Medicine. 2018 September;48(3):141–167. doi: 10.28920/dhm48.3.141-167. PMID: 30199888.)
Introduction: An individual case review of known diving-related deaths that occurred in Australia in 2012 was conducted.
Method: The case studies were compiled using statements from witnesses and reports of the police and coroners. In each case, the particular circumstances of the accident and details from the post-mortem examination, where available, are provided.
Results: There were 26 reported fatalities (four less than the previous year). Only two of the victims were female (one snorkeller and one scuba diver). Fourteen deaths occurred while snorkelling and/or breath-hold diving, 11 while scuba diving and one diver died while using surface supplied breathing apparatus in a commercial pearl diving setting. Two breath-hold divers likely drowned as a result of apnoeic hypoxia. Cardiac-related issues were thought to have contributed to the deaths of at least three and possibly seven snorkellers and four scuba divers.
Conclusions: Pre-existing medical conditions; poor organisation, planning and supervision; equipment-related problems; snorkelling or diving alone or with loose buddy oversight and apnoeic hypoxia were features in several deaths in this series.

Introduction

Each year deaths occur during scuba diving and snorkelling. Analysis of diving-related fatalities indicates that many might have been avoided through appropriate countermeasures. The aims of the DAN Dive Fatality Reporting Project are to:

- educate divers and the diving industry about good, safe diving and snorkelling practices;
- inform physicians on the causes of fatal dive accidents in the hope of reducing the incidence of similar accidents in the future and of detecting, in advance, those who may be at risk.

This report includes the diving-related fatalities between 01 January and 31 December 2012 that are recorded on the DAN Asia-Pacific (AP) database. When an accident is unwitnessed, it is difficult to determine accurately what had occurred. We have sometimes included considered speculation within the comments to provoke thought about the possible sequence of events. A summary of the possible sequence of events in each of these incidents is shown in Table 3 as part of the discussion.

Methods

Ethics approvals were received from the Victorian Department of Justice Human Research Ethics Committee (to access data from the Australian National Coronial Information System (NCIS)); the Royal Prince Alfred Hospital Human Research Ethics Committee; the Coronial Ethics Committee of the Coroner's Court of Western Australia; and the Queensland Office of the State Coroner. The methodology used for this report was identical to that described previously for the 2004 Australian diving-related fatalities.[1]

Breath-hold and snorkelling fatalities (Table 1)

There were 14 snorkelling deaths recorded, the majority (nine) occurring in the State of Queensland on the Great Barrier Reef (GBR) or associated islands.

BH 12/01

This 64-year-old (y.o.) male was an overseas tourist who was on a day trip to an island on the GBR. He had a history of stage 1 myeloma for which he was being monitored with six-monthly blood tests but not yet receiving treatment. He was taking diclofenac and omeprazole. He was reported to have been a capable swimmer who had dived and snorkelled several times before.

He was using his own mask and snorkel and hired a lycra suit. Although he was wearing bootees, it was uncertain if he had been wearing fins. The weather was sunny with a light wind and the water was calm and clear. He went to an

Table 1

Summary of snorkelling and breath-hold diving-related fatalities in Australian waters in 2012; BMI – body mass index; BNS – buddy not separated; BSB – buddy separated before incident; BSD – buddy separated during the incident; GNS – group not separated; GSB – group separated before incident; IPE – immersion pulmonary oedema; n/s – not stated

BH	Age (y)	Sex	Height (cm)	Weight (kg)	BMI ($kg \cdot m^{-2}$)	Training	Experience	Dive group	Dive purpose	Depth (msw)	Incident (msw)	Weights On	Weights kg	Disabling injury
12/01	64	M	178	74	23	n/s	Yes	Solo	Recreation	n/s	Surface	n/s	-	Asphyxia? IPE?
12/02	82	M	172	82	28	n/s	n/s	Solo? GSB?	Recreation	n/s	Surface	n/s	-	Cardiac?
12/03	37	M	175	79	26	n/s	n/s	GNS	Recreation	n/s	Surface	n/s	-	Asphyxia?
12/04	62	M	173	85	28	n/s	Yes	GSB	Recreation	n/s	Surface	n/s	-	Cardiac
12/05	68	M	175	101	33	n/s	n/s	GNS	Recreation	n/s	Surface	n/s	-	Cardiac
12/06	26	M	185	60	18	Yes	Yes	GSB	Spearfishing	10–20	n/s	n/s	-	Asphyxia
12/07	70	F	158	64	26	n/s	n/s	BSB	Recreation	n/s	Surface	n/s	-	Asphyxia
12/08	71	M	180			n/s	Yes	BSD	Recreation	n/s	Surface	n/s	-	Unknown
12/09	31	M	190	79	22	Yes	Yes	Solo	Spearfishing	20–25	During ascent	Yes	4	Asphyxia
12/10	64	M	175	104	34	n/s	n/s	Solo	Recreation	n/s	Surface	n/s	-	Asphyxia? cardiac?
12/11	45	M	178	88	27	n/s	nil	GSB	Recreation	n/s	Surface	n/s	-	Asphyxia
12/12	68	M	176	75	24	n/s	Yes	Solo	Recreation	n/s	Surface	n/s	-	Cardiac
12/13	27	M	174	74	24	Yes	Yes	GSB	Spearfishing	15	n/s	Yes	9.5	Asphyxia
12/14	38	M	170	70	24	n/s	n/s	Solo	Hunting abalone	0.5–4	Surface	rescuer off	12	Asphyxia

unsupervised area and snorkelled there alone. Approximately 45 minutes (min) later, he was seen to be floating motionless with his snorkel submerged and only his shoulder blades visible. A lifesaver and an assistant dragged him onto a paddle board, and took him to a nearby vessel. Basic life support (BLS) was commenced by another lifesaver approximately 10 min after he was first seen. After another 5–10 min an AED was attached and repeatedly indicated that no shock was advised. Supplementary oxygen was provided (method unreported). The lifeguard unsuccessfully attempted to insert a supraglottic airway (i-gel). Two doctors (tourists) became involved in resuscitation efforts and implemented advanced life support (ALS). The victim failed to respond and resuscitation was ceased after an hour. One doctor reported that the victim was very cold when she initially assessed him and estimated that he might have been dead for possibly 30 min when found.

Autopsy: (1 day post mortem). The heart weighed 408 g (normal range (n.r.) 295–445 g). There was some concentric left ventricular thickening, redundancy of the mitral valve and 50% narrowing of the coronary arteries. There was frothy fluid in the upper airways, the right and left lungs weighing 926 g (n.r. 410–892 g) and 884 g (n.r. 378–780g), respectively and oedematous. Myeloma was detected in the bone marrow. The cause of death was given as drowning but the possibility of a precursor event related to heart disease or myeloma could not be excluded.

Comments: What caused the victim to become unconscious is unknown. He had mild left ventricular hypertrophy, mild mitral incompetence and a 50% stenosis of the coronary arteries. Generally none of these factors in isolation would be severe enough to cause loss of consciousness. His decision to snorkel alone in an unpatrolled area likely led to a substantial delay in the recognition of a problem and the subsequent rescue. Despite prompt and appropriate post-rescue BLS and ALS, death was likely inevitable because of this delay. An alternate interpretation of the fluid-laden lungs at autopsy could be immersion pulmonary oedema (IPE).

Summary: 64 y.o. male; history of stage 1 myeloma and back pain; capable swimmer; had snorkelled several times before and earlier that day; snorkelling alone; found unconscious in water after probably long delay; AED attached (long delay) but no shock advised; drowning

BH 12/2

This 82 y.o. man was an overseas tourist who was on a day trip to the GBR on a large tourist vessel. He had a history of hypertension, hypercholesterolaemia, stroke and coronary atherosclerosis. His wife reported that he had "*heart surgery twice … with five stents inserted over the past five years*". His prescribed medications included fexofenadine, lisinopril, ezetimibe, simvastatin and sildenafil. He was reported to have been a strong swimmer but his snorkelling experience is unknown. The pre-dive briefing included warnings about the risks of snorkelling with certain medical conditions, including cardiac conditions. Participants were asked to declare any medical conditions but he failed to do so. He had slept well, was in a good mood and appeared confident about snorkelling on the reef.

On arrival at the pontoon, he took a mask and snorkel (it is unclear if he wore fins). He was advised to take a stinger suit and buoyancy aid, such as a lifejacket or 'noodle'. However, he refused. One witness reported that he sounded drunk. The weather was clear with a light wind, the water was calm with a low swell and the current was variously reported to be slight (by staff) to strong (by passengers). The victim entered the water wearing a t-shirt and shorts and his snorkelling gear and was initially observed to be swimming well. However, he soon appeared to be distressed and then motionless, face-down in the water with the end of his snorkel submerged. Another snorkeller went to his aid, found him to be unconscious, and alerted a lifeguard. His mask and snorkel were still in place. With the aid of another snorkeller, he lifted the victim's head out of the water and noted white frothy sputum oozing from his mouth. He was taken by tender to the pontoon, where BLS was commenced by lifeguards and continued for 40 min. It is likely that a defibrillator was attached at some point as the presence of electrode pads were noted at autopsy. However, there were no details about when this occurred and what was indicated.

Autopsy: (1 day post mortem) Examination of the brain revealed an old infarct in the left temporal lobe. X-ray of the chest showed no significant pathology. The heart was heavy, 565 g (n.r. 331–469 g). The coronary arteries showed 80% calcific stenosis of the right and circumflex coronary arteries and near occlusion of a stent in the left coronary artery. There was biventricular hypertrophy. Histological evidence was seen of scarring but not of acute ischaemia. The upper airways contained gastric contents. The right and left lungs weighed 860 g (n.r. 446–880 g) and 782 g (n.r. 348–790 g), respectively, and were oedematous. The cause of death was given as drowning secondary to arrhythmia due to ischaemic heart disease.
Toxicology: Alprozolam < 0.01 $mg \cdot kg^{-1}$; tramadol 0.04 $mg \cdot kg^{-1}$ (therapeutic)

Comments: This elderly man with a history of significant cardiovascular disease had been assessed as medically fit four months earlier although it is not clear what this assessment was for. His failure to declare his conditions to the operator resulted in a lower level of surveillance and support, although this might not have changed the outcome as the other snorkellers appeared to have recognised the problem promptly and reacted appropriately and swiftly. He was obviously overconfident about his capabilities and health and determined to snorkel. The staff repeatedly offered him a buoyancy aid but it is unclear whether this would have prevented what was likely an arrhythmia precipitated by the combination of severe cardiac disease, immersion and exertion.

The authors are not aware of studies of the physiological effects of immersion of elderly individuals taking cardioactive medications. This individual was taking a potent pulmonary vasodilator as well as medication with the potential to alter his renal response to immersion. The effects of such combinations on an individual with impaired cardiovascular reserve secondary to ischaemic heart disease may well have resulted in the sudden onset of an arrhythmia or possibly IPE.

Summary: 82 y.o. male; history of hypertension, hypercholesterolaemia, stroke and coronary atherosclerosis; capable swimmer but snorkelling ability unknown; failed to declare medical conditions; refused buoyancy aids; became unconscious in water; drowning (likely cardiac-related)

BH 12/03

This 37 y.o. male was an overseas visitor with a history of hypercholesterolaemia (medications unreported) and of several episodes of post-exercise hypotension. There was a family history of hypertrophic cardiomyopathy in three of his uncles with a known genetic mutation (MYPBC3 c.1073delC). However, a resting ECG and exercise test done eight months prior to the incident were normal. His swimming ability and snorkelling experience were unknown. He was among a group of nine conference delegates who went snorkelling from the beach of a small offshore island as part of an organised kayak tour. The weather was clear with a moderate wind and there was a surface chop. No information was available about the current. The victim was wearing mask, snorkel, fins and board shorts. He was not using a buoyancy aid.

Sometime later, the victim waved his arm indicating that he was in distress. The tour operator went to his assistance and he was loaded onto a nearby dive boat, unconscious and apnoeic. The boat transferred him to the local water police office (possibly a 10-min trip). Police boarded the vessel and began BLS. Supplementary oxygen was provided (method unknown). Paramedics arrived 20 min later and implemented ALS. The victim was initially found to be in ventricular fibrillation (VF) and so was defibrillated. However, this reverted to asystole and then pulseless electrical activity (PEA) before spontaneous circulation returned. The victim was transported to hospital but died six hours post incident.

Autopsy: (1 day post mortem) There was bruising of the tongue. The heart weighed 354 g (n.r. 331–469 g) and appeared normal. The free wall of the left ventricle was 10 mm and the inter-ventricular septum was 11 mm. The ratio of septal to left ventricular posterior wall thickness was 1.1 (in hypertrophic cardiomyopathy this ratio is greater than 1.4).[2] There was a 50% stenosis of the first diagonal artery of the left anterior descending (LAD) coronary artery. Histologically, there were occasional microscopic foci of myocardial fibre disarray of uncertain significance. The right and left lungs weighed 1014 g (n.r. 446–880 g) and 826 g (n.r. 348–790 g), respectively, and were heavy with haemorrhagic oedema and early aspiration pneumonia. Ante-mortem serum osmolarity was high, 331 $mmol \cdot L^{-1}$ (n.r. 275–295). The cause of death was given as aspiration pneumonia and multi-organ ischaemia secondary to a cardiac arrhythmia (ventricular fibrillation), diagnosed clinically.

Comments: This case strongly suggests some form of cardiac arrhythmia but the exact underlying cause is not clear. While it would be interesting to know whether he had the MYPBC3 c.1073delC mutation, the absence of asymmetric hypertrophy would tend to exclude this as a contributor. A 50% stenosis of a coronary artery would not be regarded generally as sufficient as a cause of sudden death (> 75% stenosis is usually required[3]) although isolated cases during exercise are seen occasionally. The high osmolarity suggests possible aspiration of salt water but could also reflect treatment to reduce cerebral oedema.

Summary: 37 y.o. male; history of hypercholesterolaemia and post-exercise hypotension. Swimming and snorkelling abilities unknown; snorkelling under supervision; indicated distress and became unconscious in water; aspiration pneumonia and multi-organ failure post-drowning

BH 12/04

This victim was a 62 y.o. male interstate tourist who was previously a heavy drinker and smoker. He had suffered a myocardial infarction (MI) five years earlier and had insulin-dependent diabetes, requiring four injections per day. He also was taking diltiazem, ezetimibe, atorvastatin and aspirin. He was a strong swimmer with extensive snorkelling experience, albeit not for the past three years. He and three family members were on a day trip to an island on the GBR. After arrival, the group did a moderately strenuous walk across the island to a beach. On three occasions, the victim had to rest for five minutes after becoming dyspnoeic. Once at the beach, the victim and others had lunch before setting out snorkelling.

The sea was calm with a slight current and a water temperature of 29°C. The victim was wearing a hired mask, snorkel and fins. He snorkelled for a short time before resting on the beach for over two hours. He did not take his scheduled insulin. He and his daughter then decided to snorkel, with the aid of the current, back towards the jetty. The daughter initially swam about 10 m ahead and after a short distance, on reaching shallow water, she stood and saw the victim sitting on a rock. When she asked if he was "*OK*", he answered affirmatively. He began to snorkel towards her but she was soon unable to see him as he was obscured by a large rock. When he failed to appear beyond the rock, she raised the alarm. A search involving several vessels was soon commenced and, after 45 min, the victim was found about 10 m from where he was last seen. He was partly submerged face-down in 30 cm of water between some rocks. His mask, snorkel and fins were in the water

nearby. He was unconscious, apnoeic and cyanotic. BLS was initiated by a crew member of a dive vessel. Supplementary oxygen was provided (method unknown). BLS was continued until a doctor arriving by rescue helicopter advised that resuscitation efforts be ceased. When the snorkelling equipment was examined, no significant issues were found.

Autopsy: (2 days post mortem) The heart was enlarged, weighing 663 g (n.r. 331–461 g) and with scarring and thinning of the apex of the left ventricle. The coronary arteries showed severe calcified atherosclerosis. The right and left lungs weighed 796 g (n.r. 446–880 g) and 573 g (n.r. 348–790 g), respectively, and were mildly oedematous. The cause of death was given as cardiac arrhythmia due to ischaemic heart disease.

Comments: With his history of MI and diabetes and substantial breathlessness on exertion, it is unsurprising that this man encountered difficulties when snorkelling. The effects of immersion and exertion (exacerbated by probable hyperglycaemia) likely combined to cause an arrhythmia and subsequent unconsciousness in the water.

Summary: 62 y.o. male; known history of healed MI and insulin-dependent diabetes; strong swimmer and experienced snorkeller; dyspnoeic during walk to site; missed scheduled insulin injection; separation from buddy; cardiac event

BH 12/05

This 68 y.o. male overseas tourist was visiting the GBR on a cruise. Together with over 200 others, he joined a tour with a snorkel operator to a pontoon on the reef. He was obese (BMI 33 $kg \cdot m^{-2}$) with a history of an MI 17 years earlier. He was taking glyceryl trinitrate and salbutamol. On the morning of the incident he had "*taken antacid for an upset stomach*". He was described as a competent swimmer but there was no indication of snorkelling experience. Although all participants were asked to complete a medical questionnaire, it appears that he had not done so. The sea was smooth and visibility was good but there was a strong current, estimated to be 4–5 $km \cdot hr^{-1}$. One witness noted that some snorkellers were being swept away in the current and needed to be towed back to the pontoon.

The victim was wearing his own mask and snorkel but had hired fins and a stinger suit, and did not take a floatation aid. He was in the water with about 30 others when he was seen 25–30 m distant from the pontoon and signalling that he was in difficulty. A staff member in a tender went to him and he grabbed a rope attached to the tender. When asked if he was okay he did not answer but declined boarding the tender. He held the rope and was towed to the pontoon but became unresponsive. The staff reported that his head did not submerge and his fingers had to be pried from the rope when he was dragged onto the pontoon. Frothy sputum exuded from his mouth. BLS was commenced by staff with the help of two doctors (tourists). An AED was used but no details were provided. Adrenaline was administered but the victim failed to respond and resuscitation was ceased after about 25 min.

Autopsy: (3 days post mortem) The heart was enlarged and weighed 505 g (n.r. 331–469 g). There was aneurysmal scarring of the apex and anterior wall of the left ventricle due to an old infarct. There was occlusion of the LAD coronary artery and severe calcified atherosclerosis of the right and circumflex coronary arteries. The upper airways contained no fluid. The right and left lungs weighed 845 g (n.r. 446–880 g) and 845 g (n.r. 348–790 g), respectively, and were moderately oedematous. The cause of death was given as left ventricular failure. The reviewers believe the cause of death would be more aptly described as cardiac arrhythmia due to ischaemic heart disease.

Comments: In light of this man's very significant cardiac conditions, he was highly predisposed to an exertion-related cardiac event, whether aquatic or terrestrial. It appears that he failed to declare his medical condition to the operator who stated that had he done so he would have been classified as 'high risk' and been supervised more closely. (At least one other passenger had declared a previous MI and was permitted to snorkel). However, it is unlikely that this would have made a difference as his rescue was prompt and apparently appropriate. The operator's wisdom in permitting snorkellers of varying health and abilities to snorkel in such a strong current is questionable.

Summary: 68 y.o. male; history of previous MI and current angina; competent swimmer but unknown snorkelling experience; strong current; signalled for help and became unconscious while being towed to pontoon; AED used (no details); cardiac-related

BH 12/06

This 26 y.o. male had a history of idiopathic epilepsy, with four seizures over the previous three years. He was poorly compliant with his prescribed medication (Valproic acid) and two of the seizures were associated with recent marijuana use. He was otherwise well, a strong swimmer and experienced snorkeller who owned his own equipment.

He and nine others went snorkelling and spearfishing from some rocks at a headland. The weather was clear and warm (28°C) and the water was choppy with a 1 m swell. There was little current, the visibility was 5 m and water temperature 24°C. The depth of the site varied between about 10–20 metres' sea water (msw). One of the group stayed on the rocks while the others snorkelled. The victim was wearing mask, snorkel, fins with bootees, gloves and board shorts. He was the only one not wearing a wetsuit and was warned that he would be cold. One of his fin straps broke prior to entry and was subsequently secured with a wire.

The group snorkelled separately close to the edge of the rocks in what was described as a "*radius of 30 m of each other*". The victim was last seen swimming towards the rocks after about 10 min. When one of the group returned to the rocks after about 40 min and noticed the victim's towel untouched, he became concerned as he had expected him to be the first out from getting cold. The emergency services were contacted and an air and sea search was conducted, followed by underwater searches over the next two days. The victim's body was found floating 200 m offshore three days after he was last seen. He was still wearing his shorts, gloves and one boot but the rest of his gear was missing.

Autopsy: (4 days post mortem) There were early decompositional changes (the body not recovered for three days). The brain was heavy 1618 g (n.r. < 1,500 g) and mildly oedematous but showed, only decompositional change. "*There was a defect on the left posterior aspect of the tongue which could be consistent with decomposition,*"(sic). The heart was normal and weighed 348 g (n.r. 331–469 g). The coronary arteries were patent. The upper airways contained pulmonary oedema and decompositional fluid. The right and left lungs weighed 846 g (n.r. 410–892 g) and 719 g (n.r. 378–780 g), respectively, with 500 ml of fluid in the left pleural cavity and 350 ml of fluid in the right pleural cavity (in early decomposition in salt water drowning fluid can move from the lungs to the pleural cavity).[4] The cause of death was given as drowning *Toxicology*: Alcohol 0.068 g·100 mL^{-1}; Delta–9-THC acid 0.031 $mg \cdot L^{-1}$; paracetamol 3 $mg \cdot L^{-1}$; valproic acid < 10 $mg \cdot L^{-1}$ (sub-therapeutic)

Comments: The victim might have drowned as a result of becoming debilitated by the cold. Apnoeic hypoxia was another possibility as was an epileptic seizure. No specific bruising of the tongue was described but it is unclear to the reviewers whether this defect could be evidence of seizure activity. While the anticonvulsive was noted to be sub-therapeutic, this was not thought to be significant by the coroner. Snorkelling may involve a variety of factors that can reduce the seizure threshold. These include stress, exercise, sensory deprivation, hypercapnoea, hyperventilation and hypothermia. This individual was poorly compliant with his medication and had recently taken marijuana, which had been associated with some of his seizures. He would have also been cold while snorkelling. The combination of these factors while snorkelling may have precipitated a seizure. The toxicology is hard to interpret. Alcohol can lower the seizure threshold but some or all of the alcohol detected could be due to decomposition. Metabolites of cannabis were detected but no parent compound, which indicates past exposure but not proximate use.

Summary: 26 y.o. male; history of epilepsy; strong and experienced snorkeller; not wearing wetsuit in relatively cool water; snorkelling with others but with no buddy system; body found three days later; drowning (Epilepsy? Apnoeic hypoxia? Hypothermia?)

BH 12/07

This 70 y.o. woman was an overseas tourist holidaying on the north-west coast of Australia. She had a history of hypothyroidism for which she was prescribed thyroxine. Her last medical consultation was one month prior to her trip and she was reported to have been in good health. There was no information about her swimming or snorkelling ability or experience.

She had snorkelled for an hour without incident in the morning. After lunch and a rest, she and her husband entered the water from a sandy beach surrounded by coral reef. The sea was calm and she wore a mask and snorkel but no fins and was not using a flotation device. They separated on entry as the husband was taking photos. After eight minutes, he snorkelled towards shore and then looked for his wife. He could see her approximately 30 m from shore and called to a person on a nearby boat and asked if she would check his wife. This person noticed that the victim was floating face-down but only moving with the water action. She donned her snorkelling gear, took a flotation aid and swam to the victim. She found her to be unconscious with her head submerged, her mask in place but full of blood and water, and with the snorkel out of her mouth. Blood-stained frothy sputum was visible in the victim's mouth. The rescuer also noticed blood in the water all around the victim. She removed the victim's mask and supported her with the float. She then tried unsuccessfully to detect breathing and a pulse before attempting in-water CPR, but soon abandoned this. With the aid of another rescuer, the victim was towed to shore, which took 2–3 min.

BLS was commenced using a resuscitation mask as a barrier. The victim was rolled onto her side several times to clear blood-stained frothy sputum from her mouth. The ambulance was called some 10–12 min from the time the victim was first sighted and arrived 13 min later. Supplementary oxygen was provided using a manually-triggered resuscitator (MTR) and an AED was attached but no shock advised. She was taken to the nursing post, where her airway was suctioned and an oropharyngeal airway inserted. A defibrillator was attached and indicated asystole, so BLS continued with ventilations given via a bag-valve-mask (BVM) resuscitator. However, a short time later, although the monitor of the defibrillator indicated asystole, it advised that a shock be given, which was done without effect. Resuscitation efforts were ceased after a total of 50 min.

Autopsy: (3 days post mortem) No blood was described on the face at autopsy. The heart was normal and weighed 316 g (n.r. 285–439 g), with mild coronary atherosclerosis. The upper airway was clear, the right and left lungs weighing 680 g (n.r. 305–817 g) and 638 g (n.r. 287–695 g), respectively and with marked pulmonary oedema. Subsequent histology of the lung showed early bronchopneumonia. The cause of death was given as "*consistent with drowning*".

Comments: In the absence of reports about her snorkelling ability and the depth of the water in which she was found, it is unclear why she apparently drowned. What happened to the victim and if she showed any indications of distress before becoming unconscious is unknown. The presence of blood in the mask and in the water around the body is unexplained but could have been secondary to pulmonary oedema.

Summary: 70 y.o. female; history of hypothyroidism controlled by thyroxine; entered water with buddy but separated as buddy pre-occupied with taking photographs; found unconscious; one belated shock with AED; drowning

BH 12/08

A 71 y.o. male, overseas tourist and his wife were among a group of 63 passengers on a large commercial snorkelling/diving vessel on a day-trip to the GBR. He was apparently well and, other than gout and passing a kidney stone three months earlier, had no significant medical history. He was taking unknown medication for nephrolithiasis and gout. He was described as 180 cm tall and of a "*solid build*". On his pre-snorkel declaration, he stated that he was an average swimmer (although he swam daily), but an experienced snorkeller. He owned a yacht and he and his wife sailed regularly.

As part of the pre-snorkel brief, passengers were advised to raise their hand if in need of assistance. The vessel moored about 30 m from the top of the reef and a 'mermaid line' was trailing from the stern to the reef. There was a permanent snorkelling rest station about 20–30 m from the stern. The weather was clear with a gentle to moderate breeze (20–30 km·hr^{-1}). The water was warm (27°C) and choppy, visibility varied from 8–15 m, and there was a strong current. The maximum depth at the site was 20 msw.

The victim and his wife donned masks, snorkels, fins and stinger suits and entered the water. After about 15 min the husband noticed that they had been swept away from the boat and they started to swim back towards it but were unable to make much headway owing to the current. The wife reached the rest station and raised her hand to indicate that she needed assistance. She saw the victim, who was 4–5 m away also raise his hand, before submerging in what she thought was a duck-dive. However, he surfaced and made a "*loud screaming sound*". She then lost sight of him. The wife waited at the rest station for the next 15 min, raising her hand periodically to attract attention. She was distressed and exhausted. There were 20–30 snorkellers in the water and the lookout repeatedly failed to recognise that the victim's wife needed help. The staff only responded after another passenger swam to the wife and returned to the boat to raise the alarm. After another 10–15 min, the crew commenced a surface and underwater search. The emergency services were alerted and a rescue helicopter arrived 50 min later followed several hours later by the water police. The search continued for five days but no sign of the victim was ever found.

Autopsy: Body not recovered.

Comments: In the absence of an autopsy, one can only speculate on what might have occurred based on witness statements. An autopsy provides a lot of information about an older individual, especially in regard to pre-existing medical conditions and injuries. It is probable that the victim was tired from swimming against the current and may have become incapacitated, possibly aspirating some water. Despite the absence of any supporting evidence, it is remotely possible that he was taken by a shark.

The delay in recognising the wife's distress raises substantial concerns about the training and effectiveness of the lookout system. The lookout later opined that, despite the passengers being briefed to raise their hand if needing help, a person in distress would be splashing around and not just raising a hand. This is not necessarily the case and indicates the lack of insight of the lookout. With 20–30 snorkellers to observe (as well as some scuba divers), it can be difficult for a lookout to recognise a problem, especially if they are relatively inexperienced. There is a need to ensure that there are sufficient lookouts, that they are adequately trained in appropriate observation techniques and that they are relieved after relatively short intervals to avoid fatigue or complacency.

Summary: 71 y.o. male; history of gout and nephrolithiasis; otherwise apparently well; average swimmer and experienced and regular snorkeller; strong current; fatigue; submerged, surfaced and made loud scream before vanishing; body not recovered; medical problem?/drowning?

BH 12/09

This 31 y.o. male was a strong swimmer, an experienced spearfisherman and a certified free-diving instructor. No information was provided about his fitness or medical history. He and three friends were spearfishing from a 5.5 m rigid inflatable boat which was anchored by a rocky reef with a maximum depth of around 25 msw. The weather was clear with a light breeze, the surface was calm, albeit with a 2m+ swell, little current and good visibility (15–20 m). He was wearing a mask, snorkel, fins, boots, gloves; a wetsuit with attached hood; a weight belt with 4 kg of weights; a knife and was carrying a speargun.

The group performed about 10 dives up to 25 msw before deciding to move to another site. However, the victim decided to do one more dive, which he did while the others boarded the boat. When he failed to appear after several min, the group became concerned and called for the assistance of some scuba divers in a nearby boat. The divers entered the water and found the victim's speargun, but some aborted the dive after seeing a large shark. Two divers who persisted found the victim lying in kelp (not entangled) at a depth of 24 msw. He was unconscious with blood flowing from his mouth. His weight belt was still in place. While the

divers brought him to the surface, one purged her 'octopus' regulator into his mouth in an attempt to ventilate him.

He was dragged aboard the boat approximately 35 min after he was last seen and BLS was commenced. The victim was rolled onto his side regularly to drain blood-stained sputum and water from his airway. Initially his weight belt had not been removed and stomach inflation was visible. Rescue breaths were supplemented with oxygen via a resuscitation mask with a flowrate of 15 $L \cdot min^{-1}$. When the boat reached land, some 100 min after the victim was recovered, paramedics were waiting and continued resuscitation for 15 min before efforts were ceased.

Autopsy: (4 days post mortem) Post-mortem CT scan showed extensive intra-vascular gas throughout the arterial and venous systemic and pulmonary venous system, large amount of gas filling the left side of the heart but with a relatively small amount of gas present within the right ventricle. The heart appeared to be structurally normal. There was fluid filling the central airways and diffuse, patchy centrilobular airspace changes were present throughout both lungs with some predominantly basal interlobular septal thickening. Only an external examination was performed, which showed petechiae around the eyes and on the conjunctiva and froth in the mouth possibly suggesting drowning. The cause of death was given as "*undetermined*".

Comments: This man should have had a full autopsy to establish the cause of death, especially given the gas seen on CT scan and the undetermined cause of death. The observed gas could have been due to resuscitation artefact from vigorous BLS. It is also possible that the rescuer's purging of her 'octopus' regulator into the victim's mouth during ascent caused some barotrauma and subsequent gas distribution. Although there was no report about his medical history, it is likely that his wife would have mentioned if he had a significant condition such as epilepsy. It is unknown whether or not he practiced hyperventilation – this is a question that investigators should routinely ask companions in such situations. It seems likely that the victim suffered apnoeic hypoxia during ascent as a result of extended breath-holding, with or without hyperventilation, and then sank to the bottom (evidenced by signs of facial barotrauma).

Summary: 31 y.o. male; medical history unknown; strong swimmer and trained and experienced breath-hold diver; deep breath-hold dives (24 msw); did last dive alone; recovered after delay; likely drowning post apnoeic hypoxia

BH 12/10

This 64 y.o. male was obese (BMI 34 $kg \cdot m^{-2}$) and had a history of hypercholesterolaemia and arthritis, but it is unknown what medications he was taking. There was no report of his swimming ability or snorkelling experience. He was an interstate tourist who went snorkelling with family members in a thermal pool, wearing swimming goggles and a snorkel but no fins. After entering the water, he swam upstream for a short time before turning back. He then appeared to have difficulty swimming, began to thrash around in the water and called for help. When rescuers reached him, he was unconscious, apnoeic and became cyanotic. One rescuer attempted to give in-water rescue breaths, but had difficulty due to lack of buoyancy. However, a short time later, the victim began to breathe spontaneously. A rope was tied around his chest and he was towed, floating on his back, across the pool but again became apnoeic and cyanotic. The rescuers hauled the victim onto a platform and began BLS. After a short time, nurses from a nearby clinic arrived, suctioned the victim's airway and attached an AED. No shock was advised on three occasions. Resuscitation was ceased after a total time of around 35 min.

Autopsy: (1 day post mortem) The heart weighed 430 g (n.r. 331–469 g) and was reported as enlarged. The left ventricle showed mild left ventricular hypertrophy and measured 15 mm (n.r. ≤ 14 mm). The coronary arteries showed moderate (50% narrowing) atherosclerosis. The trachea and bronchi contained a small amount of white frothy fluid. The right and left lungs weighed 460 g (n.r. 446–880 g) and 540 g (n.r. 348–790 g) respectively, and were congested, although pulmonary oedema was not described. The cause of death was given as undetermined but the possibility of a sudden cardiac event was raised.

Comments: On the basis of the available information, it is unclear what precipitated the eventual drowning in this victim. However, he had no fins, lacked buoyancy support and was wearing swimming goggles which do not isolate the nose so there was ample potential for aspiration and primary drowning.

Summary: 64 y.o. male; history of hypercholesterolaemia and arthritis; swimming ability and snorkelling experience unknown; snorkelling in thermal spring; became distressed and called for help; in-water rescue breathing attempted unsuccessfully; AED attached (probable 15–20 min delay) but no shock advised; drowning (cardiac-related?)

BH 12/11

This 45 y.o. male was an overseas tourist on a day trip to the GBR aboard a large tourist vessel. He had no medical history and was on no regular medication, although he took one sea-sickness tablet (diphenhydramine) before boarding. He was a non-swimmer and had not snorkelled before. He spoke little English but attended a multi-lingual pre-snorkel/dive briefing which included his native language. He had enrolled in a Discover Scuba Dive.

Once at the pontoon, the victim wearing shorts, a mask and snorkel (it is unknown if he wore fins) and a life-jacket decided to go snorkelling before his scuba dive. The weather

was clear, the water calm and visibility good. There was a slight current. He began to snorkel in a roped area in front of the pontoon, along with 10 snorkellers and with a lookout on duty. After about 15–30 min a lookout noticed a floating object about 120 m distant. When a tender reached the object, the driver found that it was the victim, floating unconscious with his face submerged. When lifted from the water and his mask and snorkel removed, white, frothy sputum was seen coming from his mouth. He was apnoeic and cyanotic and the driver tried to perform chest compressions as he drove the tender to the pontoon.

On the pontoon, BLS was commenced by staff who were assisted by an off-duty nurse and paramedic. It is unknown how long the victim had been unconscious before being seen, but the time from reaching the victim until commencement of BLS was approximately 5 min. The victim had to be rolled on his side to drain stomach contents from his mouth. The rescuers attempted unsuccessfully to insert a supraglottic airway (i-gel) and the victim 'vomited' so suction was used. Supplementary oxygen was provided via a BVM and an AED was attached some 10 min after BLS began. It advised no shock be given, but two shocks were given later without an apparent return of circulation. A rescue helicopter with paramedics aboard arrived about 80 min after the rescue, implemented ALS and transported the victim to hospital. No ambulance report is available so it is unknown whether spontaneous circulation was re-established, but it is reported that he died the next day. A head CT showed a subarachnoid haemorrhage and cerebral oedema.

A subsequent investigation by the local workplace authority resulted in the issuance of two improvement notices, which were: (1) "*To include in the snorkel briefing if passengers cannot swim to advise crew members of the situation*"; and (2) "*To review current snorkelling area to a distance that makes supervision of the area better.*"

Autopsy: (1 day post mortem) Post-mortem examination consisted of external examination, CT scan and toxicology, but no internal examination. The CT scan (on the day of death) confirmed subarachnoid haemorrhage and cerebral oedema. External examination showed superficial abrasions on the back, but was otherwise unremarkable. The cause of death was given as drowning post subarachnoid haemorrhage.
Toxicology: Diphenhydramine 0.04 $mg \cdot kg^{-1}$ (therapeutic).

Comments: Such a large, regular operator should have had processes in place to readily identify a non-swimmer so that suitable supervision is provided, enabling a prompt rescue in the event of an incident. With only 10 snorkellers in the water at the time, the lookout should have more promptly recognised that the snorkeller was in trouble. An important consideration for snorkelling operators to consider is the size of the area under watch and the number of lookouts required to properly scrutinise a large area, especially if there are many snorkellers.

Summary: 45 y.o. male; apparently fit and healthy with no known medical history; non-swimmer; unknown period of unconsciousness in water; 2 shocks with AED; died next day in hospital; Drowning post subarachnoid haemorrhage

BH 12/12

This 68 y.o. male overseas tourist was on a day-trip to snorkel on the GBR on a charter vessel with 32 other passengers. He had been diagnosed with borderline hypertension but was not on medication. He had visited his doctor about two weeks earlier and appeared to be relatively healthy. He was a competent swimmer and had snorkelled on several previous overseas trips.

On the morning of the incident the victim appeared to be well. The conditions were "*fine*" with calm seas, good visibility and a water temperature of 27°C. There was no mention of any current. He and his wife entered the water from the beach of a small coral cay with a guide-led group. He was wearing mask, snorkel and fins and had chosen not to take a life vest. A short time later, after helping his wife with her mask, he seems to have fallen behind the group who were reportedly snorkelling quite quickly. There was one lookout on the beach and another assistant helping guests in the water. It is unclear if there was a lookout on the boat which was moored approximately 50 m from shore. The beach lookout recalled initially noticing the victim snorkelling slowly and making headway. However, possibly 10 min later when she looked again, she noticed that he was stationary and his snorkel was out of his mouth. He was recovered from shallow water (1.2 msw), about 25 m from shore, and brought to the beach. He was apnoeic, cyanotic, his eyes were open and there was froth coming from his mouth.

BLS was initially commenced by the skipper who then stopped and left to fetch an AED and oxygen equipment from the main vessel. It appears that, in his absence, no-one performed any resuscitation as the lookouts were untrained in this. When the skipper returned, possibly 5–10 min later, he attached the AED but no shock was advised. He was soon joined by two passengers who were doctors (one a cardiologist). It appears that supplementary oxygen was provided via a resuscitation mask with oxygen inlet. It was necessary to roll the victim onto his side several times to drain water from his mouth. BLS continued until the arrival of a paramedic on a rescue helicopter. When attached, the cardiac monitor indicated asystole and, given the period of unconsciousness and the appearance of the victim (grey with fixed dilated pupils), resuscitation was ceased.

Autopsy: (2 days post mortem) The heart weighed 432 g (n.r. 295–445 g). There was mild left ventricular hypertrophy of 18 mm (n.r. ≤ 14mm), and right ventricular hypertrophy of 5 mm (n.r. ≤ 4mm). There was moderate coronary atherosclerosis (approximately 50% stenosis) and mild patchy interstitial fibrosis. There was white froth in the

right bronchus. The right and left lungs weighed 723 g (n.r. 410–892 g) and 699 g (n.r. 378–780 g) respectively, and were moderately oedematous and congested. The cause of death was given as drowning with other significant conditions including cardiomegaly and moderate coronary artery disease.

Comments: There was evidence of enough cardiac disease to suggest a possible cardiac-precipitated drowning. The histological evidence of fibrosis in association with left ventricular hypertrophy has been associated with sudden cardiac death and increased incidence of arrhythmias.[5] Despite borderline hypertension, the victim appeared to be healthy, a competent swimmer with some snorkelling experience. He did not appear to have been anxious, the conditions were good and the water was shallow. No-one noticed any indications of distress. However, at least one of the lookouts was inexperienced, another pre-occupied, and the third possibly non-existent, so signs of distress, if any, could have been missed. The outcome may not have been any different had there been an effective lookout and first aid system in place. However, this case again highlights some important considerations for snorkel operators in relation to supervision and first aid provision.

Summary: 68 y.o. male; borderline hypertension (unmedicated); competent swimmer and confident snorkeller; inefficient lookout system; noticed with snorkel displaced; BLS abandoned for extensive period while equipment being retrieved; drowning (cardiac-related)

BH 12/13

This 27 y.o. male, overseas national living in Australia was fit and healthy with no medical history except back pain for which he took herbal medicine. He was a strong swimmer (previously a part-time lifeguard) and an experienced and regular (weekly) breath-hold diver. He had been scuba and breath-hold diving for nine years. He and two friends went spearfishing off a small offshore island with depths up to 20 msw. He had dived there on many occasions, including the previous day. There was a clear sky, no wind, a slight sea and only a weak current; visibility was less than 5 m. He was wearing mask, snorkel, fins, gloves, full length 5 mm wetsuit, weight belt with 9.5 kg of weights, a knife and was carrying a speargun attached to a line and float.

The victim was the last to enter the water and took some time to reach the others. He told them that he had just dived to 15 msw – a personal record. The trio continued snorkelling with the victim swimming about 20 m behind. Before returning to shore, one of the companions looked back and saw the victim's float, presuming that he was diving. At this point it was about 150 m from shore. On reaching shore, one of the companions became concerned that the victim's float had not moved and he was still not visible so he re-entered the water, swam to a nearby boat and asked the driver to take him to the float. On arrival, he pulled in the float with the victim's speargun attached but there was no sign of the victim. The buddy then returned to shore and called the emergency services. Police arrived and a search was commenced. The victim's body was found four days later. He was lying on the bottom at a depth of 15 msw near to where he was last seen. All of his equipment was still in place. It was noted that the quick-release buckle of his weight belt was positioned behind his back, rather than at his front.

Autopsy: (4 days post mortem) The heart weighed 304 g and was normal with right dominant circulation and minimal atherosclerosis apart from myocardial bridging (25 mm long and 5 mm deep) of the LAD artery. Histologically there was no fibrosis. There was no obvious pulmonary oedema in the upper airways. The right and left lungs weighed 947 g (n.r. 410–892 g) and 764 g (n.r. 378–780 g), respectively. They were markedly hyper-inflated and there was 750 ml of red/brown fluid in the left pleural cavity, and 450 ml in the right pleural cavity (common in drowning with a longer post-mortem interval). The cause of death was given as drowning.

Comments: The clinical significance of myocardial bridging is complex and controversial. Bridging is quite common in the LAD artery and is probably only clinically significant if symptomatic, deep and long. It can cause focal proximal atheroma, may cause coronary spasm and is said to be more significant in left-dominant circulations and in cases where there is histological evidence of fibrosis. This bridge at 25 mm long and 5 mm deep was at the boundary between shallow and deep. Given the victim was relatively young with a history of exertional activities, this is unlikely to have been clinically significant. It appears most likely that this victim became unconscious and subsequently drowned as a consequence of apnoeic hypoxia (possibly post hyperventilation).

Summary: 27 y.o. male; fit and healthy with no known medical history; strong swimmer and experienced and regular breath-hold diver; spearfishing; likely intentional separation from buddies; deep breath-hold diving; drowning (probably post apnoeic hypoxia)

BH 12/14

This 38 y.o. male was a slightly-built and apparently healthy foreign national living in Australia. He was reported to have been "*not a strong swimmer*" who had little experience in collecting abalone and little knowledge of Australian coastal conditions. He had driven through the night and set out snorkelling for abalone with three friends at a site with a rocky reef and depths up to 4 msw. There was a one-metre swell, a strong surge and some current. He was wearing a wetsuit (unknown thickness) and a weight belt with four weights (possibly 12 kg), which did not have a quick-release buckle. His snorkel was attached to a three-metre length of garden hose to enable him to dive deeper and "*hold his breath*

for longer". He used an inflated tyre tube as a buoyancy aid which was attached to him by a string. One of his friends told him that he was wearing too much weight but he ignored this. It was not clear if he was wearing fins. After searching for abalone for over two hours he came ashore for a short rest, after which he abandoned the tube and re-entered the water to look for more abalone, apparently standing on submerged rocks. After another two hours, he was heard to call for help before submerging about 30 m from shore.

Two companions located him lying face-up in several metres of water, brought him to the surface after an estimated 4–5 min submersion, and ditched his weight belt. With the aid of others, they dragged him onto rocks where BLS was performed by bystanders and some of the companions. The victim's airway was soiled by white frothy sputum and regurgitated stomach contents. A first responder arrived with an AED about 20 min later. When attached, the AED indicated that no shock was advised. BLS was continued until paramedics arrived about 10 min later. Several shocks were delivered by their defibrillator but the victim remained in asystole and resuscitation was ceased.

Autopsy: (4 days post mortem) The heart weighed 332 g (n.r. 295–445 g) and was normal apart from a focal 50% stenosis at the origin of the LAD coronary artery. There was moderate bloody frothy fluid in the trachea and bronchi. The right and left lungs weighed 940 g (n.r. 410–892 g) and 764 g (n.r. 348–790 g) respectively, and both appeared hyper-expanded with severe pulmonary oedema. The cause of death was given as drowning due to focal coronary atherosclerosis.

Comments: This victim, a relatively poor swimmer and inexperienced snorkeller, was likely exhausted from lack of sleep and several hours of snorkelling. He was overweighted and using totally unsuitable snorkelling equipment which would have caused hypercapnia, possible hypoxia and increased his fatigue and discomfort. It appears likely that he stepped or slipped off submerged rocks on which he was standing and into deeper water. Being overweighted, unable to quickly release his weight belt and without access to his buoyancy aid he submerged and drowned. The pathologist listed moderate focal atherosclerosis of around 50% stenosis as relevant. However, there was no apparent fibrosis and a 50% stenosis is generally not regarded as a cause of sudden cardiac death.

Summary: 38 y.o. male; apparently healthy with no known medical history; weak swimmer; inexperienced snorkeller using snorkel attached to 3 m hose; overweighted; likely slipped off submerged rocks into deeper water; AED attached (20 min) but no shock advised; drowning

Scuba diving fatalities

There were 11 recreational scuba deaths and one occupational, surface supplied death, distributed across most of Australia, with the most (four) in the State of New South Wales.

SC 12/01

This 55 y.o. male appeared to be relatively fit and active and his work required strenuous manual labour. However, his medical history included essential tremor (propranolol, insertion of deep brain stimulator); depression (mirtazapine, fluoxetine, citalopram) and sleep apnoea (CPAP). Two months earlier he had fallen and sustained a head injury (no abnormality on head CT) and dislocated shoulder. He smoked 15 cigarettes a day. He certified as an Open Water Diver (OWD) seven years earlier, at which time he had a diving medical assessment. It is unknown if the doctor had any training in diving medicine. He had done approximately 200 dives, although none for nine months prior to the day of the incident.

The victim, his son and a friend set out in the son's small boat to dive for crayfish at a familiar site - an offshore island with undulating reef covered in kelp with a depth of 10–12 msw. The sea was calm with a slight current, the water temperature was 23°C and visibility was 20 m. He was wearing a mask, snorkel and fins; a BCD with 4.9 kg of integrated weights; a scuba unit with an 11.5 L steel cylinder; full 3 mm wetsuit with gloves and boots; a dive computer and he was carrying a 'Shark Shield', crayfish loop and catch bag. The first dive of the day was uneventful, with a maximum depth of 11 msw for 38 min. After some lunch, they moved the boat to a new site and re-entered the water after a surface interval of 58 min. The victim seemed to be in good spirits.

After what he reported to have been about 30 min of diving, the son returned towards the boat, stating that, at this time his father was about 35 m from it. However, after boarding the boat (an unknown time later), he heard his father call to him for help. The victim was on the surface, about 20 m away. The son quickly donned his snorkelling gear and swam to his father, who was unconscious and face-down but with his BCD inflated. The son attempted in-water chest compressions and rescue breaths but this proved too difficult. By now, the friend had surfaced and together they removed the victim's BCD/scuba unit, towed him to the boat and dragged him aboard. He was apnoeic and cyanotic so the son began BLS while the friend alerted the emergency services. A medical team from the island clinic reached the vessel about 15 min later, finding the victim to be asystolic with fixed dilated pupils. ALS was commenced but abandoned after a total of one hour of resuscitation.

When later tested, most of the equipment was found to be serviceable with the main exception being a faulty pressure gauge. When the scuba equipment was recovered, the gauge read 20 bar, although the actual content was 5 bar. Overall, the gauge gave substantially higher readings than actual at pressures lower than about 100 bar. The needle would not go below 12 bar, even without a gas supply. His dive computer was a very old model which did not enable a download of the dive profile, other than maximum depths, dive times and surface intervals. Although the computer indicated that

Table 2

Summary of scuba and surface-supplied diving-related fatalities in 2012; BCD – buoyancy compensator device; BMI – body mass index; BNS – buddy not separated; BSB – buddy separated before incident; BSD – buddy separated during incident; GSB – group separated before; GSD – group separated during; + sufficient air (to surface safely); ++ 1/4–1/2 full tank; +++ > 50% full; nad = nothing abnormal discovered; n/a – not applicable; n/i – not inflated; n/s – not stated; PBT – pulmonary barotrauma; CAGE – cerebral arterial gas embolism; IPE – immersion pulmonary oedema

SC	Age (y)	Sex	Height (m)	Weight (kg)	BMI ($kg \cdot m^{-1}$)	Training	Experience	Dive group	Dive purpose	Depth (msw)	Incident (msw)	Weights On/off (kg)		BCD	Remaining air	Equip. test	Disabling injury
12/01	55	M	175	90	29	Yes	Experienced	BSB	Crayfishing	17	Surface	Rescuer off	4.9	inflated	+	faulty SPG	CAGE?
12/02	40	M	184	87	26	Yes	Experienced	BNS	Recreation	4	4	n/s	n/s	n/s	n/s	nad	Cardiac
12/03	40	M	178	97	31	Try-dive	nil	BNS	Training	n/s	Surface	Self off	n/s	removed	+++	n/s	Cardiac
12/04	58	M	184	129	38	Yes	Experienced	BSD	Training	30	14	Rescuer off	n/s	n/s	+++	nad	Cardiac/IPE?
12/05	34	M	181	81	25	Yes	Experienced	BSB	Recreation	20	Surface	n/a	nil	inflated	+	nad	Asphyxia
12/06	25	M	182	97	29	Yes	some	BSD	Cave	24	n/s	on	2.7	n/s	nil	some issues	Asphyxia
12/07	33	M	n/s	n/s	n/s	n/s	n/s	BNS	Crayfishing	8	2	n/s	n/s	n/s	n/s	n/s	Trauma
12/08	42	M	178	100	32	Yes	Experienced	Solo	Crayfishing	5	Surface	Self off	15.1	removed	+	faulty SPG	Asphyxia
12/09	42	F	n/s	n/s	n/s	Yes	some	BSD	Recreation	33	15	on	9.8	n/s	nil	faulty SPG	Asphyxia?
12/10	68	M	185	105	31	Yes	Experienced	BNS	Recreation	12	Surface	Rescuer off	n/s	n/s	++	nad	Cardiac
12/11	45	M	161	81	28	n/s	n/s	Solo	Suicide	3	3	on	n/s	n/s	n/a	99% helium	Asphyxia (He)
SS																	
12/01	22	M	190	89	25	Yes	Limited	Solo	Commercial	16	Surface	on	n/s	not worn	+++	some issues	Asphyxia?/CAGE?

maximum depth of the last dive was 16.5 m for a total time of 62 min, both of the other divers reported that most of the dive was spent at 10–12 msw, which is consistent with charts of the site.

Autopsy: (Within 2 days post mortem) CT scan showed extensive intravascular gas especially within the cerebral arterial circulation, the aorta, the coronary arteries and some abdominal vessels. There was extensive, apparently intravascular gas in the liver, possibly in the biliary tree. There was a small left pneumothorax. (While this may represent pulmonary barotrauma (PBt)/arterial gas embolism (AGE) it is not possible to exclude post mortem decompression artefact or decomposition as a source of the gas). External examination showed surgical scarring on the head and chest related to a neurostimulator device in the right chest wall and leads inserted in the left and right frontal lobes of the brain acting as a deep brain stimulator for control of tremor. The heart weighed 410 g (n.r. 331–469 g) and appeared normal. The left atrium was opened underwater and no gas was detected. There was 40% narrowing of the LAD artery, no hypertrophy of the ventricles and minimal scarring of the myocardium microscopically. The foramen ovale was not described. The lungs were heavy and very oedematous, with the right and left lungs weighing 902 g (n.r. 446–880 g) and 944 g (n.r. 348–790 g), respectively. There were no focal abnormalities. The brain weighed 1660 g (n.r. 1179–1621 g). There was an acute-on-chronic subdural haematoma (100 mm x 80 mm x 8 mm) over the left parietal lobe without features of raised intracranial pressure. There were electrodes in the right and left inferior frontal lobes passing into the area of the thalamus. The pathologist gave the cause of death as drowning but suggested two possible underlying causes: (1) PBt/AGE on history with equivocal evidence at autopsy; and (2) seizure activity due to the acute-on-chronic subdural haematoma (the lesion appeared too small to cause death directly from pressure effects).
Toxicology: Chlorpheniramine < 0.01 $mg \cdot L^{-1}$ (therapeutic); propranolol 0.08 $mg \cdot L^{-1}$ (therapeutic).

Comments: The pathologist suggested the possibility of a seizure related to the acute-on-chronic subdural haematoma. The victim had no history of seizures and there was no evidence of tongue-biting or bite damage to the regulator mouthpiece. However, the incidence of seizures after severe head injury is around 7.1% after one year and the absence of tongue-biting does not exclude the possibility of seizure.[6] The presence of acute haemorrhage in the subdural makes it more difficult to ignore. The acute on chronic subdural haematoma probably arose from a combination of the fall, modified by the effects of neurosurgery and the two electrodes fixing the inferior frontal cortex affecting the interaction between the skull and the brain. It may be that this small subdural haematoma is unrelated to the diving death, but had it been diagnosed in life, it would likely have raised questions about fitness to dive. In any diving death where loss of consciousness is suspected, the pathologist needs to record the heart weight, the left ventricular thickness and whether the foramen ovale is patent or sealed.

It is likely that the victim did a hasty ascent as a result of his low air situation. Although the police testing did not indicate difficulty breathing from the victim's regulator until the air supply was almost depleted, it is possible that the victim did at depth, given that he only had 5 bar remaining. The source of the gas seen on CT is hard to establish since it was not reported how long after death the CT scan was performed. Scans need to be performed in the first eight hours and preferably in the first three hours before post mortem decompression artefact complicates the interpretation of arterial gas.[7] It should be reported by someone with experience of post mortem radiology and diving fatalities. The apparent low air situation and the history of reaching the surface and calling out are consistent with PBt/CAGE but there were no ascent alarms and little gas was detected at autopsy. It is likely that he had a significant dissolved gas load and the gas seen on CT scan is probably at least partly post-mortem decompression artefact, decomposition and possibly resuscitation.

Unfortunately, as the dive computer only recorded minimal details, there was no indication of the actual profile and ascent characteristics. The dive computer model used is very permissive on repetitive dives, more so than most current models. If the profiles were essentially rectangular and 10–12 msw, the victim would have been close to or may have missed a decompression obligation recommended by other decompression guides (which could manifest as gas due to post-mortem decompression artefact). If the depth recording was accurate, it appears that the victim went deeper than the others at some stage during the dive, possibly after separating from his buddy.

Summary: 55 y.o. male; history of essential tremor, depression, sleep apnoea and head injury; experienced; good conditions; intentional separation; unconsciousness on surfacing; drowning (subsequent to CAGE or seizure?)

SC 12/02

This 40 y.o. male had a history of depression for which he was prescribed escitalopram. However, he had reportedly undergone "*a comprehensive medical check and found to be in good health*" although no details were available about this assessment. He was a strong swimmer, an experienced and long-time scuba diver and dive instructor. On this occasion, he took his 12 year-old nephew for a scuba dive from the shore at a sheltered dive site. The sea was calm and there was no mention of any current. There was no report of exactly what the divers wore, other than that the victim wore the only scuba unit and the nephew swam alongside and breathed through the 'octopus'.

After a few minutes at a depth of 3–4 msw, the nephew noticed that the victim was "*twitching*" and his regulator fell from his mouth. The nephew dragged the victim to the surface by the regulator hose and some bystanders who were on the shore nearby jumped in and helped to drag him onto a rock ledge. He was seen to take a couple of gasps (probably agonal respirations) before it was decided that he was apnoeic. BLS was commenced and was accompanied by considerable stomach inflation. An ambulance arrived about 10 min later and paramedics found the victim to be apnoeic with fixed dilated pupils and no palpable pulse. An AED was attached (possibly 25 min post-event) and indicated that no shock be given. The victim was transferred to hospital by ambulance and resuscitation efforts were ceased 90 min after the commencement of the dive.

The equipment was later tested, both on the bench and during a test dive and no identifiable faults were found. The cylinder air met relevant purity standards.

Autopsy: (4 days post mortem) The heart weighed 442 g (n.r. 331–469 g) with left ventricular hypertrophy (LVH) (17 mm, n.r. ≤ 14 mm) and 70–80% stenosis of the LAD coronary artery by atherosclerosis. There was mild, patchy fibrosis but no evidence of acute ischaemia. The atrio-ventricular and sino-atrial nodes were histologically normal. There was gas in the chambers of the heart but no foamy blood. The lungs were heavy and oedematous with the right and left lungs weighing 858 g (n.r. 446–880 g) and 664 g (n.r. 348–790 g), respectively. There was a small amount of mucus and pus in the upper airway. The cause of death was given as cardiac arrhythmia due to ischaemic heart with possible secondary drowning.
Toxicology: Citalopram 0.1·mg·L^{-1} (therapeutic); Delta-9-tetrahydrocannabinol 0.006 mg·L^{-1}; Delta-9-THC acid 0.025 mg·L^{-1}; Carbon monoxide 2% saturation (probably not significant and could be from being a smoker (up to 8%) or possibly low levels of carbon monoxide in the air fill.)

Comments: Anecdotal evidence from a friend suggested that "*he had been under a lot of stress for a long time and drank Red Bull like water*". Both the unrecognised ischaemic heart disease and LVH are independent risk factors for sudden cardiac death.[8] While, the death is most likely due to the 70–80% narrowing of the LAD, it should also be noted that the antidepressant citalopram can cause dose-dependent prolongation of the QT interval,[9] which, combined with the increased risk of arrhythmias associated with LVH, makes this also a reasonable hypothesis.

Summary: 40 y.o. male; history of depression; recently assessed as in good health; experienced diver and instructor; sharing single scuba unit with buddy; good conditions; seen to twitch and become unconscious underwater soon after submerging; BLS attempted; AED indicated no shock; LVH and severe coronary atherosclerosis; cardiac death

SC 12/03

This 40 y.o. male overseas tourist was on a day trip to the GBR and enrolled in an Introductory Scuba Dive. He was obese (BMI 31 kg·m^{-2}) but his medical history was unknown. Nothing was declared on his pre-dive medical questionnaire. There were no reports on the diving conditions or site or of the equipment worn or its functionality. Whilst breathing from his regulator on the surface he lifted his head and said "*I don't think this is for me*". He indicated that he did not wish to continue and exited the water five minutes after entering.

Possibly 5–10 min later, he walked up to the air-conditioned wheelhouse and sat down. The skipper heard him breathe heavily before rubbing his temple and slumping in the chair. His eyes were open but he was unresponsive. A crew member arrived and reported that the victim was drooling with his fingers, hands and forearms clenched, his eyes open and staring and he was unresponsive. Several of the crew placed the victim in the recovery position where he remained unresponsive and with "*abnormal breathing*". His airway was cleared (there was no indication of what) and BLS was commenced (with supplementary oxygen by mouth-to-mask) and continued for 45 min until a doctor arrived by rescue helicopter and implemented ALS. When attached, the defibrillator indicated asystole and adrenalin was administered. ALS was continued for another six minutes before resuscitation attempts ceased. No medications were found in his personal effects and no personal information was available as there was difficulty contacting family or friends after the event.

Autopsy: (1 day post mortem) The heart was heavy and weighed 509 g (n.r. 331–469 g). There was mild concentric LVH, scarring of the posterior wall of the left ventricle and severe atherosclerosis of the right, left circumflex and LAD coronary arteries. Histology showed scarring and subacute ischaemic changes but no neutrophils. The lungs were oedematous and heavy, the right and left lungs weighing 1,096 g (n.r. 446–880 g) and 969 g (n.r. 348–790 g), respectively. The cause of death was given as "*an arrhythmia complicating coronary artery atheroma*".

Comments: In addition to immersion and anxiety, factors such as exertion and salt water aspiration can contribute to the occurrence of cardiac arrhythmia so it is always useful to consider these. Given that the sudden death occurred 5–10 min after immersion, the pulmonary oedema observed was likely cardiac in origin. This victim had very significant and apparently undiagnosed ischaemic heart disease with evidence of old infarction (posterior left ventricle) and more recent "*days to weeks*" ischaemia and, thus, was at risk of a cardiac event in any circumstances.

Summary: 40 y.o. male; declared no significant medical conditions or medications; obese; only breathed scuba on surface for 5 min with no submersion; collapsed unconscious on boat; cardiac-related

SC 12/04

This 58 y.o. male nurse had a past history of anxiety and depression. He was severely obese (BMI 38.1 $kg \cdot m^{-2}$) and his recent history included reflux oesophagitis, atrial fibrillation (AF) and shoulder pain. He was reported to have been non-compliant with his AF medications and had presented to emergency departments on four occasions over the previous seven years with related symptoms, on one occasion requiring cardioversion. There was some evidence of "*light-headedness*" associated with his AF which appeared to be worsening. He suffered twice-monthly episodes of palpitations lasting 1–6 hours. Several months before his death a cardiologist had recommended the fitting of a pacemaker, which had not been done. Although he had previously been prescribed amiodarone for his AF, his current long-term medications were esomeprazole, ibuprofen, metoprolol and tadalafil. He was also reported to have been taking panadeine forte for shoulder pain.

The victim was reported to have been an experienced diver in the past but had not dived for approximately 20 years until very recently, when he participated in some shallow river and shore dives. He had now enrolled in an 'Advanced Diver' course. On the pre-dive medical questionnaire, he indicated that he was taking medications, although he failed to declare that he suffered from a cardiac condition, among other required declarations. Although his declaration of taking medications should have led to an automatic referral to a doctor and subsequent assessment prior to diving, this was not done. He undertook the course and participated in three, shallow, uneventful dives.

The next day, the victim was part of a group of nine divers who participated in the 'deep dive' of the course. The group comprised the instructor, two trainee divemasters (TDM), the victim and four other students. The dive was from a boat at a site with an underwater wall. Conditions were described as calm and sunny with a light breeze. There was a slight swell and the water temperature was around 16°C. Visibility and current were not reported. The victim was wearing mask, snorkel, fins, a 7 mm one-piece wetsuit, a 12 L steel cylinder scuba unit with a BCD with integrated weight pockets (unknown weights) with attached torches and knife and a dive computer.

After diving to 30 msw for about 10 min, the group ascended along the wall. The victim lagged behind and when a TDM went to check on him, he signalled that he was out of air. The TDM checked the victim's gauge which read 130 bar, at which point the victim indicated that he was okay but wished to ascend, (they were at 14 msw at that stage). The TDM released his surface marker buoy and signalled for the victim to swim over and hold onto it. However, after looking away for a moment, he saw the victim ascend quickly past him in a prone positon, apparently unable to control his buoyancy. The TDM signalled for him to dump air from his BCD and the victim appeared to attempt this. Believing the victim to be fine, the TDM completed a safety stop before noticing the victim on the surface, floating face-down with his regulator out. He immediately surfaced, swam to the victim, rolled him over and checked for breathing and pulse, both of which appeared to be absent. The victim's jaw was stiff and he was "*bubbling and foaming a brownish liquid from his mouth*". The TDM began in-water rescue breathing and called to the boat operator to assist. Despite dumping the victim's gear, they were unable to lift him into the boat until the instructor joined them. A substantial amount of water and some frothy sputum was drained from his mouth. BLS was begun (soon with supplementary oxygen) and continued until they reached the jetty. Gurgling sounds were heard with each ventilation. Paramedics arrived soon afterwards and initiated ALS. An intercostal catheter was inserted (presumably for suspected pneumothorax). The victim failed to respond and was pronounced dead at the scene.

When later inspected, the remaining equipment was found to be in good condition and fully serviceable. The cylinder contained nitrox with 31.7% oxygen. Although the water content of the gas was high (160 $mm \cdot m^{-3}$; Australian Standard < 50 $mm \cdot m^{-3}$) this, or any equipment fault were not believed to have contributed to the victim's demise.

Autopsy: (1 day post mortem) Post mortem CT scan was not undertaken, making it difficult to assess the possibility of PBt/CAGE. There was bruising of the tongue consistent with biting of the tongue. The heart weighed 506 g (n.r. 331–469 g) with a globose shape with dilatation of the right atrium and both ventricles. There was a 30% stenosis of the LAD artery. Histology of the heart showed mild, patchy subendocardial and perivascular fibrosis and myocyte hypertrophy but no acute ischaemic changes. There was a moderate amount of blood-stained pulmonary oedema fluid in the upper airways and mouth. The lungs were heavy and oedematous, the right and left lungs weighing 930 g (n.r. 446–880 g) and 953 g (n.r. 348–790 g), respectively. The cause of death was given as unexpected death while scuba diving with significant contributing risk factors given as obesity and atrial fibrillation.

Comments: This severely obese man with a history of paroxysmal AF failed to declare this and other relevant conditions as required on the pre-dive medical declaration. However, the fact that he did declare medications should have triggered a medical examination or at least some further inquiry. The dive operator was remiss in not ensuring this. It appears that the declaration was signed just prior to beginning the course and this in itself, at such a late stage, can create a strong temptation to fail to declare relevant conditions so as not to disrupt immediate plans. On the other hand, as a nurse, it is likely that the diver was aware to some extent of the potential dangers his medical conditions posed and, in his determination to go diving, could have set out to deceive.

There was a significant cardiac history with features of a possible dilated cardiomyopathy and a documented cardiac dysrhythmia for which insertion of a pacemaker had been recommended. Given the victim's age and medical history, it is likely that the effects of immersion, exertion and breathing compressed gas (especially with an elevated PO_2) at depth precipitated a cardiac arrhythmia in this susceptible diver. The β-blocker taken to control the victim's AF may have reduced his ability to respond to the physical demands of the dive and contributed to his feeling of being 'out of air'. It is also possible that, if he went into AF, this may have triggered pulmonary oedema in the setting of immersion, evidenced by his difficulty breathing and later by the brown frothy sputum. He was using a 32% oxygen mixture, which at a depth of 30 msw, would produce a PO_2 of around 1.2 bar and it has been suggested that an elevated PO_2 may be a risk factor for IPE.[10] The effect of tadalafil (which may act as a pulmonary vasodilator) in this setting is unknown. Although there was strong evidence of pulmonary oedema at autopsy, this could also have resulted from drowning.

The rapid and uncontrolled ascent may have resulted in CAGE. It is unclear on what basis an intercostal catheter was inserted. However, in the absence of CT scanning (requested but not done) evidence of CAGE is hard to assess.

Summary: 58 y.o. male; little recent experience; severely obese; history of increasingly frequent AF, reflux oesophagitis, anxiety and depression; 'deep dive' (30 msw) on training course; signalled 'out-of-air' despite having 130 bar and functioning equipment; uncontrolled ascent alone; in-water rescue breathing; difficulty lifting victim into boat because of his size; BLS attempted; drowning (cardiac-related?/IPE?/CAGE?)

SC 12/05

This 34 y.o. apparently healthy man had been certified as a diver for two years and dived weekly, having logged around 100 dives. On this occasion he was diving with a regular, albeit less experienced buddy at a site that was familiar to both of them. The victim was wearing mask, snorkel, fins, a BCD with scuba unit with twin 7 L steel cylinders (linked by a manifold with central isolation valve) a drysuit with undergarments, socks and a hood. His equipment weighed 30 kg and he was carrying no additional weights.

At the time, the water conditions were described as "*good*" and they entered the water from the rocky shore. The water temperature was about 22°C. After about 30 min underwater, at an average depth of 12 msw, the buddy's gauge read 120 bar and he signalled to turn back towards shore. They did so and continued for a while, reaching a maximum depth of 20 msw, until the buddy's air ran low and he indicated that he wanted to surface. The buddy spent two minutes at a safety stop and was surprised that the victim did not do so, but was not concerned as he could see him on the surface. On surfacing the buddy joined the victim, who seemed fine, and they began to swim towards shore on their backs. However, this was difficult due to the increasing swell so they re-descended and swam underwater for a short time, at a depth of 5–7 msw. They again surfaced as the buddy was very low of air and again kicked on their backs towards the exit point. However, they became separated as the swell and current pushed the victim towards the rocks.

On reaching shore, the buddy saw the victim struggling to make headway and then heard him scream for help. He left the scene briefly to arrange for someone to call the emergency services. On his return several minutes later, he saw the victim floating on his back and a boat approaching him. A nearby dive boat had heard the emergency call and came to assist. The now unconscious victim was lifted onto the boat and BLS was commenced an estimated 15 min after his cry for help. A life-saver rescue boat arrived and he was transferred onto it and BLS continued as he was taken to shore. Paramedics arrived and implemented ALS and this continued until the arrival of a doctor by rescue helicopter.

On later inspection, there were no significant faults found with his equipment

Autopsy: (3 days post mortem) No CT scan was performed, but no gas was described at autopsy. There were no traumatic injuries apart from a chest abrasion thought to be due to cardiopulmonary resuscitation. The heart weighed 340 g (n.r. 295–445g) and was normal, with no significant coronary atherosclerosis. The left ventricle measured 14 mm (n.r. ≤ 14mm). The right and left lungs weighed 630 g (n.r. 410–892 g) and 842 g (n.r. 378–780 g), respectively, with moderate pulmonary oedema but no frothy fluid in the upper airways. The cause of death was given as drowning. *Toxicology*: Carbon monoxide 2% (probably not significant)

Comments: It is probable that this healthy, experienced diver found it particularly difficult swimming on the surface encumbered by twin cylinders, a drysuit and undergarments in relatively warm water. Although the fins he was wearing provided good propulsion underwater, they were relatively rigid and might not have been as effective on the surface. It is likely that he became exhausted, was swamped by a wave, aspirated and drowned. Adverse sea conditions can be a challenge and divers should never be complacent about this.

Summary: 34 year-old male; healthy; experienced and regular diver; twin tanks; returning to shore on surface with large swell and current; likely exhaustion; drowning

SC 12/06

This 25 y.o. male was an overseas national studying at university in Australia. He was certified as an open water diver (OWD) overseas four years earlier and received an advanced certification shortly afterwards. His logbook indicated that he was not on medication at that time but no subsequent medical history is available. He had logged a

total of 24 dives, three of which were between 24–30 msw. His last dive was a shallow shore dive 19 days earlier.

On this occasion he was diving in more temperate waters. He arrived at the dive centre lodge with three friends at 2–3 am and would only have had a few hours of sleep. He owned his own equipment which had never been used. It included mask, snorkel, fins; a 'wing' BCD; and regulator with 'octopus'. He checked this in at the dive shop prior to departure and all seemed functional. He hired a 5 mm one-piece wetsuit, a weight belt with 2.7 kg of weight, and a 10.5 L steel cylinder. The group's first dive was uneventful. The conditions were described as good and they did a multi-level 'orientation' dive to 15–20 msw. The divemaster (DM) reported that they all performed well, none appearing to have any problems during the dive.

After a surface interval of at least an hour, the group of four and the DM descended to the entrance of a sea cave at 24 msw. Before entering the cave, the DM asked them to check that they all had at least 150 bar of pressure in their cylinders and that their torches were functioning. After this, the group entered the cave with the DM leading, followed by the divers in single file in reverse order of their experience, the least experienced being directly behind him. The victim was the second last and was followed by a friend who was a Rescue Diver with a history of 100 dives.

There are two 'chimneys' near the entrance and the DM swam into the second and waited for the divers. He saw the first two torch beams but, when the third diver didn't arrive the DM descended the 'chimney' and then noticed a torch beam above him in the first chimney near the cave's entrance. He went there and found the victim unconscious, floating prone near the ceiling with his regulator out of his mouth and mask missing. There was no gas escaping from his equipment. He grabbed the victim and dragged him out from the cave and to the surface and then on to the nearby dive boat. The victim was noted to be unconscious, apnoeic and his face was a "*grey colour*". BLS was begun by staff, assisted by some off-duty police officers who were on the vessel, and resuscitation was coordinated by the DM, an ex-paramedic. BLS was continued while other divers were retrieved and throughout the 25-minute boat trip. Supplementary oxygen was available but was not used. Ambulance paramedics were waiting at the jetty and continued resuscitation efforts for a short time before pronouncing the victim to be dead. It was estimated by the operator that the victim had been underwater for a total of around 10 min, was unconscious underwater for possibly 4–5 min of this and was apnoeic for around 10 min before BLS was commenced on the boat.

It was later reported by the diver following the victim that, when she entered the cave, she saw vast amounts of bubbles escaping from the victim's equipment. The victim then inadvertently kicked her in the head, causing her to become disoriented and so to exit the cave.

His equipment was photographed by the dive operator and secured by the police. The 'octopus' second stage was missing and cylinder contained several litres of sea water. Three days later police divers found the victim's mask, one fin and octopus second stage at the accident scene. They reported that the 'octopus' had not been screwed tightly to the hose and had subsequently come undone. No significant fault was found with his other equipment when tested, although there was some visible damage, probably from contact with the cave.

Autopsy: (3 days post mortem) The post mortem examination was confined to external examination. On admission to the mortuary there was a large plume of pulmonary oedema coming from the mouth which had dissipated by the time of post mortem examination. There were bilateral conjunctival haemorrhages, a small laceration on the vertex of the head and minor lacerations to the left upper cheek and right lower lip. Post-mortem vitreous sodium and chloride was 277 $mmol \cdot L^{-1}$ (> 259 is reported to be consistent with salt water drowning[11]) The cause of death was given as drowning. *Toxicology*: Carbon monoxide 2% (see prior comment)

Comments: It appears that the victim's 'octopus' became detached after he entered the cave, causing panic, loss of the face mask and regulator, exhaustion of air supply, aspiration and subsequent death from drowning. It is likely that he had assembled the equipment himself, failed to secure the 'octopus' and it progressively unscrewed. When it separated, he would have been surrounded by a plume of bubbles with a rapidly depleting air supply, his 'octopus' hose would have been whipping around and it would have been difficult to orientate. Being inexperienced, he likely panicked and may have crashed into the cave wall or ceiling, sustaining facial injuries. Given the equipment failure and loss of face mask which make aspiration highly probable, internal examination of the body, though ideal, would probably not have changed the determined cause of death.

Summary: 25 y.o. male; relatively inexperienced with 26 previous dives; entering sea cave single file; octopus separated from hose; bubble plume and fast-depleting air supply; disorientation and panic; probable drowning

SC 12/07

This 33 y.o male was apparently healthy. His diving qualification and experience were not reported. He and his brother went diving for crayfish in the victim's 5 m boat. After a short uneventful dive about 4 km from shore, where visibility was very poor, they moved and re-anchored at another site 1.5 km offshore. The site comprised patches of reef and a depth of 8 msw. There were two other boats anchored 100–300 m away and their occupants were fishing. One of the fishermen later reported that fish had stopped biting and he had observed balls of baitfish and suspected there was a "*predator*" nearby.

The sky was overcast, there was a light wind and the water was choppy. However, the visibility near the surface was 6–7 m and there was little or no current. The divers geared up and rolled into the water, the victim about 3 m ahead of his brother. The victim descended along the anchor line and when he had only reached a depth of about 2 msw, a very large grey shark (with a white underside) reported to be 4–4.5 m long emerged rapidly from deeper water and engulfed the victim's head in its mouth. The shark thrashed around while retaining its grip on the victim. The brother stabbed the shark repeatedly with his knife but it just flicked him off several times, before suddenly discarding the victim and swimming away.

The victim's body was floating on the surface. His head and left arm were missing, his torso was torn open and some internal organs were visible. The brother screamed for help, grabbed the victim's remains, dragged them onto the boat and covered them with a towel. The other boats came alongside and then escorted the brother as he drove the boat to the nearest jetty. The victim's head, left shoulder and left arm and part of a wetsuit were found six days later and approximately 100 km away from the dive site.

Autopsy: (5 days post mortem) Traumatic amputation of the head and left arm and deep injuries to the chest and abdomen. There was no apparent natural disease. The cause of death was given as "*multiple injuries*".

Comments: This diver was in the wrong place at the wrong time. The shark appears to have been a very large Great White Shark (*Carcharodon carcharias*). The divers were not carrying any crayfish and had done nothing to provoke the attack. However, it is possible that the shark was attracted to the area by the nearby fishing. There were no reports that the fishermen had been 'burleying'

Summary: 33 y.o. male; just entered water with buddy; fishing activity nearby; attacked by large shark (probably Great White); multiple trauma

SC 12/08

This 42 year-old man was reported to have been very active and healthy though overweight (BMI 31.6 $kg \cdot m^{-2}$). His partner stated that he was "*fearless*", although a safety-conscious diver. She was not aware of any medical conditions and he was on no medication. He had last seen a doctor for a physical check-up and blood test 18 months prior and no concerns were found. He was a non-smoker but drank 2–3 cans of rum and coke each day. A strong swimmer, he was a keen and relatively experienced diver. Apparently, he did some training when he was much younger and had done some diving before taking a break for many years. His diving began again in earnest a few months earlier, sometimes as often as four times a week. He was a keen crayfish catcher who often dived alone and reportedly had little fear of potentially adverse diving conditions.

Two days before the incident, he had done up to six dives with one to 28 msw to retrieve crayfish pots. Without a dive watch or computer he was unaware of the times of the dives or of any decompression obligations. Following the dive he complained of unusual fatigue and malaise. Despite this, he planned to dive with friends two days later and took amphetamines at some point in the 24–48 hours preceding the dive. He also had a can of rum and coke on the way to the dive site. His friends thought it unusual that he only planned to do one dive this day as he was normally very keen to do more.

The conditions were benign, with little wind or swell. The water temperature was 17°C and visibility about 10 m. There was no mention of current but it is likely that one was present, at least later in the dive. The victim and his two regular buddies were hoping to collect crayfish and abalone and set off from shore at mid-tide and waded across reef to a small island some 100 m distant. They had all dived the site on many occasions. He was wearing a mask, snorkel, fins, wetsuit, hood, gloves and boots, a BCD/scuba unit with 11.6 L aluminium cylinder (no alternate air source), weight belt with 15 kg of weights, torch, knife and carried a catch bag. On arrival, he set off alone to dive on the seaward side of the island while the others dived on the more sheltered, landward side.

After about 40 min the friends surfaced and returned to shore, not initially concerned about the victim. However, some 20 min later he was seen to surface and hold his left arm above his head. He was not waving it or calling out so the friends and another bystander did not think that he was in distress. He then disappeared from view and after 10–20 min they became concerned and began a land and water search.

His body was washed ashore by waves about 500 m from where he was last seen and recovered by fishermen. He was floating on his back, with his mask and snorkel under his chin, and frothy sputum bubbled from his mouth. His BCD/scuba unit and weight belt were missing but his fins were in place. One of the fishermen checked for a pulse without success and tried to clear the victim's mouth, but froth continued to flow. He then gave some chest compressions but soon abandoned these believing them to be futile given that the victim had been in the water for so long (around 60 min). One of the buddies then called the emergency services.

The next day, police divers located the missing equipment where he was last seen and at a depth of 5 msw in an area free of kelp or other natural entrapment hazards. The BCD and cylinder were buoyant but the high-pressure hose and pressure gauge were entangled with the weight belt and pinned to the seabed by it. The pressure gauge read zero and the BCD shoulder straps had not been unclipped. The catch bag was empty and the torch was still on. When tested, most of the equipment functioned correctly although it was noted that the pressure gauge read low, indicating zero when the master gauge showed 8 bar. Analysis of the remaining air

showed that it met relevant purity requirements, other than the likelihood of an elevated moisture content.

Autopsy: (2 days post mortem) Post mortem CT scan of the body showed diffuse pulmonary oedema consistent with drowning. There were patchy, intravascular gas bubbles in the liver and in the coronary arteries but not in the cardiac cavities. There was some bruising on the left side of the neck. The heart weighed 440 g (n.r. 331–469 g) and appeared mildly enlarged with LVH of 20 mm (n.r. < 14 mm). The coronary arteries showed no significant atherosclerosis. There was mild, focal fibrosis in the heart on histology. There was pale, frothy fluid in the mouth and airways. The right and left lungs weighed 870 g (n.r. 446–880 g) and 725 g (n.r. 378–790 g), respectively, and were overexpanded with moderate to severe pulmonary oedema. Histology examination revealed sarcoidosis in the lungs, hilar lymph nodes and liver. There was water in the stomach. The cause of death was given as drowning in an out-of-air situation. *Toxicology*: Ethanol (< 0.01 g·100 ml^{-1}); amphetamine (blood) 1.3 mg·L^{-1}; amphetamine (urine) > 2.5 mg·L^{-1}; cathinone detected in urine (a monoamine alkaloid found in the shrub *Catha edulis* (khat) and is chemically similar to ephedrine).

Comments: It appears most likely that the victim was low on air and may have attempted to ditch his weight belt. Given that it was found entangled with the HP hose, it is possible that the weight belt caught on the pressure gauge and the victim quickly removed his BCD in order to become buoyant. His ability to react appropriately to his situation might have been hampered by fatigue (possibly from DCS from earlier dives), amphetamines and alcohol. While doing so, he probably aspirated water and became unconscious. Without the presence and prompt action of a buddy, he had little chance of survival.

Prolonged use of stimulants can cause left ventricular hypertrophy. It is possible that the combination of amphetamines and left ventricular hypertrophy may have resulted in a cardiac arrhythmia. However, the out-of-air situation and the changes of drowning are probably more significant. The stimulants could impair judgement regarding the safe length of the dive. Amphetamine can be detected up to 46 hours after ingestion.[12]

Summary: 42 y.o. male; overweight but otherwise healthy; little training but recent experience; recent use of amphetamines and alcohol; intentional buddy separation; low on air; surfaced then sank; entanglement of weight belt and scuba unit; drowning

SC 12/09

This 42 y.o., apparently healthy woman had a negative medical history other than a 25 pack-years smoking history, but had stopped four year earlier. She certified as an OWD three years earlier at which time she was assessed as fit to dive by a doctor trained in dive medicine. She subsequently did an advanced course one year before this incident and had logged a total of 55 dives, including five deeper than 28 msw, but only one over 30 msw.

She and her buddy booked two dives with a charter operator. The buddy had done 40 dives over two years, of which nine were with the victim during the previous year. He reported that the victim had consistent problems with buoyancy control, often related to difficulty finding and using the inflator and deflator buttons. The weather was fine and sunny, with a light offshore breeze, and calm sea, described as "*ideal winter diving conditions*", with a water temperature of about 12°C. She owned all of the equipment she was wearing; this consisted of mask, snorkel, fins, 7 mm full wetsuit, hood, boots, gloves, BCD and scuba unit with a 10.5 L steel cylinder, dive computer, fabric weight belt, knife, camera and torch. She was wearing a total of 9.8 kg of weights distributed between her weight belt (3.4 kg), integrated pouches in the BCD (3.4 kg) and her BCD pockets (1.5 kg in each).

The first dive was on a large wall to a maximum depth of 26 msw. There was a strong current and she ran low on air and had an uncontrolled, rapid ascent, missing a safety stop. The buddy grabbed her but was unable to stop her without also being dragged to the surface. Once on board the boat, the divemaster scolded her for missing the stop. She explained that she had had a problem locating her BCD deflator and, after demonstrating that she could now find and use it, the divemaster permitted her to do the next dive. During the 2.5-hour surface interval the victim appeared to be fine and insisted on doing the second dive, despite her buddy being reluctant due to the cold.

The next site was a wreck sitting on a sandy bottom at a depth of 33–35 msw, 4 km offshore. It was sunny the water was calm with very little swell and there was a current of 2–3.5 km·hr^{-1} on the surface. After a briefing from the divemaster and with fresh tanks, the pair descended with 11 others down the shot-line. The buddy held the victim's hand during the descent, as was their usual practice. There was little current on the bottom but the visibility was less than 5 m. The planned maximum depth was 30 msw but the victim exceeded this by landing on the bottom at 33 msw. The buddy subsequently shortened the planned dive time to accommodate this and they began ascending after 15 min. During the dive, several divers had seen the victim and noted that she had poor buoyancy control and finning technique and was swimming near-vertically.

During the ascent, the buddy carefully followed his computer and observed that the victim's ascent rate was variable, at times she was above him and sometimes below, although this was not unusual. On reaching approximately 10–12 msw, the buddy focussed on his gauges for what he estimated was 15 seconds, and, when he looked up to check on the victim,

she was not visible. He presumed that she had ascended before him, again due to poor buoyancy control. When he had last seen her she was 4–5 m below him. He completed his safety stop and, still unable to see his buddy, he surfaced with 20 bar of air remaining, about 8–10 min after last seeing the victim. Once aboard the boat, he asked others if they had seen the victim, which they had not. When she failed to surface and no bubbles were visible, a pair of divers re-descended to look for her, without success. Emergency services were promptly notified of a missing diver. A black object was seen to surface briefly in the distance but was not identified or seen again. An extensive air, sea and land search began and was continued for four days.

The following day the police used a remotely operated vehicle to scan the wreck and its vicinity for the missing diver. The scan showed the presence of a seven gill shark (*Notorynchus cepedianus*) approximately 2 m long around the site. During a search dive the next day, police found some of her equipment lying on the bottom about 50 m from the wreck. This included the victim's BCD/scuba unit, a severed weight belt with damaged pockets, a mask (with several strands of her hair) with a severed strap, snorkel, knife strap, camera, a torch and a slate. The equipment had distinctive damage in the form of rips, tears and fraying, consistent with shark bites. The weight belt was still buckled but had been severed. When the equipment was recovered it was noted that it had a "*distinct unpleasant odour*", consistent with that of bodies recovered post-submersion.

Analysis by a shark expert suggested that the damage was consistent with shark predation of a victim lying on her back on the sea bed, rather than mid-water. The pattern of damage was thought to have been most consistent with the jaw morphology and dentition of one or more Sevengill sharks 2–3 m in length. Her cylinder contained 4.5 L of water, indicating that it had been completely depleted and the pressure gauge read high by 10–15 bar. When tested, no faults were found with her BCD inflator/deflator mechanism.

Autopsy: In the absence of a body, the cause of death was given as drowning. Damage to the equipment suggested post-mortem predation.

Comments: It appears clear that, despite having logged 55 dives and done post-basic training, the victim still consistently had problems with buoyancy control and might have greatly benefitted from some specific training in this. She confided in a friend that she sometimes felt panicky underwater and this likely contributed to her poor buoyancy skills, especially at depth where she appeared to have been grossly overweighted. Ascending along a shot or anchor line provides a good datum in open water, such as this site. However, it appears likely that this pair hadn't done so and this would have contributed to the difficulty of ascent control. She had consumed air much faster than her buddy on the previous dive and probably also did so on this one. It is likely that her demise arose from a combination of low, or lack of air during the last part of the ascent, poor buoyancy control (possibly involving dumping air in error) and failure to ditch her weight belt. The most likely scenario is that she aspirated water, became unconscious and sank. One or more opportunistic sharks then predated on her body.

Summary: 42 y.o. female; no significant medical history; logged 55 dives but still had poor buoyancy control; buddy separation on ascent; not seen to surface; equipment showed signs of predation by large shark(s); body never found; probable drowning followed by post-mortem shark predation

SC 12/10

This 68 y.o. male was an overseas tourist on a day trip to the GBR on a commercial vessel with 30 people on board. His medical history is unknown but he did not declare any conditions or medications on his pre-dive medical declaration and no medications were later found with his belongings. Although only certified as an OWD, he was an experienced diver with a history of nearly 300 dives. He had brought his own mask, snorkel, fins, wetsuit and boots, BCD with integrated weights and regulator with octopus. The operator provided him with a cylinder, and possibly weights. The first dive of the day (around 10 msw for 25 min) was uneventful and the victim appeared to be well and "*in good spirits*". After a large lunch, he and five others (including an instructor) prepared to dive again. There was a moderate wind, the water had a 0.5 m chop, visibility was 10 m and there was what was variously described as a mild to strong current (possibly around 1 $km \cdot h^{-1}$ in places).

The depth at the site was 32 msw but there was no plan go that deep. The group descended the mooring line with the victim at the rear. Finding it difficult to descend and make headway against the current, the victim signalled the instructor for another weight, which he was given. The group then descended to 12 msw. The victim appeared to be competent and relaxed and took photographs. They ascended to shallower water with less current, but, after a total time of 10 min, he signalled that he wanted to ascend and did so quickly. When the instructor reached the surface, the victim told him that he was exhausted and appeared to be wide-eyed and breathless. He returned to the boat unassisted while the instructor re-joined the rest of his group.

The victim reached the dive platform and sat on it. A witness noticed blood-stained, frothy sputum at one corner of his mouth. He passed his camera to that witness before collapsing backwards and floating semi-conscious above the platform. He was quickly dragged aboard the boat, placed in the recovery position and his equipment was removed. Oxygen was administered via a non-rebreather mask. Another passenger, an ED nurse, reported that he initially responded to command but did not speak, was breathing weakly and had a weak pulse. At one point he sat up briefly before collapsing unconscious, apnoeic and without a palpable pulse. The nurse commenced BLS, assisted by

crew members. No AED was available but supplementary oxygen was provided via a BVM. BLS continued until the rescue helicopter with paramedics arrived 30 min later and ALS was implemented, without success.

When tested, all equipment was found to be functional and the remaining air met relevant purity standards. However, the primary demand valve appeared to have a relatively high 'cracking pressure' and showed signs of poor maintenance. Based on the dive computer, the victim's respiratory minute volume (RMV) during the dive was 33.2 $L \cdot m^{-1}$ which was considerably higher than on the previous dive (unreported).

Autopsy: (6 days post mortem) A CT scan (possibly 24 hours post mortem) showed gas in the coronary arteries and a small amount of gas in the left ventricle. There was extensive portal venous gas and diffuse opacification of the lung fields (possibly ante-mortem adult respiratory distress syndrome). The heart was heavy, weighing 492 g (n.r. 331–469 g). Both ventricles showed hypertrophy – the left ventricle measured 18 mm (n.r. < 14 mm), and the right ventricle 7 mm (n.r. < 4 mm).The coronary arteries were all severely narrowed by calcified atherosclerosis. The lungs were heavy and oedematous, the right and left lungs weighed 1,011 g (n.r. 446–880 g) and 930 g (n.r. 348–790g), respectively. The cause of death was given as ischaemic heart disease.

Comments: The victim had significant ischaemic heart disease, although this may have been undiagnosed. It seems likely that the effects of exertion from swimming against the current, and immersion, possibly exacerbated by breathing resistance from the demand valve, triggered a cardiac arrhythmia. He also developed either pulmonary oedema or secondary drowning. While it is possible that he ascended rapidly enough to develop a CAGE, he appears to have had problems before he ascended. The gas found on CT could have been due to either resuscitation or post-mortem decompression artifact. Given his heart disease and pulmonary oedema, gas exchange was probably impaired increasing the amount of decompression artefact. The victim's calcified coronary artery atherosclerosis may have been visible on cardiac CT scan. In a case such as this, with a witnessed collapse with rapid BLS, prompt access to an AED might have been beneficial.

Summary: 68 y.o. male; unknown medical history; experienced; exertion against moderate to strong current; fast ascent; collapsed on boat; prompt BLS but no AED; substantial IHD at autopsy; cardiac-related

SC 12/11

The medical and diving history of this 45 y.o. man were unavailable. A dive shop filled his 7 L cylinder with helium, which he said was to be used for filling balloons for his son's party. He was subsequently reported missing and police found his clothing and mobile phone on rocks at the edge of a dam. A stream of small bubbles was visible 10 m from the water's edge. Police divers located the victim's body the next day. He was face-down, 10 m from shore at a depth of 3.3 metres' fresh water (mfw), with his regulator out of his mouth. In addition to his scuba equipment, he was wearing a one-piece full wetsuit and boots, fins, mask (and presumably weights). When examined, the victim's cylinder was reported to have had a small leak, but still contained 730 L of 99% helium.

Autopsy: (2–9 days post mortem) The body showed early decompositional changes. The heart weighed 349 g (n.r. 331–469 g) and was normal with mild atherosclerosis. The right and left lungs weighed 521 g (n.r. 446–880 g) and 423 g (n.r. 348–790 g), respectively, and were moderately expanded. Based on the gas analysis reporting 99% helium in his cylinder, the cause of death was given as asphyxia by helium inhalation.
Toxicology: Carbon monoxide 1%; paracetamol 5 $mg \cdot L^{-1}$ (therapeutic)

Comments: Suicide by asphyxiation due to helium inhalation is reportedly an increasingly commonly promoted method suggested by 'right to die' advocates.[13] It seems clear that this was the victim's intent

Summary: 45 y.o. male; medical and diving history unknown; cylinder filled with 99% helium; dived alone in dam lake; asphyxiation by helium inhalation; likely suicide

Surface-supplied breathing apparatus (SSBA) fatality

SS 12/01

This fit 22 y.o. male was on no medications. He had surgery the previous year to remedy shoulder dislocations. He was a qualified recreational diving instructor with a history of more than 600 dives, but had no previous training or experience as a commercial diver. He took a job with a large pearling company and was enrolled in a three-day industry-established course in the use of surface-supplied breathing apparatus (SSBA) and other relevant equipment for pearl diving. Although covering a variety of relevant skills, the practical component included only one or two 'bail-out' dives in a pool. He was assessed as being competent.

The weather was poor, so the victim had the opportunity to spend eight days doing a relatively simple job using the SSBA equipment in sheltered conditions checking shells in a pearl farm. He told his parents that he felt much more confident after this. When the weather settled, the victim and eight other divers, five of whom were also novice pearl divers, set out to sea to collect oysters with the rest of the fleet. This is done by a specific mode of drift diving.[14] Booms are secured either side of the boat's stern and weighted shot lines and air hoses (100–150 m long) are suspended from these. Work lines run from the shot lines and drag behind them. Each diver hangs onto his work line and drifts over the sea bed, collecting oysters and placing them in a shell bag

slung over his neck. When this bag is full, the diver swims to the shot-line at the front of his work line and empties the contents into another bag. There are four divers regularly spaced each side of the stern. There may or may not be visual contact, depending on the clarity of the water, which is sometimes poor.

On the first day the victim did nine dives. On the second day he had done seven dives prior to the fatal one wearing a full 1 mm wetsuit, weight belt, hood, gloves, fins and bail-out bottle but no BCD. It appears that in the preceding dives he had at least one incident involving separation from his line in poor visibility. He complained to one of the other divers that the work was exhausting and that at the end of each dive his hands and whole body ached. He also stated that he "*almost drowned*".

The eighth dive of the second day was planned to be 13–15 msw for 45 min with 10 min of decompression. He surfaced after about 30–35 min and waved and shouted for help, possibly submerging briefly before re-surfacing and again calling for help. Two crew members heard his cry and saw the victim to be what was variously reported 40–100 m from the boat. By the time the crew member returned from alerting the captain in the wheelhouse, the victim was no longer on the surface. The skipper slowed the boat and the other divers were recalled and surfaced after completing decompression. A crew member then began to haul in the victim by hand by his hose, standing on the boom to do so. This was difficult as the boat was still moving slowly.

He was hauled to the surface and the skipper and a crew member jumped into the water to try to support him. It appears that the skipper did not have a mask or fins or other means of support and this hampered his efforts. They were unable to keep the victim's head above water and there were no reports of attempts at in-water rescue breathing. There was difficulty in bringing the victim back onto the boat and there appears to have been no specific equipment or procedure for recovering an unconscious diver. As a result, more time was lost. When the victim was finally brought aboard (hand-hauled up a 2 m ladder), one leg was tangled in the air hose. He was still wearing his weight belt and the shell bag (which contained 16 oysters) around his neck and was missing one fin. His bail-out bottle was in place and not activated. There were no apparent signs of trauma on his body.

BLS was commenced by the skipper some 20 min after the victim originally surfaced in distress. This was complicated by regurgitation of stomach contents and water. Although there was a lot of oxygen on board for use with decompression, there was no equipment to provide positive pressure oxygen ventilation. The skipper called a medical consultant some 30 min later for advice on the BLS as the victim was not responding. There was an Epipen available and the doctor suggested that nothing would be lost by injecting some adrenalin (into the thigh). BLS continued for 45–50 min but was unsuccessful. There was no AED available. The boat returned to port and the victim was taken to hospital, arriving over eight hours after the accident.

He had been using his own demand valve (attached to the SSBA) and both lugs were missing from the mouthpiece, appearing to have been bitten off. His dive computer indicated that he had dived to a maximum depth of 16 msw, surfaced for a short time, before re-submerging to a maximum of 9.5 msw for at least 12–29 min, before being hauled to the surface.

Autopsy: (4 days post mortem) The CT scan performed three to four days post-mortem was not reported. There were sub-conjunctival hemorrhages in both eyes, a healed scar on the left shoulder and early decompositional change. The heart weighed 414 g (n.r. 295–445 g) and was normal as were the coronary arteries. There were several large gas bubbles in the left atrium. There was a small amount of frothy fluid in the trachea and bronchi and the lungs were distended and oedematous with the right and left lungs weighing 802 g (n.r. 410–892 g) and 760 g (n.r. 348–790 g) respectively. Both pleural cavities contained dark-coloured fluid (fluid can move from the lungs and accumulate in the pleural cavity post-mortem). Much of the pectoralis musculature on the right side of the chest was absent. The cause of death was given as consistent with drowning. However, after further expert advice the Coroner later revised this to CAGE (mainly on the basis of a probable rapid ascent, followed shortly afterwards by unconsciousness).

Comments: Despite his experience as a recreational diver and instructor, the victim was a novice pearl diver who appeared to be having problems dealing with the unfamiliar equipment, diving techniques and physical requirements of his new job. The physical demands could have been exacerbated by the absence of his pectoralis musculature. It might have been difficult for him to hold onto the line and this could have contributed to his previous incident of separation. He was out of visual contact of the supervising diver (and others) for much of the dive and, in the absence of verbal communication equipment between divers themselves and those on the boat, it is unclear what problem he had underwater that caused him to surface in apparent distress.

It is unknown when the lugs were bitten off the mouthpiece. If this occurred while he was conscious, it would have made it difficult to retain the demand valve which may have fallen from his mouth, causing him to panic and ascend. Although he was trained to use the bail-out cylinder, this skill would not have been embedded and might have been difficult, or overlooked. Even if the mouthpiece was retained, aspiration of salt water would have been likely. It is also possible that the lugs were bitten off as a result of trismus, with or without the pulling from the air hose. However, what is apparent is that there was no standby diver to assist him. There also appears to have been no effective equipment or procedure for rapidly extracting an unconscious diver from

the water. This caused substantial delays to the initiation of resuscitation attempts, making recovery highly unlikely. In addition, there was no oxygen resuscitation capability, unacceptable in such a setting. An AED would likely have made little difference to the outcome in this incident largely due to the delay to retrieval.

The company was charged with and pleaded guilty to failing to maintain a safe workplace environment and was fined $60,000 as a result. This was largely based on the absence of appropriate systems to identify and promptly recover a distressed diver. They were not charged with causing the death of the victim. Improved initial training, better supervision and communication (including the presence of a stand-by diver); appropriate and practiced diver extraction protocols and relevant equipment; the wearing of BCDs; and ready access to suitable oxygen resuscitation equipment and an AED would reduce the likelihood of such an incident and improve the chances of successful rescue.

Summary: 22 y.o. male; absent pectoralis major (Poland syndrome); experienced recreational diving instructor; novice commercial pearl diver; second day of drift diving for oysters; previous incidents of separation from line and distress; poor supervision and no stand-by diver; surfaced and called for help then sank; long delays in rescue; drowning?/CAGE?

Discussion

A summary of the possible sequence of events in each of these incidents is shown in Table 3.

DEMOGRAPHICS

Once again, older males were over-represented in this series. There was only one female victim in each of the snorkelling and compressed gas diving cohorts. The mean (SD) age of the snorkellers was 55 (19) with a median (interquartile range, IQR) of 63 (37, 68). The compressed gas group were younger with a mean (SD) age of 42 (13) and median (IQR) of 41 (32, 48). Eight of the 14 snorkellers were overseas visitors compared to only three of the 11 scuba divers. A large proportion of the snorkel-related deaths were in Queensland, consistent with the high number of snorkellers on the GBR, many of them overseas tourists.

MEDICAL CONDITIONS

Associated with increased age is an increase in co-existing disease, both known and occult.[15–18] Six of the snorkellers and at least two of the scuba divers had a known medical condition which could have affected their fitness to snorkel or dive. These conditions included hypertension, ischaemic heart disease, diabetes, atrial fibrillation and epilepsy. However, three of the snorkellers and five of the scuba divers had no significant medical history, and the history was unknown in one of the snorkel and two of the scuba victims. In five of the snorkelling incidents and two of the scuba incidents, the pre-existing condition was believed to have contributed to the death.

Epilepsy

Although there remains some debate about the safety of diving for those with a history of (apparently) controlled epilepsy,[19] there is no doubt that a diver or snorkeller is exposed to a variety of potential triggers. With his poor compliance with medication, recent intake of marijuana and likely extended apnoea, BH 12/06 put himself at great risk. All epileptics need to be closely watched in aquatic situations, and, with no direct oversight and the associated opportunity for a swift rescue, the outcome is unsurprising.

Failure to declare

It is not uncommon for intending snorkelling or scuba participants to fail to declare current or past medical conditions; this was the case in at least two snorkelling and one scuba incident (BH 12/02, BH 12/05 and SC 12/04). This failure may at times result from a poor briefing by the operator, and/or inattention or misunderstanding by the intended participant, or simply due to time pressures. However, some individuals intentionally withhold information for fear that they will be prevented from participation. Given his medical background and his significant health issues, it appears likely that SC 12/04 would have been aware of an increased risk and may have intentionally withheld information from the dive operator in order to continue with his diving. A candid consultation with a diving physician would have provided the opportunity for an independent risk assessment and might have prevented this fatality.

Had the snorkellers declared their conditions, it is likely that increased supervision would have been offered or insisted upon. However, these events might still have occurred despite closer supervision and, given that some of the rescues often appear to have been relatively prompt, the outcomes might have been the same. Although sometimes appropriate and even necessary for the safety of the individual and/or others, it can be difficult for an operator to dissuade a keen customer from snorkelling or diving, as indicated in SC 12/09. It is worth noting that recent changes have been made to the Code of Practice relating to snorkelling in Queensland. These include strengthening the declaration process, a requirement for snorkellers identified as high risk (according to health or swimming ability) to use a flotation device, use of a clearly-marked snorkel and allocation to a buddy pair. The requirement for commercial operators to carry an AED has also been strengthened.[20] Although supportive of the changes, these authors have reservations about their impact in the absence of better-targeted medical screening and improved supervision, as discussed below.

Table 3
Chain of Events Analysis of snorkelling and compressed gas diving-related fatalities in Australian waters in 2012; CAGE – cerebral artery gas embolism; He – helium; IPE – immersion pulmonary oedema; OoG – out of gas

Case	Predisposing	Trigger	Disabling agent	Disabling injury	Cause of death
BH12/01	Planning	Unknown	Unknown	Asphyxia?/IPE?	Drowning
BH12/02	Health	Environmental/ Exertion	Medical	Cardiac incident?	Drowning
BH12/03	Health	Unknown	Unknown	Asphyxia?	Delayed drowning
BH12/04	Health	Environmental/ Exertion	Medical	Cardiac incident	Cardiac-related
BH12/05	Health	Environmental	Medical	Cardiac incident	Cardiac-related
BH12/06	Health/Planning	Environmental	Medical?	Asphyxia	Drowning
BH12/07	Supervision	Unknown	Unknown	Asphyxia	Drowning
BH12/08	Organisational	Environmental	Unknown	Unknown	Unknown
BH12/09	Planning/Activity	Extended apnoea	Apnoeic hypoxia	Asphyxia	Undetermined (Drowning?)
BH12/10	Equipment	Unknown	Unknown	Asphyxia?/ Cardiac incident?	Undetermined (Drowning?)
BH12/11	Organisational/ Experience/Skills	Exertion	Medical	Asphyxia	Drowning
BH12/12	Organisational/Planning & Supervision/Health?	Environmental/ Exertion	Medical	Cardiac incident	Drowning
BH12/13	Planning/Activity	Extended apnoea	Apnoeic hypoxia	Asphyxia	Drowning
BH12/14	Equipment/Experience/ Skills	Gas supply	Buoyancy	Asphyxia	Drowning
SC12/01	Equipment/Heath/ Supervision	Unknown (gas supply?)	Ascent?	CAGE?	Drowning
SC12/02	Health/Equipment/ Planning	Environmental/ Exertion?	Medical	Cardiac incident	Drowning
SC12/03	Health	Environmental/ Anxiety	Medical	Cardiac incident	Drowning
SC12/04	Health	Environmental	Medical	Cardiac incident/ SDPE?	Cardiac-related
SC12/05	None identified	Environmental	Gas supply	Asphyxia	Drowning
SC12/06	Equipment/Experience	Equipment	Environmental/Equip/ then OoG/Impact	Asphyxia	Drowning
SC12/07	Activity	Environmental	Environmental (shark attack)	Trauma	Trauma
SC12/08	Health	Gas supply	Equipment	Asphyxia	Drowning
SC12/09	Skills/Planning	Buoyancy/ Diver error	Gas supply/Buoyancy	Asphyxia?	Drowning?
SC12/10	Health	Environmental	Medical	Cardiac incident	Cardiac-related
SC12/11	Health/Activity	Gas supply	Gas supply	Asphyxia (He)	Asphyxia (He)
SS 12/01	Organisational/Training/ Experience/Supervision	Unknown	Unknown/Gas supply?/Ascent?)	Asphyxia?/ CAGE	Drowning

APNOEIC HYPOXIA

At least two (BH 12/09 and BH 12/13) and possibly three (BH 12/06) deaths appear to have been associated with apnoeic hypoxia. As is typical, these cases occurred in relatively young, experienced spearfishermen who were diving with others, but with poor oversight. The 'one-up, one-down' breath-hold buddy system was not used and in all cases body recovery was delayed. Unfortunately, the police reports gave no indication of whether or not the divers practiced pre-dive hyperventilation. This should be a routine question of buddies in such investigations as it can provide important educative information.

SEA CONDITIONS

Adverse sea conditions, in particular strong currents, were contributory in at least three snorkelling and two compressed gas fatalities. In some of these, the exertion involved probably precipitated a cardiac event. In others, it likely led to exhaustion with subsequent aspiration and drowning. All divers and snorkellers, even those with considerable experience, should never be complacent about the potential impact of a strong current and should carefully consider how to manage it if they proceed with the dive. Commercial operators need to be mindful of the conditions into which they permit their customers to snorkel or dive.

SUPERVISION

In at least two cases in this series (BH 12/08 and BH 12/11), inadequate supervision in commercial snorkel operations was identified as a concern. This is a recurring theme in these reports. With groups often ranging from 20–30 snorkellers or more to observe, it can be difficult for a lookout to recognise a problem, especially if they are relatively inexperienced, tired or distracted. There is a need to ensure that there are sufficient lookouts to effectively supervise an area, considering the size, shape and geographical features of the site, the prevailing conditions, the number of snorkellers and the effectiveness of the vantage point. These lookouts need to be adequately trained in appropriate observation and monitoring techniques,[21,22] remain vigilant at all times and be relieved at regular intervals to avoid fatigue or complacency.

Lifeguards patrolling pools or beaches are trained in methods to detect a swimmer in distress and these techniques are directly applicable to surface lookouts for snorkelling and diving activities and should be reviewed and adopted by the latter.[23] Prompt identification of a distressed or unconscious snorkeller, together with rapid rescue, will maximise the chances of survival. However, substantial delays in recognition do occur as it can sometimes be difficult to determine whether or not a motionless snorkeller is unconscious or just quietly observing the scene below. It is not uncommon for distressed and exhausted swimmers, snorkellers and divers not to wave or call for help. Lookouts need to be aware of this; it is better to have a high index of suspicion and run the risk of over-reacting. In addition, suitable rescue techniques need to be identified and practiced to ensure that they can be done swiftly and effectively when needed. Of interest, the use of flotation aids by snorkellers can be beneficial but incidents still occur, as evidenced in BH 12/11.

AED USE

AEDs were attached to at least seven of the snorkellers and one of the scuba victims. In six cases, the AED reported that there was no shockable cardiac rhythm. In one case, two belated shocks were advised and given, albeit unsuccessfully. There were no details about the AED advice or action in the other incident. In most cases, it is appears that the AED was attached at least 10 min after the victim was discovered unconscious, so the likelihood of successful defibrillation was low.[24] It would be valuable to collect data on AED use in the diving environment, including the likely timings from unconsciousness to the attachment of the AED, for police and other on-site investigators and details of the advice provided by the AED and any shocks given.

BUDDY SYSTEM

Consistent with many other fatality reports, the lack, or failure, of an effective buddy system may have influenced the outcome in at least six of the snorkelling and at least two of the scuba diving incidents. In some incidents separation was intentional while in others it was unintended. Buddy separation can often occur unintentionally underwater due to buoyancy issues, as evidenced with SC 12/09.

OVERWEIGHTING/BUOYANCY MANAGEMENT

Overweighting was likely a contributory factor in the deaths of BH 12/14 and SC 12/09. Both individuals were described as slightly-built, the weights worn seemed disproportionate and both had problems with managing their buoyancy. It is concerning that SC 12/09 had such poor buoyancy control skills after having completed 55 dives, as indicated by her uncontrolled ascent on the dive prior to her death and the problems described by the buddy on previous dives. Correct weighting and good buoyancy control are important tools for safe diving and need to be mastered early. They are especially important on deeper dives with the associated variations in buoyancy with depth.

EQUIPMENT

Faulty equipment in the form of a poorly assembled alternative air source was the trigger for the death of SC 12/06. It is likely that this relatively inexperienced victim assembled the regulator himself and failed to secure it properly. Although many divers assemble and adjust some components of their equipment, they need to have the

knowledge, skills and care to do this correctly. In the absence of these, it is better to take the gear to their dive shop and have it done by someone with appropriate expertise. Faulty gauges might have contributed to the deaths of SC 12/01 and SC12/08 who were both found with very little remaining air. It is unknown whether or not these divers were aware of their inaccurate gauges. Equipment-related problems are commonly reported to be associated with diving incidents, whether fatal or non-fatal.[25–29] The importance of having well-maintained and correctly functioning diving equipment is obvious.

IMMERSION PULMONARY OEDEMA (IPE)

IPE was discussed by the authors as a possible contributing factor or differential diagnosis in several of the above cases. However, a definitive diagnosis can be elusive in the absence of a clear clinical history, as autopsy findings can readily be attributed to cardiac disease or drowning.

LEFT VENTRICULAR HYPERTROPHY

Eight of the victims in this series were noted at autopsy to have LVH of varying degrees. Up to five of these may have been disabled by an arrhythmia. LVH is a known risk factor for sudden cardiac death and individuals with LVH have an increased incidence of serious arrhythmias.[5,30,31] The cardiovascular effects of immersion may increase the likelihood of an arrhythmia[32,33] and being in the hostile environment will reduce the chances of survival. In a comparative study of matched scuba diving and traffic accident victims, both heart mass and left ventricular wall thickness were greater in the scuba victims.[8] This may suggest that LVH is a risk factor for sudden death while diving. However, most studies on the physiological effects of immersion have been done on relatively young, fit individuals. Further research is needed on more elderly individuals with medical conditions and multiple medications.

Conclusions

There were 26 reported diving-related fatalities during 2012, 14 occurred while snorkelling and/or breath-hold diving and 11 while scuba diving; one diver died while using SSBA in a commercial pearl diving setting. Contributory or causal factors included pre-existing medical conditions (predominantly cardiac); poor organisation, planning and supervision; equipment-related problems; snorkelling or diving alone or with loose buddy oversight and apnoeic hypoxia. With snorkellers, the main disabling injuries were asphyxia and cardiac causes while, in compressed gas divers, asphyxia, cardiac causes and CAGE were identified. There were several cases where IPE was a possibility but there was insufficient evidence to confidently support this. LVH was noted at autopsy in almost one third of the victims, many of whom appeared to have been disabled by an arrhythmia. This is an area deserving further research. Factors that may reduce mortality include routine fitness-to-dive assessments in older divers by doctors with relevant training, improved supervision in commercial snorkelling operations; better assessment of the suitability of sea conditions; improved buddy oversight and avoidance of pushing breath-hold limits, especially without direct supervision with rapid response.

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Impact of various pressures on fracture resistance and microleakage of amalgam and composite restorations

Elnaz Shafigh[1], Reza Fekrazad[2,3], Amirreza Beglou[1]

[1] *Operative Dentistry Department, Faculty of Dentistry, AJA University of Medical Sciences, Tehran, Iran*
[2] *Department of Periodontology, Dental Faculty – Laser Research Centre in Medical Sciences, AJA University of Medical Sciences*
[3] *International Network for Photo Medicine and Photo Dynamic Therapy (INPMPDT), Universal Scientific Education and Research Network (USERN), Tehran, Iran*
Corresponding author: *Amirreza Beglou, Department of Operative Dentistry, AJA University of Medical Science, Daneshjou Boulevard, Evin, Tehran, Iran, PO 19839*
amir.beglou@gmail.com

Key words
Diving; Aviation; Dental; Barotrauma

Abstract
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Background: Pressure changes can influence dental restorations especially among divers. The aim of the current study was to evaluate the fracture resistance and microleakage of mesio-occluso-distal (MOD) amalgam and composite restorations undergoing pressure changes via diving and aviation simulation.
Methods: For the fracture resistance test, 60 sound maxillary premolar teeth were randomly allocated to two groups. Each group was then divided into three subgroups ($n = 10$) for simulating scuba-diving (pressure cycle to 203 kPa, 2 bar), flight (50.6 kPa, 0.5 bar), and control (atmospheric pressure). The teeth underwent pressure cycles for one month and then the fracture resistance test was conducted on them using the Instron testing machine. Microleakage scores were afterwards recorded by using a 2% methylene blue dye for 24 hours.
Results: Composite restorations showed significantly higher fracture resistance values compared to the amalgam group ($P < 0.05$). The control group had significantly higher fracture resistance values compared to the dive group, whereas there was no significant difference between the control group and the flight group ($P = 0.083$). No significant difference in the level of microleakage was observed between restoration materials or pressure cycles ($P > 0.05$).
Conclusions: Composite restorations showed promising fracture resistance compared to the amalgam group. Diving pressure cycles demonstrated adverse effects on the fracture resistance values of the two restorative materials.

Introduction

With the growing number of scuba divers and aircrew members, dentists will increasingly encounter oral conditions relating to pressure changes and these would require careful attention.[1,2] These phenomena are mainly related to the law of Boyle–Mariotte, which states that at a constant temperature the volume and pressure of an ideal gas are inversely proportional.[3,4] Among these oral conditions, barodontalgia is known as the toothache that is related to ambient pressure changes.[5–7] In a diving environment, this pain is commonly called 'tooth squeeze'. Although uncommon, in-diving or in-flight barodontalgia has been recognized as a potential cause of diver or aircrew-member vertigo and sudden incapacitation, jeopardizing the safety of diving or flight, respectively.[8] Odontocrexis is another condition describing tooth or restoration structure destruction associated with pressure changes.[9] Dental barotrauma describes the damage to tooth structure when pressure changes may occur with or without pain. All these conditions potentially may cause incapacitation that could jeopardize the safety of diving or flight.[8]

Defective dental restorations, leakage and secondary caries are assumed to be the most important predisposing factors of dental barotraumas. In-flight bruxism in aircrew members was reported to be the main factor of amalgam restoration failures in World War II.[10] Excessive bite forces were also proposed by the United States Air Force (USAF) symposium of aviation dentistry in 1946 as a predisposing factor for restoration dislodgment.[11] In divers, there is an argument about the effect of clenching on mouthpieces on the deterioration of dental restorations.[12] Based on our literature review, there appear to be no studies examining the effect of pressure changes on the properties of dental restorations.

This study aimed to assess the fracture resistance and microleakage of mesio-occluso-distal (MOD) amalgam and composite restorations undergoing pressure changes. The null hypothesis was that the pressure changes and dental

material used for restoring the tooth have no effect on the microleakage and fracture resistance of teeth via diving and aviation simulation.

Methods

SPECIMEN PREPARATION

A total of 60 sound single rooted mandibular premolars and 60 maxillary premolars, free of any microcracks and caries were extracted for orthodontic reasons within a three-month period and stored in normal saline solution at room temperature. Two weeks before use, all teeth were immersed in a 0.5% chloramine T trihydrate solution for infection control. Sixty maxillary premolar teeth of equal buccolingual dimension were used for the fracture resistance test and 60 mandibular premolars were used for the microleakage test. Care was taken to ensure that none of the teeth lost moisture. For each test, teeth were randomly divided into two groups, 30 teeth in each group, and treated as follows. Table 1 shows the groups and subgroups of this study.

Standard mesio-occluso-distal (MOD) cavities (a cavity on the mesial, occlusal, and distal surfaces of a tooth) were prepared using a coarse cylindrical flat-end diamond bur (MIC46078, Amalgadent, Australia). Each bur was changed after 10 preparations. The outline of the cavities was first drawn on the teeth using a digital calliper. The buccolingual widths of the cavities were considered half the inter-cuspal distance. The gingival margins of the cavities were placed 1mm above the cement-enamel junction (CEJ) with the pulpal floor 2 mm below the central groove. The depth of the axial wall was set at 1.5mm. The convergence of the buccal and lingual walls towards the occlusal was ensured. The cavosurface angle in all the walls was approximately 90 degrees.

In the amalgam groups, amalgam (SDI Ltd) was used according to the manufacturer's instructions to restore the teeth using the tofflemire (DS-DI-1166, Delmaks Surgio, Pakistan) retainer and a stainless steel (SS) matrix band. Filtek Z250 (3M ESPE, USA) was used to restore the teeth in the composite groups in accordance with the manufacturer's instructions. The teeth were etched for 15 seconds using 37% phosphoric acid; they were then rinsed for 10 seconds with water after which they were air dried until a shiny hydrated surface of moist dentin was achieved. Adper single bond II (3M ESPE, USA) was applied in two layers with disposable applicators, each layer was air dried for 5 seconds to ensure solvent evaporation and then light cured for 20 seconds with a light-emitting diode (LED) (650mW/cm2) (Optilux 501, Kerr, Danbury, CT, USA). Then, using the SS matrix bands and tofflemire, the teeth were restored with A2 shade composite resin. The oblique layering technique was performed with the first layer not thicker than 1 mm in gingival and pulpal floors. Following this, increments were placed in 2 mm thicknesses having contact with only two walls of the cavity. Each increment was light cured for 40 seconds from the occlusal surface. After removing the matrix band, additional curing was performed from buccal and lingual planes for 40 seconds each. Moreover, 24 hours after restoration, both amalgam and composite groups were finished and polished according to standard methods. All the procedures were performed by a single dentist (ARB) trained and experienced in the cavity preparation and filling procedures described above and blinded to which group teeth were to be allocated.

PRESSURE CHANGE SIMULATION

To simulate pressure changes during dives and flights, an experimental chamber was designed with an external manometer. Compressed air was used to increase the chamber pressure and a vacuum pump to decrease pressure. The speed of pressure change was set to 1 bar (101.3 kPa) per minute. The diving descent was simulated by increasing the pressure to 203 kPa (2 bar) roughly equivalent to a depth of 10 metres under water. Decreasing the pressure to 0.5 bar (50.6 kPa) is equivalent to 5,500 m above sea level. Each tooth underwent 30 simulated dives (D) or flights (F) according to their subgroups. For the dive subgroups, the teeth were maintained in 203 kPa pressure for 45 minutes each day before returning to ambient pressure. For the flight subgroups, the teeth were de-pressurised to 50.6 kPa for 45 minutes in the same manner. The control subgroups were stored at ambient pressure for one month.

FRACTURE RESISTANCE TEST

All specimens were mounted in self-cure acrylic resin up to 2 mm below the CEJ. A dental surveyor was employed to ensure uniform alignment of all specimens parallel to the analysing rod. All specimens were then placed in a jig, which allowed loading at the central fossa parallel to the long axis of the teeth. The Instron universal testing machine (Z010, Zwick GmbH, Ulm, Germany) was used to deliver compressive load at a crosshead speed of 1 $mm \cdot min^{-1}$ until fracture. The fracture resistance scores were recorded in Newtons (N).

MICROLEAKAGE TEST

The entire tooth surface was covered with two layers of nail polish, except for the restoration and a 1 mm margin around it on the tooth surface. The root apices were sealed with sticky wax. The specimens were then immersed in 2% methylene blue for 24 hours and rinsed under running water to remove excessive dye. The teeth were subsequently sectioned mesio-distally with a water-cooled low-speed saw (TC-3000, Vafaei Industrial Co., Tehran, Iran). Two sections of each specimen were examined under the stereomicroscope at 16X magnification. Dye penetration was quantified in gingival margins of the restoration using a 0–3 scale system, where 0 – no dye penetration, 1 – dye penetration limited

Table 1
Experimental groups and subgroups

Groups (*n*)	**Subgroups** (abbreviation) (*n*)
Amalgam restorations (30)	Flight (FAF) (10)
	Diving (FAD) (10)
	Control (FAC) (10)
Composite restorations (30)	Flight (FCF) (10)
	Diving (FCD) (10)
	Control (FCC) (10)
Amalgam restorations (30)	Flight (MAF) (10)
	Diving (MAD) (10)
	Control (MAC) (10)
Composite restorations (30)	Flight (MCF) (10)
	Diving (MCD) (10)
	Control (MCC) (10)

Figure 1
Boxplots of fracture resistance (Newtons) in hypobaric, normobaric and hyperbaric pressures based on the material group (amalgam/composite). Differences in the Amalgam group not significant, whereas in the composite group, the most fracture resistance was observed in the normobaric group ($P = 0.034$, ANOVA)

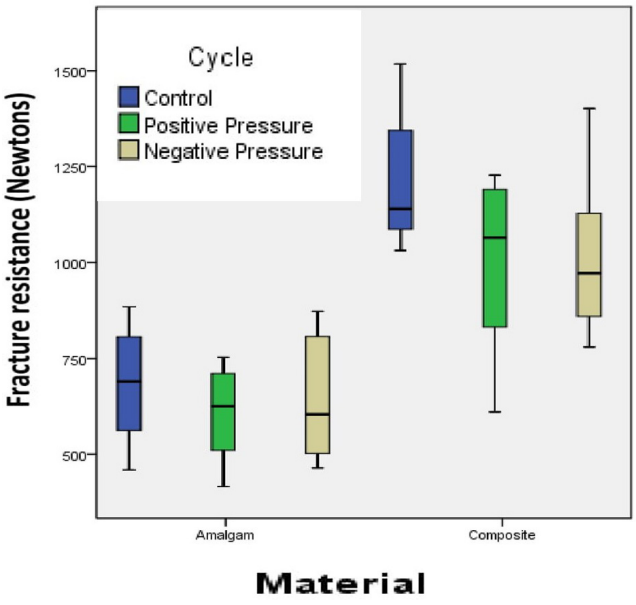

Table 2
* The abbreviations represented in Table 1; ** Microleakage scores refer to a dye penetration scale; 0 – no dye penetration; 1 – dye penetration limited to the enamel of the gingival wall; 2 – dye penetration into the dentin in the gingival wall; 3 – dye penetration past the gingival wall and involving the axial wall

Subgroups (*n*)	**Microleakage scores**** (*n*)			
	0	**1**	**2**	**3**
MAF* **(10)**	2	2	6	0
MAD (10)	1	0	8	1
MAC (10)	2	1	6	1
MCF (10)	2	6	1	1
MCD (10)	1	4	3	2
MCC (10)	3	5	2	0

to the enamel of the gingival wall, 2 – dye penetration into the dentin in the gingival wall, 3 – dye penetration past the gingival wall involving the axial wall. The highest scores were recorded.

STATISTICAL ANALYSIS:

Data analysis was carried out using SPSS software (SPSS version 18.0, SPSS, Chicago, IL, USA). In order to compare the fracture resistance and microleakage amounts between groups Kruskal–Wallis and Mann-Whitney tests were used.

Results

FRACTURE RESISTANCE

The normality of distribution of the data was confirmed using Shapiro-Wilk test ($P > 0.05$). The mean values of fracture resistance of the groups are shown in Figure 1. Both the materials used ($P < 0.001$) and pressure change cycles ($P = 0.027$) had significant effects on fracture resistance amounts. Composite restorations showed significantly higher fracture resistance values compared to the amalgam group ($P < 0.001$). A pairwise comparison of the cycles demonstrated that the control group had significantly higher fracture resistance values ($P = 0.034$) in comparison with the dive groups, whereas there was no significant difference between the control group and the flight group ($P = 0.083$).

MICROLEAKAGE

The microleakage scores of different groups are shown in Table 2. There were no significant differences in the microleakage amounts among the six subgroups (Kruskal–Wallis test, $P = 0.076$; Figure 1). In both the amalgam and composite groups there were no statistically significant differences ($P = 0.341$ for amalgam groups and $P = 0.228$

for composite groups, respectively). The Mann-Whitney test also revealed no significant differences between the C, D, and F groups.

Discussion

To the best of our knowledge, this is the first investigation that has assessed the effect of pressure changes on the mechanical properties of teeth restored with different restorative materials. Amalgam and composite materials were selected because they are the most frequently used. It is reported by various authors that among a variety of predisposing factors for barodontalgia and odontocrexis, leaking restorations rather than caries are of great importance.[6,11] On the other hand, excessive bite forces applied to teeth from clenching and bruxism during flights and dives are also reported to be a crucial factor in tooth destruction.[13–15] Therefore, the effects of pressure changes and restorative materials were examined in this study by means of fracture resistance and microleakage.

Regarding the fracture resistance test, the results revealed that teeth restored with amalgam in all groups were significantly more prone to fracture compared to composite groups. This is in accordance with previous studies which reported on the effect of cusp reinforcement by means of adhesive dentistry.[16–18] On the other hand, the fracture resistance of teeth restored with composite restorations is still significantly lower than intact teeth and composite restorations are not able to fully restore the mechanical properties of teeth.[18,19] Also, it is worth mentioning that different clinical conditions like thermocycling can have adverse effects on the reinforcement impact of adhesive restorations.[20,21]

The normal biting force on maxillary premolars has been observed to be 100–300 N.[21] In our study, none of the groups have shown fracture resistance values lower than these amounts, and even the lowest group (amalgam in diving simulation) showed a mean fracture resistance value of 622 N. Although the clinical conditions and the forces applied to the teeth in the oral cavity are different from the design of this study, these numbers do have clinical relevance.

There remain concerns regarding the fact that patients are having clenching or bruxism, especially in amalgam restorations, as occlusal forces have been reported to be as high as 520–800 N.[21] The weakening effect of clenching on tooth structures in pilots and divers has been noted by different studies.[12,13] Researchers have reported aircrew members and divers to have a higher prevalence of jaw parafunctional activity.[6,11] It was estimated that 60–70% of pilots in World War II had suffered from bruxism, whilst more recently, the prevalence of clinically important bruxism in a military environment occurred in 69% of aircrew members.[13] In scuba divers, there is an argument that clenching on the mouthpiece during diving increases in cold water and with stress and this may contribute to the deterioration of dental restorations.[12] Higher prevalence of clenching in scuba divers has been reported in other studies.[6,14] Owing to the higher prevalence of jaw parafunctional activities in aircrew members and divers and its subsequent weakening effect on tooth structures, the use of amalgam restorations seems to be controversial regarding fracture resistance.

In both divers and pilots the air void trapped in a dental restoration expands according to Boyle's law during each ascent owing to the decrease in pressure and weakens the restoration structure.[9,22] This explains the fracture resistance decrease in both flight and dive groups compared to control groups in the current study. However, this reduction was only statistically significant in dive groups, not in the flight groups, probably related to the greater range of pressure change in the dive groups compared to the flight groups (1bar versus 0.5 bar pressure changes).

The microleakage test showed no statistically significant differences among the different groups, whereas in a previous study, higher microleakage was reported with amalgam than with posterior composite resin.[23] Similarly, in another study, the microleakage of amalgam restorations in primary molars had more leakage compared to the composite restorations at the occlusal margins. On the other hand, the same study revealed no significant difference between amalgam and composite restorations in cervical margins, which is consistent with the present results.[24] In contrast, composite restorations demonstrated higher microleakage than amalgam restorations in a study which evaluated microleakage in both *in vivo* and *in vitro* around Class I restorations, though our findings in control groups were similar to their control results.[25]

Among different techniques for microleakage test, dye penetration is the most widely used method to assess microleakage because of its sensitivity, ease of use, and convenience,[26] and stereomicroscopic examination at 16 × magnification was chosen for this study as this provides a well-magnified two-dimensional view of the surface to be examined.

These contradictory results may be because of the variations in leakage evaluation techniques, test conditions, cavity design and dimensions, restorative materials, type of teeth and observation time and underline the obvious importance of standardized testing parameters for leakage studies.[25] The use of composite restoration is suggested for divers and aircrew members as it showed superior fracture resistance values compared to amalgam.

Conclusions

Composite restorations showed promising fracture resistance compared to amalgam. Diving pressure cycles demonstrated adverse effects on the fracture resistance values of both

restorative materials. Despite the experimental conditions of this study not fully mimicking the conditions of the oral cavity during diving or flight experiences, as teeth are subjected to a mixture of different factors outside the laboratory setting, some important clinical relevance can still be inferred. This is a pioneer study and further studies with different pressure cycles, longer durations, different restorative materials and different tests are required to fully understand the effect of ambient pressure changes on tooth structures and their restoration.

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Meta-analysis on the effect of hyperbaric oxygen as adjunctive therapy in the outcome of anastomotic healing of experimental colorectal resections in rats

Robin J Brouwer[1], Alexander C Engberts[1], Boudewijn LS Borger van der Burg[1], Thijs TCF van Dongen[1,2], Rob A van Hulst[3,4], Rigo Hoencamp[1,2,5]

[1] *Department of Surgery, Alrijne Hospital, Leiderdorp, The Netherlands*
[2] *Defense Healthcare Organization, Ministry of Defense, Utrecht, The Netherlands*
[3] *Department of Anesthesiology, Amsterdam Medical Center, Amsterdam, The Netherlands*
[4] *Maritime Medical Expertise Center, Diving Medical Center, Royal Netherlands Navy, Den Helder, The Netherlands*
[5] *Department of Surgery, Leiden University Medical Center, Leiden, The Netherlands*
Corresponding author: *RJ Brouwer, Department of Surgery, Alrijne Hospital, Simon Smitweg 1, 2353 GA Leiderdorp, The Netherlands*
rjbrouwer@alrijne.nl

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Surgery; Gastrointentestinal tract; Animal model; Hyperbaric research; Systematic review

Abstract
(Brouwer RJ, Engberts AC, Borger van der Burg BLS, van Dongen TTCF, van Hulst RA, Hoencamp R. Meta-analysis on the effect of hyperbaric oxygen as adjunctive therapy in the outcome of anastomotic healing of experimental colorectal resections in rats. Diving and Hyperbaric Medicine. 2018 September;48:173–185. doi: 10.28920/dhm48.3.173-185. PMID: 30199890.)
Background: Colorectal cancer is the third most common form of cancer and colorectal surgery is the treatment of choice in local disease. Anastomotic leakage following colorectal surgery is a major complication with a high incidence and mortality. Adjuvant hyperbaric oxygen treatment (HBOT) may be associated with reduction of anastomotic leakage. A systematic review was conducted regarding HBOT as an adjunctive therapy to colorectal surgery.
Methods: Systematic review (1900–2017) using PubMed, Cochrane, EMBASE, Web of Science and EMCARE. All original published studies on the effect of HBOT as an adjunctive therapy for colorectal surgery with the creation of an anastomosis were considered.
Results: Thirteen small animal trials were included for qualitative synthesis. We found no human trials. Eleven trials used bursting pressure whilst eight used hydroxyproline levels as a marker for collagen synthesis as primary outcome to assess the strength of the anastomosis. A meta-analysis performed for normal and ischaemic anastomoses showed that postoperative HBOT improves bursting pressure and hydroxyproline levels significantly in both normal ($P \leq 0.001$ and $P = 0.02$ respectively) and ischaemic anastomoses ($P \leq 0.001$ and $P = 0.04$ respectively).
Conclusion: Postoperative HBOT has a positive effect on colorectal anastomoses in rats. Further research should focus on a larger systematic animal study.

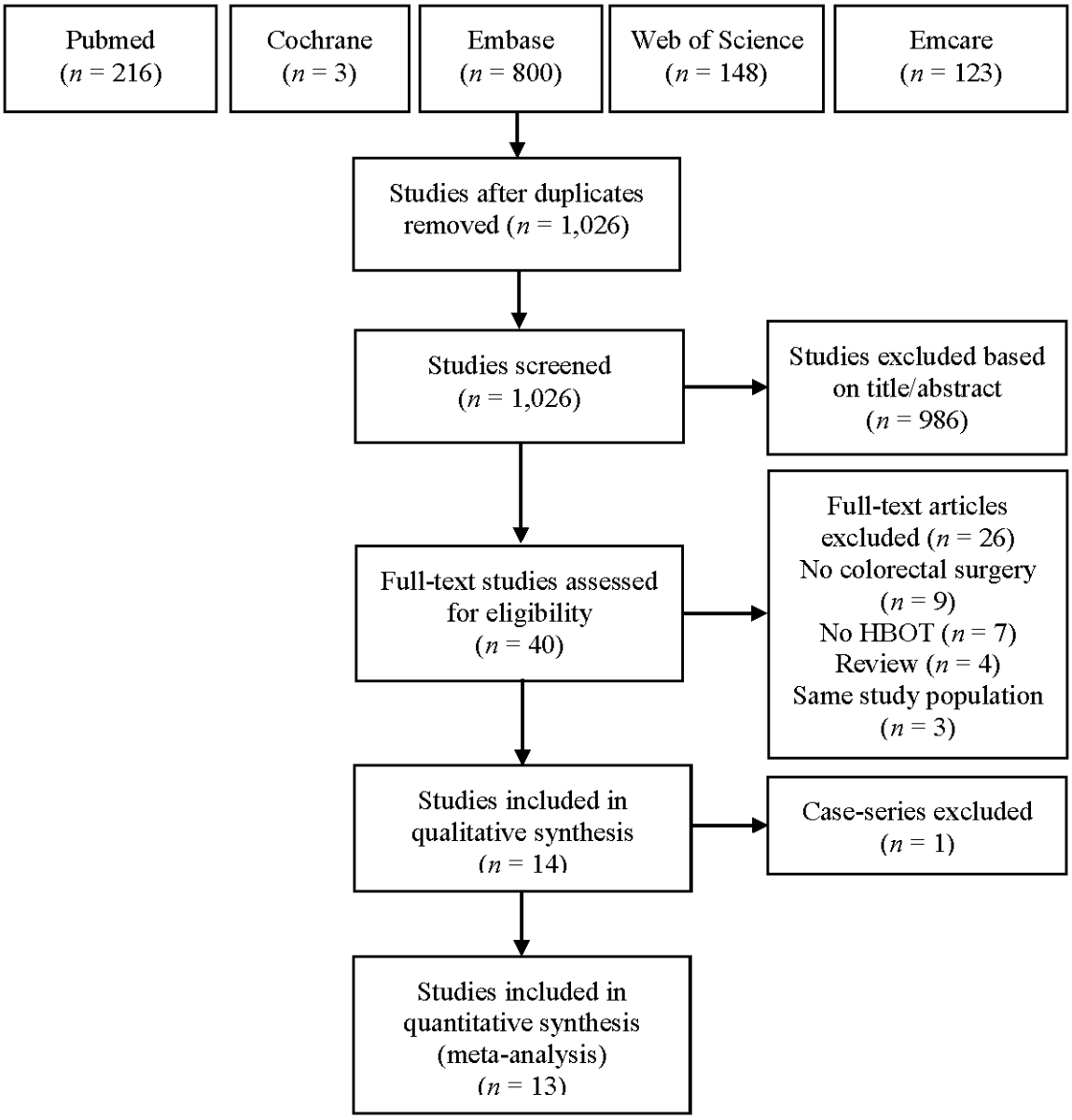
Introduction

Colorectal cancer is the third most common form of cancer with an incidence of almost 1.4 million cases in 2012 according to the WHO.[1] Colorectal surgery is the treatment of choice in local carcinoma.[2,3] A major complication following colorectal surgery is anastomotic leakage (AL) with a reported incidence of 10–13% and a mortality of up to 33%.[4] A recent meta-analysis showed that AL is associated with local recurrence and reduced survival.[5] Hyperbaric oxygen treatment (HBOT) has been suggested as adjunct therapy to reduce the risk of AL.

HBOT involves breathing 100 percent oxygen at two to three times normal atmospheric pressure and results in elevated oxygen tension in arteries and tissue.[6] HBOT is already being used widely as a treatment for a variety of indications as set out in published recommendations of the Undersea and Hyperbaric Medical Society and the European College of Hyperbaric Medicine.[7,8] HBOT has a variety of mechanisms of action: it improves tissue oxygenation; inhibits the pro-inflammatory reaction by reducing cytokines; improves neo-vascularization; has a bacteriostatic effect on anaerobic bacteria and stimulates stem cells and growth factors.[9] HBOT is considered a low-risk therapy. Described side effects are middle ear barotrauma (up to 43%, usually mild), myopia, aerosinusitis, (acute and chronic) oxygen poisoning including seizures and lung failure.[10,11]

Preconditioning with HBOT might be useful as an adjunct for various types of surgery. For instance, a better outcome in left ventricular function was demonstrated

Figure 1
PRISMA flow chart for meta-analysis



after on-pump coronary artery bypass surgery after pretreatment with three HBOT sessions,[10] whilst in patients undergoing pancreaticoduodenectomy, a single preoperative HBOT appeared to improve outcome.[12] Furthermore, preconditioning with HBOT is associated with a reduction in the interleukin inflammatory markers IL-6 and IL-10.[12]

The effect of HBOT on cancer depends highly on the type of cancer; it might even have an inhibitory effect on certain types of cancer.[13] The current consensus is that there is no scientific evidence that HBOT has a cancer-promoting effect.[13,14] including in colorectal cancer.[15] The latter study concluded that HBOT does not promote the growth or recurrence of colorectal cancer, but that treating colorectal cancer solely with HBOT does not seem to have a beneficial effect.

Although strong evidence is still lacking, HBOT could potentially be an adjunct in the treatment of colorectal cancer. The primary aim of this systematic review and meta-analysis is to provide the best evidence to date regarding the effects of HBOT as an adjunctive therapy on anastomotic healing after colorectal surgery.

Methods

The protocol for objectives, literature search strategies, inclusion and exclusion criteria, outcome measurements, and methods of statistical analysis was prepared *a priori*, according to the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) statement,[16,17] and is described in this section.

LITERATURE SEARCH STRATEGY

A systematic review (1900–2017) was performed in PubMed, Cochrane, EMBASE, Web of Science and EMCARE. The keywords used in the search were "*hyperbaric oxygenation*" and its synonyms in combination with "*colorectal surgery*", "*colectomy*" and their equivalents. Also, the combination of "*surgery*" and its synonyms, with "*colon*", "*rectum*", "*sigmoid*" and their equivalents was used. The search was limited to original studies published in English.

Inclusion and exclusion criteria, data extraction and outcomes of interest

Two authors (RJB, ACE) independently identified the studies for inclusion and exclusion and extracted the data. The accuracy of the extracted data was further confirmed by a third author (RH). Studies were included when they used colorectal surgery, including the formation of an anastomosis, in combination with HBOT.

QUALITY ASSESSMENT

The quality of trials was assessed using the Systematic Review Center for Laboratory Animal Experimentation (SYRCLE) risk-of-bias tool.[17] This tool is designed to assess bias in animal studies and contains ten items to investigate bias in selection, performance, detection, attrition and reporting. Ten points are scored for every item complied with. No points are awarded when the study does not meet the criterium or when documentation is unclear. The total score ranges from 0 to 100 with 0 being the worst, with a high chance of bias, and 100 being the best score, seemingly free from bias.

OUTCOME MEASURES

The main outcome measures of the included studies were bursting pressure (BPR) and hydroxyproline levels (HP). BPR involves a measurement whereby air is instilled in a closed segment of bowel with the anastomosis, and established by means of a sudden decline in pressure or visualization of air bubbles in a submerged anastomosis. Hydroxyproline is formed during the synthesis of collagen and has proven to be a good predictor for AL.[18] Other outcomes measured in some studies were histopathological analysis (HA), various biochemical analyses and the total energy of rupture biomechanical test (ETR).

STATISTICAL ANALYSIS

The software package Review Manager 5.3,[19] was used to perform a meta-analysis of the primary outcome sources, which was determined after careful study of the results. An inversed variance test was used for the meta-analysis. In all cases, $P < 0.05$ (two-sided) was considered statistically significant.

Results

The PRISMA literature search and study selection are shown in Figure 1. Thirteen animal trials were included for qualitative and quantitative synthesis (Table 1).[4,20–31], Appendix A* identifies where each item in the PRISMA checklist may be found in this report. In additon, Appendix B* presents the full electronic search strategy such that it could be repeated.

STUDY PROTOCOLS

All animal trials reported the effect of HBOT on colonic anastomoses in rats (Table 1). Ten studies[20–22,24–29,31] used postoperative HBOT, one study used a combination of pre- and postoperative (combined) HBOT,[4] one had two study groups researching postoperative and combined HBOT[30] and the last study had three study groups analyzing preoperative, postoperative and combined HBOT.[23] All studies performed open surgery with one exception which used a laparoscopic technique.[30] There is wide variation in the HBOT protocols in terms of the treatment intervals, durations of treatment, length of the HBOT courses and pressures.

QUALITY ASSESSMENT

The quality assessment using the SYRCLE tool is shown in Table 2. None of the studies met all quality criteria. Six studies[4,24,26,29–31] randomized the study and control groups, but none of the studies provided baseline statistics, potentially concealing selection bias. None of the studies randomly selected the animals for outcome assessment or described blinding the outcome assessor. In only one study were the investigators blinded.[30] In all but one[23] of the seven studies that included pathologic analysis,[4,23,24,27–29,31] the outcome assessor for the analysis was blinded, decreasing the chance of detection bias. Overall, the included studies generally lacked steps in their protocols to minimize the chance of (any kind of) bias.

NON-ISCHAEMIC ANASTOMOSIS

Ten[4,20,21,23,25,27–31] of the thirteen studies focused on non-ischaemic anastomosis in normal conditions (Table 3). One study used three study groups – preoperative, postoperative and combined HBOT,[22] whilst another used two study groups – postoperative and combined HBOT,[29] resulting in a total of thirteen different study groups. Of these thirteen study groups, five study groups from five different studies reported a significant improvement of the anastomosis after HBOT

*** Footnote:** Follow this link to Appendices A and B. Alternatively these files may be obtained from the corresponding author at: rjbrouwer@alrijne.nl

Table 1

Study protocol of included animal trials; HBOT – hyperbaric oxygen therapy; M – male; F – female; d – days; ATA – atmosphere absolute; CRT – chemoradiotherapy; CT – Chemotherapy; GH – growth hormone; BPR – bursting pressure; HP – hydroxyproline; PPg-glucan – poly B1-6 glucotriosys B1-3 glucopyranose glucan; PA – pathological analysis; MMP – matrix-metalloproteinase; BA – biochemical analysis; ETR – total energy of rupture biomechanical test

Author	**n**	**Species/ sex**	**Intervention**	**Pre/postop HBOT**	**HBOT** days / length (min) / pressure (kPa)	**Additional interventions**	**Measured outcomes**
Boersema[4]	10	Wistar, M	Colectomy	Pre- and postop	7 preop + 3 postop / 1 x 90 / 243	None	BPR+PA
Hamzaoğlu[20]	10	Wistar, M	Left colon resection	Postoperative	4 / 1 x 60 / 253	Induced ischaemia	BPR+HP
Erenoglu[21]	10	Wistar, M	Colectomy	Postoperative	7 / 2 x 90 / 203	Preoperative CRT	BPR+HP
Guzel[22]	10	Wistar, F	Induced ischaemia + colonic anastomosis	Postoperative	4 / 1 x 60 / 253	PPg-glucan	BPR+HP
Yagci[23]	10	Wistar, M	Left colon resection	Preoperative	2 / 2 x 90 / 284	Induced ischaemia	BPR+HP+PA
Yagci[23]	10	Wistar, M	Left colon resection	Postoperative	4 / 2 x 90 / 284	Induced ischaemia	BPR+HP+PA
Yagci[23]	10	Wistar, M	Left colon resection	Pre- and postop	2 preop + 4 postop / 2 x 90 / 284	Induced ischaemia	BPR+HP+PA
Sucullu[24]	8	Wistar, M/F	Induced peritonitis + colectomy	Postoperative	3 or 7 / 1 x 90 / 253	None	BPR+HP+PA
Azevedo[25]	10	Wistar, ?	Colectomy	Postoperative	7 / 1 x 90 / 203	Induced ischaemia	HP+MMP1+MMP9
Rocha[26]	15	Wistar, F	Induced peritonitis + colectomy	Postoperative	4 / 1 x 120 / 203	None	ETR
Adas[27]	10	Wistar, M	Left colon resection	Postoperative	4 / 3 x 60 / 253	GH	BPR+PA
Kemik[28]	10	Wistar, F	Left colon resection	Postoperative	4 / 4 x 80 / 253	CT	BPR+PA
Yildiz[29]	12	Wistar, F	Left colon resection	Postoperative	5 / 2 x 90 / unknown	Preoperative CRT	BPR+HP+PA
Poyrazoglu[30]	7	Sprague-Dawley, M	Left colon resection	Postoperative	4 / 1 x 120 / 284	None	BPR+BA
Poyrazoglu[30]	7	Sprague-Dawley, M	Left colon resection	Pre- and postop	2 h preop + 4 postop / 1 x 120 / 284	None	BPR+BA
Emir[31]	10	Wistar, M	Laparoscopic left colon resection	Postoperative	10 / 1 x 60 / 213	None	BPR+HP+PA

Table 2

Quality assessment using the SYRCLE risk of bias tool; 1 = yes, 2 = no, 3 = unclear; 10 points are scored for every item complied withand. No points are awarded when the study does not meet the criterium or when documentation is unclear; total score ranges from 0–100, 0 being the worst, with a high chance of bias, and 100 being the best score, seemingly free from bias

Author	SYRCLE tool[17] question number													
	1	2	3	4	5	6	7	8	9	10	Yes	Unclear	No	Score
Hamzaoglu[20]	3	3	3	2	3	3	3	1	1	2	2	2	6	20
Erenoglu[21]	3	3	3	2	3	3	3	1	1	2	2	2	6	20
Guzel[22]	2	3	2	1	2	3	3	1	1	2	3	4	3	30
Yagci[23]	3	3	3	1	3	3	3	1	1	2	3	1	6	30
Sucullu[24]	1	3	3	1	3	3	3	1	1	2	5	1	4	50
Azevedo[25]	3	3	3	1	3	3	3	1	3	2	2	1	7	20
Rocha[26]	1	3	3	1	3	3	3	1	3	2	3	1	6	30
Adas[27]	3	3	3	1	3	3	3	1	1	2	3	1	6	30
Kemik[28]	3	3	3	1	3	3	3	1	1	1	4	0	6	40
Yildiz[29]	1	3	3	1	3	3	3	1	1	2	4	1	5	40
Poyrazoglu[30]	1	3	3	2	3	3	3	1	1	2	3	2	5	30
Boersema[4]	1	3	3	2	3	3	3	1	1	1	4	1	5	40
Emir[31]	1	3	1	1	1	3	3	1	1	2	6	1	3	60

treatment.[4,20,25,27,30] In the other eight study groups, analyzed by six different studies, any observed improvement of the anastomosis did not reach statistical significance.[21,23,28–31] There was no association between HBOT and anastomosis strength in the study groups assessing preoperative or combined HBOT.[4,23,30]

The BPR was measured in twelve study groups, from nine different studies, and was higher in the HBOT group in all study populations.[4,20,21,23,27–31] A significant increase of BPR was observed in three study groups, analysed by three different studies.[20,27,30] HP was measured in ten study groups from seven different studies.[20,21,23,25,29–31] Of these ten study groups, HP was significantly higher in seven study groups, analysed by five different studies.[20,21,23,25,30] There was a marked variation in HP levels (Table 3). Six studies[20–22,25,30,31] measured HP in grams in tissue, while two studies[23,29] measured HP molarity in tissue. One study[22] measured HP in wet tissue, whilst another[31] dried the tissue for 24 hours before analysis. The remaining six studies[20,21,23,25,29,30] measuring HP did not describe how they prepared the tissue for analysis.

HISTOPATHOLOGICAL ANALYSIS

The histopathological analysis varied between studies. Three studies assessed anastomotic line fibrosis and found no significant difference between any groups.[23,24,29] Another assessed the formation of a mucosal layer and the severity of inflammation at the anastomosis and found no significant differences.[30] Three studies found a significant increase in neovascularization in the HBOT group.[4,27,28] The same three studies assessed collagen deposition, but only one found a significant increase in collagen deposition in the HBOT group.[26] No significant differences were found in necrosis, epithelialization or granulation.[26,27] All tissue biochemical markers changed in the study group that received only postoperative HBOT.[29] Malondialdehyde (MDA), an indicator of fat oxidation, and myeloperoxidase, an indicator of inflammation, were lowered and superoxide dismutase and glutathione peroxidase, both indicators of the antioxidant response, were elevated.[29] In the study group that received both pre- and postoperative HBOT, only MDA was significantly lower.[29] In another study measuring nitric oxide, MDA and catalase in serum and tissue, a significant decrease was demonstrated only in serum MDA in the HBOT group.[30]

ISCHAEMIC ANASTOMOSES

Seven study groups from five different studies assessed the influence of HBOT on ischaemic anastomoses (Table 4).[20,22,23,25,27] In six groups from the five studies, HBOT had a positive effect on the anastomosis. The only exception was

Table 3

Outcome of studies assessing normal anastomoses; HBOT– hyperbaric oxygen reatment; Prob. – probablility; ALF – anastomotic line fibrosis; U – unknown; MMP – matrix-metalloproteinase; ns – not significant; ↑ – significantly increased; ↓ – significantly decreased; NV – neovascularization; CD – collagen deposition; N – necrosis; E – epithelialization; G – granulation; FML – formation of mucosal layer; SI – severity of inflammation; tMDA – tissue malondialdehyde; tMPO – tissue myeloperoxidase; tSOD – tissue superoxide dismutase; tGSH-Px – tissue glutathionperoxidase; sMDA – serum malondialdehyde; sNO – serum nitric oxide; sCAT – serum catalase; tNO – tissue nitric oxide; tCAT – tissue catalase

Author	Bursting pressure (mmHg)			Hydroxyproline				Pathology	Other	Improved
	HBOT	Control	P ≤ 0.05	HBOT	Control	Units	P < 0.05			
Hamzaoğlu[20]	123 ± 18.4	104 ± 18.9	Yes	10.12 ± 4	7.4 ± 2	mg·mg tissue^{-1}	Yes	–	–	Yes
Erenoglu[21]	221 ± 6.05	190.2 ± 18.14	No	22.88 ± 2.38	9.01 ± 2.04	µg·10 mg tissue^{-1}	Yes	–	–	No
Yagci[23]	115.5 ± 21.1	107.2 ± 37.5	No	13.89 ± 3.43	9.95 ± 2.65	µM·mg tissue^{-1}	Yes	ALF ns	–	No
Yagci[23]	113.6 ± 16.9	107.2 ± 37.5	No	13.11 ± 4.39	9.95 ± 2.65	µM·mg tissue^{-1}	Yes	ALF ns	–	No
Yagci[23]	119.2 ± 16.7	107.2 ± 37.5	No	13.25 ± 3.27	9.95 ± 2.65	µM·mg tissue^{-1}	Yes	ALF ns	–	No
Azevedo[25]	–	–	–	Unknown	Unknown	µg·mg tissue^{-1}	Yes	–	MMP1 ns; MMP9 ns	Yes
Adas[27]	93.3 ± 20.5	81.4 ± 20.1	Yes	–	–	–	–	CD↑; NV↑; N ns; E ns; G ns	–	Yes
Kemik[28]	93.4 ± 24.8	84.5 ± 24.4	No	–	–	–	–	NV↑; CD ns; N ns; E ns; G ns	–	No
Yildiz[29]	133.7 ± 29.7	110.7±18.6	No	17.3 ± 6.6	15 ± 5.8	µM·mg^{-1} tissue^{-1}	No	ALF ns	–	No
Poyrazoglu[30]	152.9 ± 18	122.6 ± 16.7	Yes	83 ± 11.1	60.4 ± 14.4	mg·g protein^{-1}	Yes	–	tMDA↓; tMPO↓; tSOD↑; tGSH-Px↑	Yes
Poyrazoglu[30]	126 ± 13.6	122.6 ± 16.7	No	63.7 ± 18.7	60.4 ± 14.4	mg·g protein^{-1}	No	–	tMPO↓; tMDA ns; tSOD ns; tGSH-Px ns	No
Boersema[4]	162.4 ± 39.7	141.1 ± 73.3	No	–	–	–	–	NV↑; CD206+↑; M2/M1↑; CD ns; iNOS+ ns	–	Yes
Emir[31]	213 ± 27	197 ± 9.1	No	26.5 ± 4.1	26.8 ± 4.36	µg·10 mg tissue^{-1} (dry)	No	FML ns; SI ns	sMDA↓; sNO ns; CAT ns; tMDA ns; tNO ns; tCAT ns	No

Table 4

Outcome of studies assessing ischemic anastomoses; HBOT– hyperbaric oxygen treatment; ALF– anastomotic line fibrosis; ns – not significant; MMP – matrix-metalloproteinase; ↑ – significantly increased; ↓ – significantly decreased; CD – collagen deposition; NV – neovascularization; N – necrosis; E – epithelialization; G – granulation

Author	Bursting pressure (mmHg)			Hydroxyproline			Pathology	Other	Improved
	HBOT	Control	Prob. < 0.05	HBOT	Control	Units	Prob. ≤ 0.05		
Hamzaoğlu[20]	102.2 ± 14.8	77.5 ± 22.1	Yes	6.04 ± 2	4.76 ± 2	mg·mg tissue^{-1}	Yes	–	Yes
Guzel[22]	104 ± 19.4	69.5 ± 16.7	Yes	12.1 ± 1.1	4.3 ± 0.6	mg·100 mg wet tissue^{-1}	Yes	–	Yes
Yagci[23]	81.2 ± 9.2	79.3 ± 7.7	No	9.51 ±. 87	8.42 ± 2.1	µM·mg tissue^{-1}	No	ALF ns	No
Yagci[23]	97.9 ± 17.9	79.3 ± 7.7	Yes	10.01 ± 1.88	8.42 ± 2.1	µM·mg tissue^{-1}	No	ALF ns	Yes
Yagci[23]	109.0 ± 8.4	79.3 ± 7.7	Yes	11.06 ± 1.95	8.42 ± 2.1	µM·mg tissue^{-1}	Yes	ALF ns	Yes
Azevedo[25]	–	–	–	Unknown	Unknown	µg·100mg tissue^{-1}	No	–	MMP1↑; MMP9↑	Yes
Adas[27]	109.9 ± 25.3	62 ± 21.19	Yes	–	–	–	–	CD↑; NV↑; N↓; E ns; G ns	Yes

the group that received preoperative HBOT only.[23] Five study groups analyzed by four studies, found a significant improvement in the BPR.[20,22,23,27] Three study groups from three of these studies, also found a significant improvement in HP.[20,22,23]

ANASTOMOSES DURING PERITONITIS

Two studies[24,26] investigated the effect of HBOT on colonic anastomoses created during peritonitis. One[24] observed an improvement in the anastomosis during peritonitis with a significantly higher BPR, but this observation using ETR as outcome measure was not supported by the other.[25]

META-ANALYSIS

Meta-analyses on the studies using BPR and HP as outcome measures were performed and included the studies assessing normal and ischaemic anastomoses.[4,20–23,25,27–31] The results are displayed as Forest plots in Figures 2 through 5. Only one study analyzed the effect of preoperative HBOT on both normal and ischaemic anastomoses, and the effect of combined HBOT on ischaemic anastomoses.[23] Therefore, this meta-analysis will not provide extra insights for these groups. For the BPR group, the mean difference (MD) is displayed. Because of the variety in the test determining HP, a standardized mean difference (SMD) was used and because of the high variance of the HBOT protocols between the studies, a random effect was chosen for this meta-analysis. For meta-analysis including the studies using BPR to assess normal anastomoses, a low statistical heterogeneity was found (I_2 = 12%). The other three meta-analyses showed high statistical heterogeneity (I_2 = 74%, 88% and 84% respectively) and, therefore, should be interpreted with caution.

The BPR and HP in the postoperative group of normal anastomoses are significantly improved as shown in Figure 2 and 3 (MD = 20.8 mmHg (14.4, 27.3), $P \leq 0.001$; SMD = 1.2 (0.20, 2.23), $P = 0.02$). The BPR and HP of the studies performing combined HBOT do not show a significant improvement (MD = 6.8 mmHg (-6.3, 19.9), $P = 0.31$, SMD = 0.7 (-0.20, 1.51), $P = 0.14$). The postoperative group of ischaemic anastomoses (Figures 4 and 5) show significant improvement in both BPR and HP (MD = 29.8 mmHg (17.9, 41.7), $P \leq 0.001$, SMD = 2.6 (0.11, 5.13), $P = 0.04$).

Figure 2

Forest plot showing the effect of HBOT on bursting pressure (BPR) in normal anastomoses; HBOT – hyperbaric oxygen treatment; SD – standard deviation; IV – inverse variance, Random – random effect, CI – confidence interval; preop – preoperative; Z – Z-test; P – probability; postop – postoperative; Chi2 – chi-square test; I2 – I-square test for heterogeneity; df – degrees of freedom

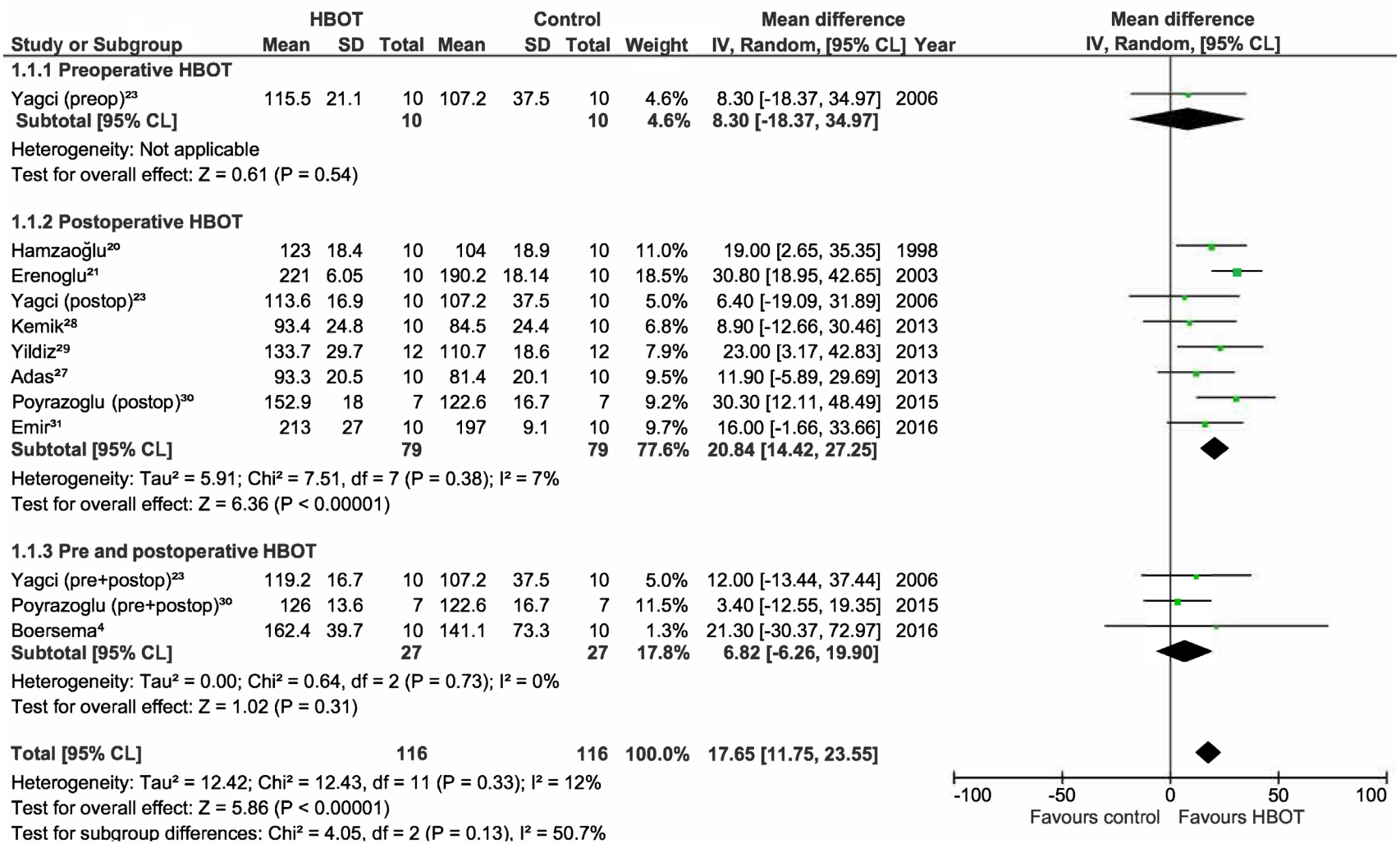


Figure 3

Forrest plot showing the effect of HBOT on hydroxyproline levels (HP) in normal anastomoses; HBOT – hyperbaric oxygen treatment; SD – standard deviation; IV – inverse variance, Random – random effect, CI – confidence interval; preop – preoperative; Z – Z-test; P – probability; postop – postoperative; Chi2 – chi-square test; I2 – I-square test for heterogeneity; df – degrees of freedom

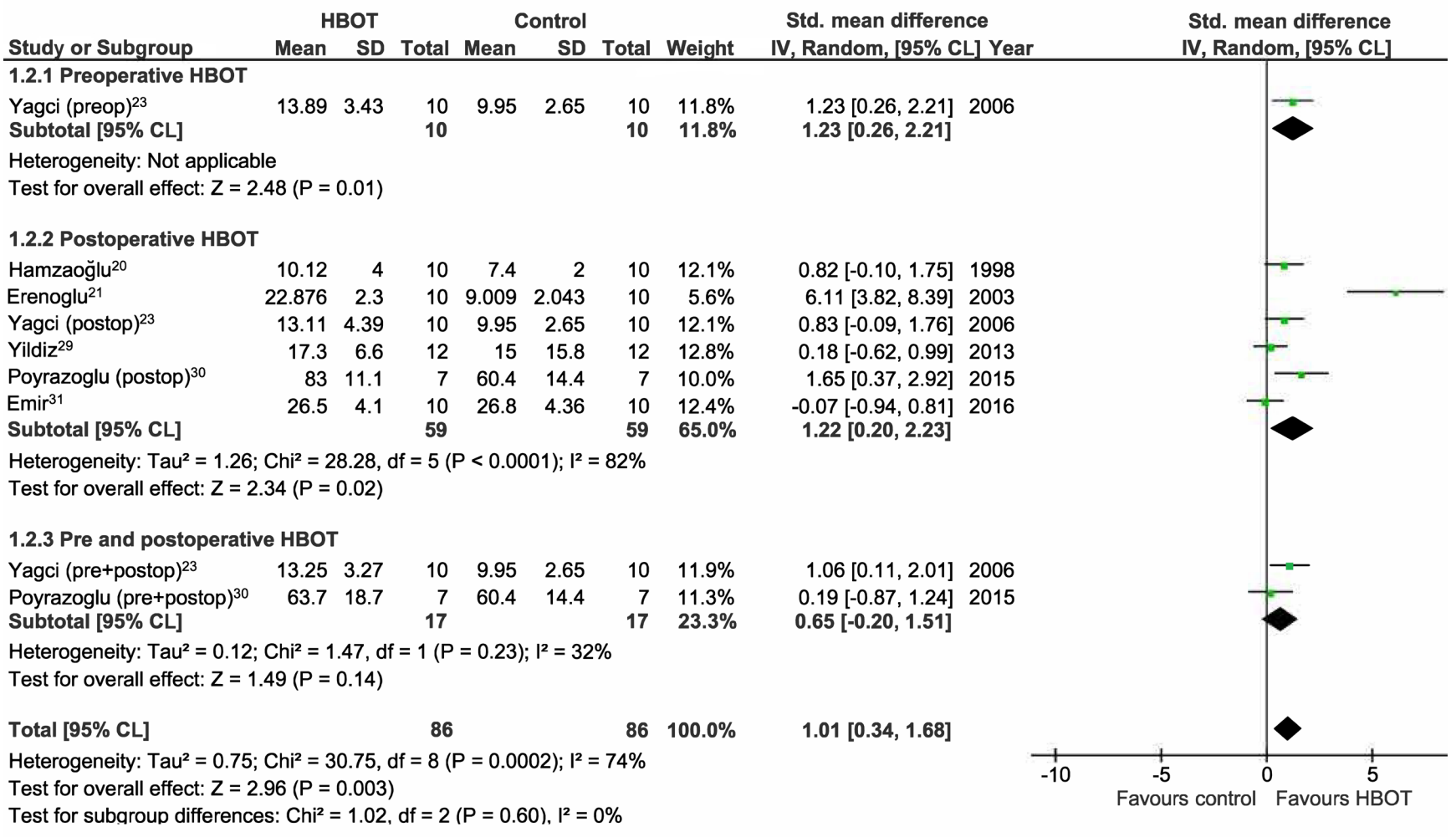


Figure 4

Forest plot showing the effect of HBOT on bursting pressure (BPR) in ischemic anastomoses; HBOT – hyperbaric oxygen treatment; SD – standard deviation; IV – inverse variance, Random – random effect, CI – confidence interval; preop – preoperative; Z – Z-test; P – probability; postop – postoperative; Chi2 – chi-square test; I2 – I-square test for heterogeneity; df – degrees of freedom

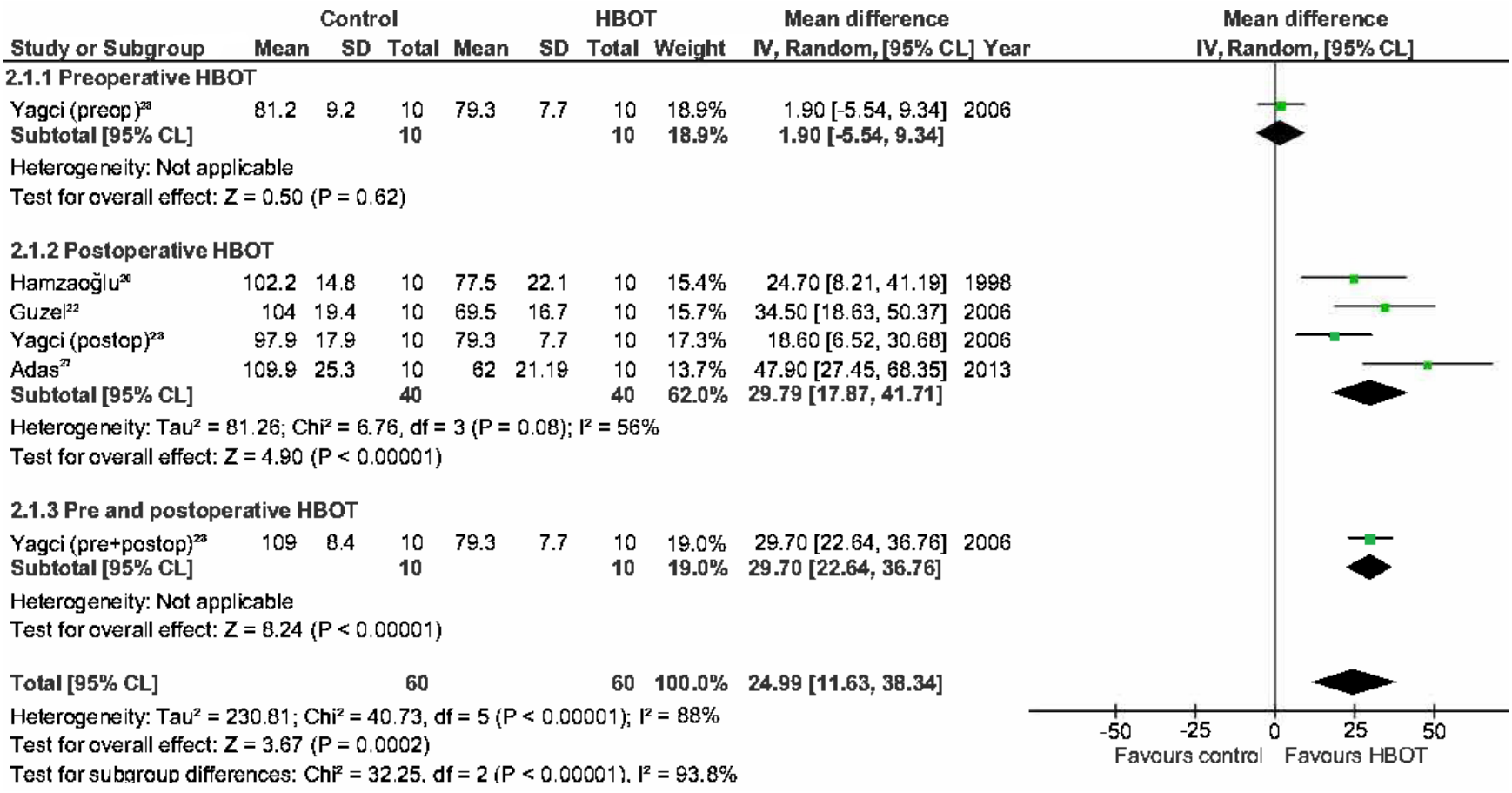
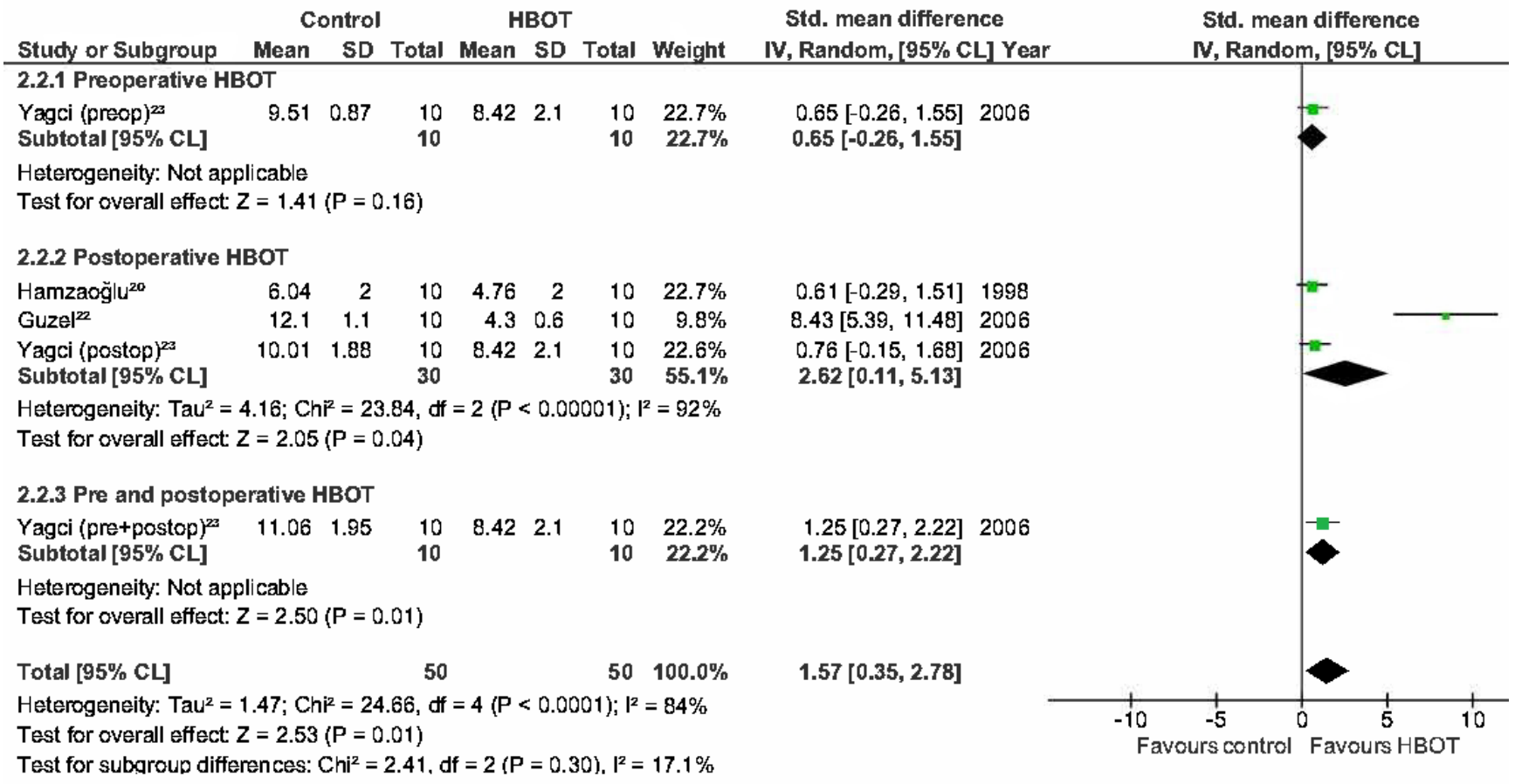


Figure 5

Forest plot showing the effect of HBOT on hydroxyproline levels (HP) in ischemic anastomoses; HBOT – hyperbaric oxygen treatment; SD – standard deviation; IV – inverse variance, Random – random effect, CI – confidence interval; preop – preoperative; Z – Z-test; P – probability; postop – postoperative; Chi2 – chi-square test; I2 – I-square test for heterogeneity; df – degrees of freedom



Discussion

This is the first meta-analysis describing the effect of HBOT on the outcome in colorectal surgery and shows significant improvement of BPR and HP in both normal and ischaemic anastomoses in rats after postoperative HBOT. HP is considered a reliable marker for the strength of the anastomosis and risk of AL in a rabbit model.[18] Therefore, these results could be useful in the complex pathophysiology regarding HBOT and oncology in humans.

The exact mechanism of HBOT in the improvement of colorectal anastomoses is unknown. However, some steps within this pathway are becoming more clearly defined:

- HBOT reduces the risk of AL by lowering the pro-inflammatory response;[9]
- Elevated immune parameters like IL-1, IL-6, IL-10 and tumour necrosis factor (TNF-α) are associated with AL, indicating a connection between AL and a pro-inflammatory response;[32]
- HBOT reduces the risk of AL by improvement of neovascularization.[4,26,27]

Only three studies used preoperative HBOT as a part of their HBOT protocol.[4,23,30] Of these, a significant difference was only found in the combined HBOT group of one study assessing ischaemic anastomoses,[23] but not in the other two.[4,30] The meta-analysis for postoperative HBOT showed a stronger association between HBOT and the prevention of AL than that for preoperative HBOT. The reasons for this difference are not yet identified. Regarding the results shown in Figure 2, preoperative HBOT might possibly prevent the positive effect of postoperative HBOT.

The major limitation of the current review is the quality of the available evidence. According to the SYRCLE tool there is a risk of bias in most of the included studies. Also, the protocols varied between studies, making it problematic to combine them in a meta-analysis. Different HBOT doses (pressure and time) might influence outcome. Furthermore, the statistical heterogeneity between included studies was high, and only the meta-analysis of the subgroup using BPR as outcome measure for postoperative HBOT in non-ischaemic anastomoses could be regarded as trustworthy. The results of the other three subgroups should be interpreted with caution. Finally, most colorectal resections are performed on patients with a malignancy, whereas these studies are performed on rats without a malignancy. Although the current consensus is that HBOT does not promote cancer, further research might be needed before recommending HBOT as a routine for patients with colorectal cancer.

There is only one reported human HBOT case series of five patients who underwent an ultra-low anterior resection with a temporary loop ileostomy and who developed AL with chronic pelvic sepsis.[32] All five received postoperative HBOT (90 minutes at 203–243 kPa, five days per week for six weeks), four also receiving adjuvant chemo-radiotherapy. All the patients showed improvement in the degree of anastomotic separation and sepsis.[32]

Conclusion

This meta-analysis provides some evidence to suggest HBOT may be a useful adjunct in colorectal surgery. Postoperative HBOT increases the strength of the colorectal anastomosis in rats without a malignancy, this effect appearing to be stronger in ischaemic anastomoses. To investigate the full potential of HBOT to prevent AL in human patients undergoing colorectal surgery, a pilot study should be performed. Since it would be hard to obtain the large numbers of human patients that would be necessary, further research should focus primarily on a larger systematic animal study using postoperative HBOT and with AL as the primary outcome measure.

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Review article

Inner ear barotrauma in divers: an evidence-based tool for evaluation and treatment

Stefan W Rozycki[1], Matthew J Brown[1], Macario Camacho[2]

[1] *NSSC Pearl Harbor 822 Clark St Suite 400 Joint Base Pearl Harbor Hickam, Hawaii, USA*
[2] *Otolaryngology-Head and Neck Surgery, Tripler Army Medical Center, Hawaii*
Corresponding author: *Macario Camacho, Otolaryngology-Head and Neck Surgery, 1 Jarrett White Road, Tripler Army Medical Center, Hawaii, USA*
drcamachoent@yahoo.com

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Hearing loss, sudden; ENT; Decompression sickness; Systematic review

Abstract
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Objective: To systematically search the literature for studies evaluating the typical presentation and testing that is performed for divers with inner ear symptoms and then to create a tool for clinicians when evaluating a diver with inner ear symptoms.
Methods: Nine databases, including PubMed/MEDLINE were systematically searched through 31 January 2018. The PRISMA statement was followed.
Results: Three-hundred and two manuscripts were screened, 69 were downloaded and 21 met criteria to be included in this review. The articles were evaluated for symptomatic trends and initial evaluation work-up primarily focusing on inner-ear barotrauma (IEBt) and inner ear decompression sickness (inner ear DCS). The trends for IEBt were compared to typical inner ear DCS presentation based on large study inner ear DCS results consistent with the plethora of research available. Finally, the HOOYAH Tool was developed to assist the receiving provider to better determine the most likely diagnosis and thus initiate appropriate treatment. The HOOYAH Tool is comprised of the following: 1) H: hard to clear; 2) O: onset of symptoms; 3) O: otoscopic exam; 4) Y: your dive profile; 5) A: additional symptoms and 6) H: hearing. For each of these components, the typical presentation is described allowing the provider better to discern the correct diagnosis.
Conclusion: The diagnosis of IEBt remains difficult to define short of visualization through surgical exploration. Early treatment is defined by conservative management with a subsequent observational period to determine symptomatic resolution and need for surgery. However, a similar differential diagnosis is inner ear DCS which requires early recompression. The HOOYAH tool provides a method for assisting the provider in forming a more confident decision regarding the underlying pathology and facilitation of the appropriate treatment.

Introduction

For underwater divers, whether novice or experienced, there are many risks assumed whenever a new dive is planned. Amongst these risks, there are the ever-present spectra of decompression illness and barotrauma. This article focuses on the intersection of these two processes in inner ear barotrauma (IEBt) and inner ear decompression sickness (inner ear DCS).

The inner ear is a complex system encased within the bony labyrinth consisting of two major functional parts; the vestibular apparatus concerned with balance and the cochlea dedicated to hearing, converting sound waves into electrical impulses passed to the brain. The cochlea is composed of three main divisions called scala within the bony labyrinth, the scala vestibuli most superior, scala media in the middle, and the scala tympani most inferior. These scala are separated by membranes, Reissner's membrane between the scala vestibuli and media and the basilar membrane between scala media and tympani. The scala vestibuli and tympani are filled with a fluid called perilymph and scala media with a similar fluid called endolymph. Although very similar, these fluids are unique in their ionic compositions, which is important since the Organ of Corti, which is where sound waves are converted to electrochemical impulses, is located in the *scala media* on the basilar membrane and is bathed in endolymph.

The pressure of the stapes bone in the middle ear moving in and out as it vibrates causes pressure waves within the perilymph fluid. These waves travel through the perilymph, pass to the endolymph and into the Organ of Corti where they are transformed into nerve impulses sent to the brain along the vestibulocochlear nerve. This is a fragile system, with any break of the basilar membrane causing disruption

of the Organ of Corti and consequential hearing loss; and disruption of Reissner's membrane causing mixing of endolymph and perilymph with similar outcomes. At the base of the cochlea, inferior to the oval window in the vestibule of the membranous labyrinth sits the round window, which compensates for the pressure changes in the fluid by flexing in and out in time with the fluid waves to prevent damage to the sensitive membranes. This places the round window, as well as the oval window at risk of barotrauma with sudden changes in pressure.

There are two main ways the inner ear can experience barotrauma, through explosive or implosive means. As a diver descends, the tympanic membrane (TM) will be pressed medially as the external pressure increases. This will in turn press the stapes into the oval window causing increased pressure in the cochlea and bulging of the round window. At a pressure differential of > 90 mmHg (12 kPa) the Eustachian tube will 'lock' closed preventing a successful Valsalva.[1] As the diver feels increased pressure, an attempt will be made to equalize, and when this is unsuccessful the diver will often resort to repeated increasingly forceful Valsalva manoeuvres which increase intracranial pressure. This is transmitted through the perilymphatic duct to the cochlea, further increasing the pressure in the cochlea, which can cause a rupture of the round window or annular ligament. In cats, a pressure as low as 13.6 cm H_2O can rupture the round window.[2]

The rupture caused by an increase in perilymphatic fluid pressure is termed an explosive rupture. On the other hand, rupture caused by a decrease in perilymph fluid pressure is an implosive rupture. This occurs with a sudden increase in middle ear pressure after a forceful Valsalva that is successful in opening the Eustachian tube. With the sudden increase in size of the middle ear, the TM expands rapidly outwards, pulling the bones of the ear with it, and with the bones, so too the oval window through the attachment of the annular ligament creating low pressure in the cochlea. This can cause a tear of the oval or round window due to the velocity of change. It is also reasonable to imagine these forces can have the added effect of rupturing an internal membrane of the cochlea, Reissner's or basilar membrane. Only small pressure differentials are required to rupture the membrane causing mixing of endolymph and perilymph,[3,4] and these values are reached with a round or oval window rupture.

Inner ear DCS is less well understood, but is thought to be brought about by one of two mechanisms, or some combination of the two. Firstly, autochthonous bubble formation may occur in the endolymphatic and perilymphatic spaces due to local supersaturation of inert gas, with the vestibular apparatus more at risk than the cochlea as it has lower arterial perfusion and slower inert gas washout.[5] Secondly, it is thought a bubble from the venous circulation travels through a right-to-left shunt and lodges in the labyrinthine artery which supplies the vestibular apparatus and cochlea.[6]

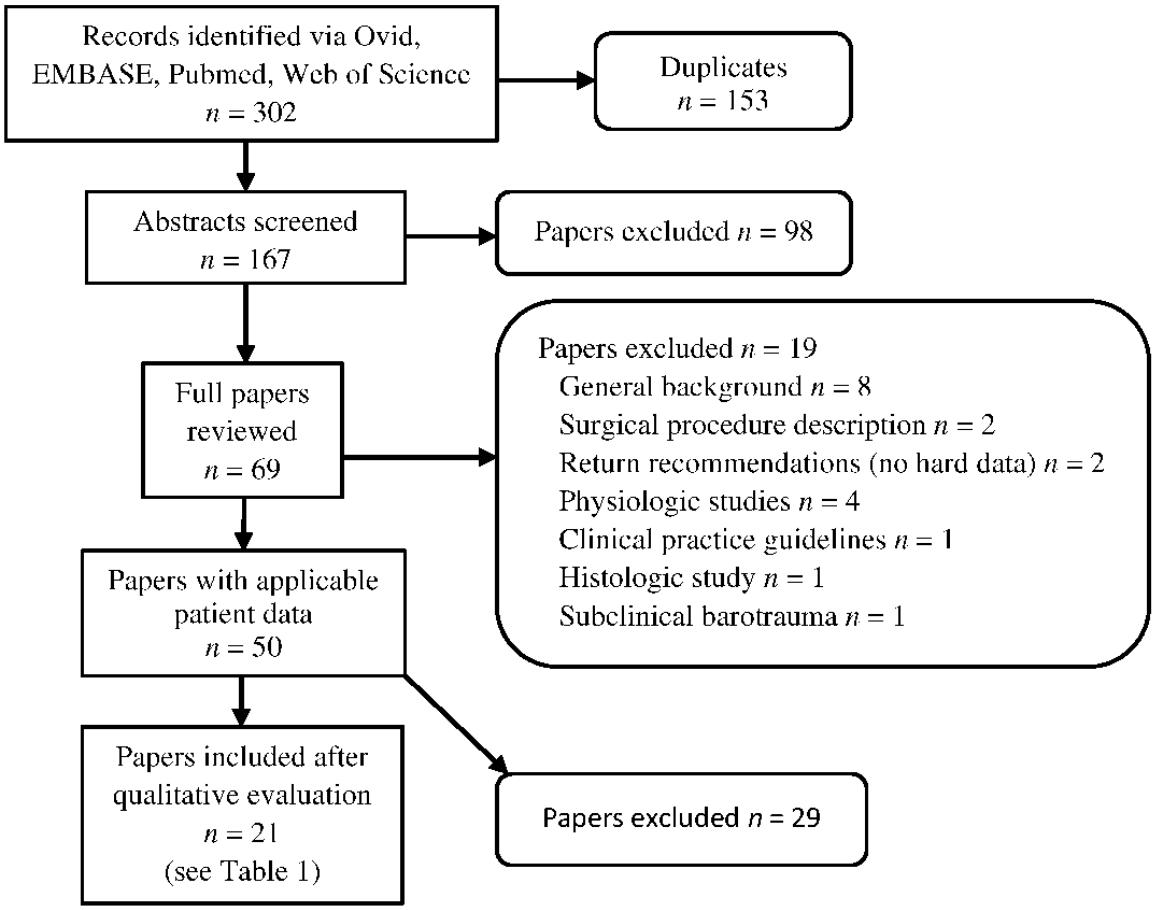
As both injuries affect the inner ear, both inner ear DCS and IEBt can present with similar symptoms of hearing loss and tinnitus (cochlear symptoms), as well as nausea, vertigo, and nystagmus (vestibular symptoms). This leads to difficulty distinguishing between the two diagnoses potentially delaying appropriate treatment. IEBt often requires surgical management if symptoms fail to improve with conservative treatment, with some recommendations including an observational period of up to 10 days from the known acute injury.[7] Contrarily, the gold standard treatment for inner ear DCS is early recompression with oxygen (HBOT), ideally initiated within six hours from injury.[8] Delayed treatment beyond this results in some degree of permanent inner ear damage in 90% of casulties.[9] Permanent damage has been reported even when treatment is started one hour after the known injury occurred.[10] Therefore, it is crucial that providers have an awareness of the typical presentations of both IEBt and inner ear DCS so that the appropriate treatment is initiated in a timely fashion. This paper explores the current literature to evaluate the typical presentation and testing in an attempt to create a tool for clinicians to utilize when evaluating a diver with inner ear symptoms, thus facilitating the most appropriate and prompt treatment.

Methods

Two authors (SWR, MJB) independently searched the international literature – in all languages – from the inception of each database through 31 January 2018. The databases that were systematically searched included PubMed, Ovid MEDLINE, EMBASE, and Web of Science. An example of a search strategy for Ovid MEDLINE is: 'Inner ear barotrauma', 'diving', 'middle ear', 'inner ear', 'barotrauma', and 'inner ear decompression illness'. Results included a total of 320 papers with 153 duplicates identified through Prepostseo plagiarism/duplication identification software (https://www.prepostseo.com/plagiarism-checker) leaving a total of 167 unique papers for evaluation. The remaining titles and abstracts were then personally reviewed by SWR and MJB to determine possible relevance to the diagnosis and treatment of IEBt and inner ear DCS. Reasons for exclusion included: only addressed middle ear barotrauma; focus on anatomy outside of the ear; focus on alternate pathology associated with diving (e.g., facial nerve baroparesis, hypothermia, alternobaric vertigo); foreign language, unable to access full text of paper. In total, 69 papers were identified as pertinent and the full text was reviewed for inclusion in the study (Figure 1). Additionally, a manual search was completed of texts of diving medicine that address IEBt and inner ear DCS including lecture and workshop material from the US Navy Diving and Salvage Training Centre.

Of the 69 full papers reviewed, 19 were determined to be outside the scope of this paper (Figure 1). The 50 papers identified as germane to the topics at hand were taken, and the reported patient data were compiled and examined for

Figure 1
Flow chart for study selection; *n* – number of studies



general trends. Those 50 papers were then examined (by SWR and MJB) for quality of evaluation, study design and comprehensive scope of evaluation. This resulted in 21 primary studies for analysis according to the quality assessment of cases series studies checklist from the National Institute for Health and Clinical Excellence (NICE; https://www.nice.org.uk) (Table 1).

1. Was the case series collected in more than one centre, i.e., multi-centre study?
2. Is the hypothesis/aim/objective of the study clearly described?
3. Are the inclusion and exclusion criteria (case definition) clearly reported?
4. Is there a clear definition of the outcomes reported?
5. Were data collected prospectively?
6. Is there an explicit statement that patients were recruited consecutively?
7. Are the main findings of the study clearly described?
8. Are outcomes stratified? (e.g., by abnormal results, disease stage, patient characteristics)?

The articles selected were evaluated for symptomatic trends and initial evaluation work-up primarily focusing on IEBt. These trends for IEBt were compared to typical inner ear DCS presentation based on large study inner ear DCS results that are consistent with the plethora of research available. Finally, a tool (HOOYAH) was created to assist the receiving provider to better determine the most likely diagnosis.

Results and discussion

TINNITUS

Tinnitus was reported as a symptom of IEBt in 21 of the full-length articles. Of the articles that specifically delineated patients with tinnitus symptoms, it was found that tinnitus was present in 187/256 (73%) of the patients with diagnosed IEBt, with findings ranging from 30%–81%. There were small studies that had 100% of patients reporting tinnitus, however, these sample sizes are too small to be of statistical significance. Resolution of the tinnitus after IEBt appears to be fairly impressive. Only two studies tracked follow-up of the symptoms, with tinnitus resolving in all patients in one, and in the other, tinnitus completely resolved in 15/19 patients who required surgical repair.[11] One study differentiated between perilymphatic fistula (PLF), inner ear hemorrhage (IEH) and intracochlear membrane tear (ICMT), but there was no significant difference in the prevalence of tinnitus between the different locations of injury.[12]

Table 1

General characteristics and quality criteria of included studies; quality assessment of cases series studies checklist from National Institute for Health and Clinical Excellence (NICE) (see text for details of the eight questions in the checklist)

First author Study design	Country	Evidence level	Outcomes analyzed	1	2	3	4	5	6	7	8
Shupak[1] **Case series**	Israel	4	Nystagmus, audiometry, CT	N	Y	Y	Y	N	N	Y	N
Klingmann[5] **Case series**	Germany	4	Symptoms, timing of symptoms and association with right-to-left shunt	N	Y	Y	Y	N	N	Y	N
Nachum[8] **Case series**	Israel	4	Vestibular symptoms, cochlear symptoms, residual symptoms post treatment	N	Y	Y	Y	N	N	Y	N
Shupak[9] **Case series**	Israel	4	Clinical symptoms, Nystagmus, ENG, Audiometry	N	Y	Y	Y	Y	N	Y	N
Healy[11] **Systematic review** **Case series**	USA	4	Nystagmus, Romberg and fistula tests, hearing loss	Y	Y	N	Y	N	N	Y	N
Parell[12] **Case series**	USA	4	Audiogram, vestibular symptoms, worsening tinnitus after return to diving	Y	Y	Y	Y	N	N	Y	Y
Roydhouse[13] **Prospective observational**	New Zealand	4	Hearing loss, balance, tinnitus pre and post op	N	Y	N	Y	Y	N	Y	N
Edmonds[14] **Case series**	Australia	4	Audiogram, ENG, tinnitus	N	Y	Y	Y	N	N	Y	N
Cantais[15] **Case series**	France, USA	3	Association of DCS symptoms and right-to-left shunt	Y	Y	Y	Y	N	N	Y	Y
Goplen[16] **Prospective longitudinal**	Norway	3	Audiometry	N	Y	Y	Y	Y	N	Y	Y
Podoshin[17] **Case series**	Israel	4	CT, ENG, fistula test, audiometry	N	Y	Y	Y	N	Y	Y	N
Klingmann[18] **Cross-sectional controlled**	Germany	3	Audiometry	Y	Y	Y	Y	N	N	Y	Y
Zulkaflay[19] **Cross-sectional**	Malaysia	3	Hearing loss, age	N	Y	Y	Y	Y	N	Y	Y
Vartiainen[20] **Retrospective series**	Finland	4	Fistula test, audiogram, ENG	N	Y	Y	Y	N	N	Y	Y
Taylor[21] **Cross-sectional, descriptive survey**	Australia, USA	4	History of "*squeezes*", membrane rupture, hearing loss, tinnitus, balance	Y	Y	Y	Y	N	N	Y	N
Freeman[22] **Case series**	Australia	4	Audiogram	Y	Y	Y	Y	N	N	Y	N
Lo[23] **Case report**	Taiwan	4	Clinical symptoms, surgical outcome	N	Y	N	Y	N	N	Y	N
Duplessis[24] **Observational, cohort**	USA	3	Otoacoustic emission test for transient emission shift	N	Y	N	Y	Y	N	Y	N
Gempp[25] **Case series**	France	4	Dive profile, clinical symptoms, presence of right-to-left shunt	N	Y	Y	Y	N	N	Y	N
Smerz[26] **Case series**	USA	4	Epidemiology associated with inner-ear DCI	N	Y	Y	Y	N	N	Y	N
Livingstone[27] **Systematic review of case series**	Multiple papers	4	Pathophysiology, diagnosis and treatment of otologic complications	Y	Y	Y	N	N	N	Y	N

VERTIGO

Vertigo was reported in 28 of the reviewed articles. Overall prevalence of symptomatic vertigo in patients with IEBT was found to be 71/156 (46%), with findings ranging from 28–77%. One study of 1,110 cases of diving-related otologic complaints found vertigo in 203 cases (18%); however, the paper did not specify if all of these cases were IEBt versus other causes of diving-related vertigo (e.g., caloric vertigo alternobaric vertigo or inner ear DCS).[13] Not surprisingly, the study that delineated between anatomic variations of IEBt found differing incidences of vertigo: 4/4 PLF; 3/10 IEH and 3/10 with ICMT.[12] This is expected, given the more isolated injury in IEH and ICMT that is more typically associated with isolated hearing loss/tinnitus. Symptomatic response to treatment was also very successful. In 50 Australian cases of IEBt, 16 reported vertigo, and all improved over the span of weeks-to-months with conservative treatment only.[14] Conservative management was by far more utilized than surgical intervention; even so, surgery carried a dramatic immediate improvement with 12/13 cases showing immediate post-surgical improvement in vertigo, primarily when PLF was identified or suspected and subsequently patched.

MIDDLE EAR BAROTRAUMA (MEBt)

It is commonly thought of that inner ear barotrauma is associated with MEBt.[28] However, in the studies evaluated, only three discussed MEBt to any extent. In one study, 26/50 divers with IEBt had concomitant MEBt.[14] However, in 150 randomly selected occupational divers studied for chronic audiological complaints, 40 (27%) had a history of MEBt, but there were no reported cases of IEBt. Similarly, in 67 occupational divers over a span of more than 27,000 dives, there were no cases of IEBt, further demonstrating the low prevalence of this injury.[16] A chronic threshold shift was not associated with a history of MEBt.[16] Therefore, whilst it can be concluded that there is a relatively high incidence of MEBt when a diver presents with IEBt, MEBt is often isolated and does not necessarily have any connection with inner ear damage whether in the initial case or after a repeated history of MEBt. Therefore, providers, particularly on the dive-site or initial receiving providers (emergency room, primary care, etc.), should avoid treatment modalities used for IEBt (e.g., steroids, bed rest, etc.) if only isolated MEBt is suspected.

ELECTRONYSTAGMOGRAPHY

Electronystagmography (ENG) can be utilized to better localize the site of pathology for vertigo (e.g., central or peripheral), and can be executed in the hospital setting to better quantify the severity of the reported vestibular symptoms. The variety and complexity of the tests are beyond the scope of this paper, however, the utility is important to discuss. In the retrospective study of 50 divers with IEBt, the reported symptoms were verified with pure tone audiometry (PTA) and/or ENG.[14] Another study of 53 suspected cases of PLF found that ENG had a sensitivity and specificity of about 77%. It is important to note that those cases were restricted to only those suspected to be PLF, which is only one subset of IEBt, as previously discussed.[17] However, when considering more global IEBt, the ENG revealed pathology in only four of 50 divers when there were no subjective complaints.[14] The body's adaptive and restorative capabilities are displayed in this study as all vestibular symptoms resolved over weeks-to-months, and the only evidence of persistent vestibular pathology was on repeat ENG testing.

HEARING LOSS

Hearing loss is extremely important for US Navy Divers. Divers are monitored closely with annual audiograms in order to identify any possible development of disabilities. Historically, it has been suspected that occupational divers have a higher rate of hearing loss than the general public. Whilst this may be true, the underlying cause is always in question: Is it due to repeated exposure to pressure, or isolated incidents of trauma? In a study of 60 divers of all ages, no statistically significant difference in hearing threshold was found between divers and non-divers.[18] This study specifically excluded divers with a history of IEBt or inner ear DCS, suggesting that non-acute exacerbations do not increase hearing loss.[18] This was further clarified in another study which found that chronic hearing loss in divers was likely due to a single event (e.g., IEBt or inner ear DCS) rather than chronic exposures to higher atmospheric pressure.[19]

Adding to this, hearing loss was the most common symptom reported for cases of IEBt. Of the studies evaluated, 16 discussed hearing loss and, of the aggregate, 228/253 (90%) of patients presented with hearing loss. As mentioned above, hearing loss was the most prevalent symptom noted more so than vestibular complaints. One large study found that all patients with vertigo reported hearing loss, whereas hearing loss was commonly reported as an isolated symptom, with vertigo almost never an isolated symptom in the acute phase.[13] This is intuitive when considering the various anatomic locations where IEBt can occur (IEH, ICMT, and PLF), seeing as how IEH and ICMT are more likely to be associated with hearing loss and having no effect on the vestibular system.[12]

Based on the available studies, recovery of hearing loss is inconsistent. Of 50 divers with IEBt, cochlear symptoms resolved within hours/months, whereas recovery of hearing loss occurred in less than two-thirds of the divers.[14] In a study primarily focusing on PLF, 44/51 patients receiving surgical correction did not have hearing improvement > 20 dB; surgery was considered to be an unsuccessful intervention for hearing loss.[20] Divers with chronic hearing

Table 2

In analyzing presenting symptom trends, diver history, and various tests available to the clinician, the authors have created a tool to provide insight into the aetiology of a diver with inner ear symptoms: the HOOYAH criteria. * N.B. It is essential that patients undergo a full otologic exam to include otoscopic, tuning fork and bedside vestibular testing (e.g., head impulse test). While these tests will provide insight into the type of hearing loss and/or vestibular patterns, potentially identifying conductive versus sensorineural hearing loss, there was no evidence to suggest improved ability to differentiate between inner-ear decompression sickness and inner-ear barotrauma; therefore, these are not included in the following criteria

	HOOYAH criteria	Typical in IEBt	Typical in inner ear DCS
H	Hard to clear	Present on descent or ascent; forceful Valsalva	Difficulty clearing not associated with inner ear DCS
O	Onset of symptoms	May occur on descent, ascent or on surface after diving	May occur on ascent (technical diving) or on surface after diving
O	Otoscopic examination	Association with MEBt	Normal otoscopic exam
Y	Your dive profile	Profile with no/low risk of DCS; fast ascent or descent	Decompression diving; missed decompression stops; repetitive dives; any dive profile with risks for DCS
A	Additional symptoms	Isolated inner ear	Association with other DCS symptoms
H	Hearing	Very common; high frequency loss; fluctuating hearing loss	Vestibular symptoms more common; often right-sided symptoms

loss had a history of MEBt or recurrent middle ear squeezes, and it was postulated by these authors that these patients likely had some previously undiagnosed IEBt which caused the residual deficits.[21] The studies focusing on recovery primarily involved patients with PLF, which is the most extreme variation of IEBt; therefore, it is not unexpected that they carry a higher incidence of chronic disability.

PURE TONE AUDIOMETRY (PTA)

Utilizing audiometry is difficult in these patients because of the non-specific nature of the tests. However, PTA is essential in the diagnosis of IEBt, and is recommended universally. The pattern of hearing loss is variable, with a large majority showing flat or down-sloping audiograms of varying severity and/or high frequency loss,[22] but also isolated of cases of low frequency loss.[12] These isolated low frequency losses may be secondary to a localized IEH or possibly ICMT. It is also important to note that given the mechanism of injury and the correlation of possible MEBt, that a diver may present with a mixed hearing loss pattern.[23] Acknowledging this is doubly important when considering that patients may be suspected to have only MEBt, but a sensorineural hearing loss (SNHL) also needs to be considered in order to facilitate appropriate care (e.g., in combined MEBt and IEBt conservative treatment would include steroid therapy and bed-rest.)

Based on the research analyzed, PTA was the best and most readily available audiometric test to facilitate in the diagnosis and observation of IEBt. Alternate audiometric tests have been studied, including otoacoustic emissions (OAE) testing.[24] This study investigated the potential of OAE to identify clinical and subclinical IEBt (defined as a transient emission shift without an accompanying transient threshold shift on PTA). OAE testing identified significant transient emissions shift in an intense repetitive diving protocol, supporting the theory that clinical and subclinical IEBt can lead to chronic SNHL.[19] Based on these results, while not evaluated in the acute setting, it was postulated that OAE can be used for medical surveillance and support early intervention.

COMPARISON TO INNER EAR DCS

There are some important differences from IEBt presentation in dive casualties with suspected inner ear DCS. Typically, symptomatic onset for inner ear DCS presents soon after a dive. Six different studies consisting of 138 dive casualties found that the average onset of symptoms was around 36 minutes after surfacing (range 13–206 minutes).[5,8–10,25,26] There are documented cases of symptom onset while on ascent from a dive, but these are far less common.[10] However, in review of all of the articles related to IEBt, there was no identifiable trend, even within individual studies, regarding timing of symptom onset: IEBt symptoms are likely to present themselves on either descent, ascent or soon after surfacing.

In contrast to IEBt, where the large majority of cases present with cochlear symptoms, inner ear DCS casualties have a preponderance of vestibular symptoms. One retrospective

study found that patients presented with a pure vestibular disorder in up to 75% of cases, with only 6% having isolated cochlear symptoms, whilst combined cochleovestibular symptoms occurred in 17.5%.[25] Other reviews have found higher rates of cochlear symptoms, although far less than that observed in IEBt.[8,26,27] This could be explained by the comparatively lower perfusion and slower inert gas washout in the vestibular apparatus leading to increased risk for DCS symptoms in the vestibular apparatus. Stated simply, the vestibular apparatus has a higher tissue volume to blood supply ratio in comparison to the cochlea, leading to higher rates of local supersaturation and arterial microbubble load.[5] Interestingly, one study of 115 cases of inner ear DCS found that symptoms lateralizing to the right in 72% of cases.[8]

It is important for the clinician to understand that inner ear DCS can occur in isolation, or in conjunction with other DCS symptoms. A key indicator of likely inner ear DCS versus IEBt would be the presence of such DCS-related symptoms. However, the evidence is not straight forward. Whilst previous studies found that 24–34% of divers with DCS reported cochleovestibular symptoms,[8,15] a more recent study found only 17% of inner ear DCS patients had other DCS-related symptoms.[5] These findings indicate inner ear DCS to be a discrete pathological process that commonly presents in isolation, but may also appear with a constellation of other DCS symptoms. Therefore, it is important that in any divers with inner-ear symptoms a thorough, full history and examination be undertaken in order to establish the most likely diagnosis. By better understanding the variations in presentation based on past research, the HOOYAH criteria was developed as a way to assist forming a more confident decision regarding the underlying pathology and facilitation of the appropriate treatment (Table 2).

Should a scenario arise where there is a concern for an injury involving both inner ear DCS and IEBt, we recommend that bilateral tympanotomies be performed prior to initiating HBOT, similar to the scenario where there is concern for the patient's ability to equalize. We do not recommend tube placement initially prior to pressurisation as simple tympanotomies should suffice for acute hyperbaric management. Tube insertion would require more specialized providers and equipment that would delay treatment. If the patient needs additional HBOT, then insertion of tympanostomy tubes may be coordinated on a less emergent basis.

Conclusions

The diagnosis of IEBt remains difficult to define short of visualization through surgical exploration. Early treatment is defined by conservative management with a subsequent observational period to determine symptomatic resolution and any need for surgery. However, a similar differential diagnosis is inner ear DCS which requires early recompression. The HOOYAH tool provides a method for assisting the provider in forming a more confident decision regarding the underlying pathology and facilitation of appropriate treatment.

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Case report

A recurrent, ischaemic ileocolonic anastomosis ulcer refractory to surgery treated with hyperbaric oxygen

Puraskar Pateria, Andre Chong

Department of Gastroenterology and Hepatology, Fiona Stanley Hospital, 11 Robin Warren Drive, Murdoch, Western Australia 6150

Corresponding author: *Puraskar Pateria, Work Address: Department of Gastroenterology and Hepatology, Service 4, CD09, Fiona Stanley Hospital, 11 Robin Warren Drive, Murdoch, Western Australia 6150*

Puraskar.Pateria@health.wa.gov.au

Key words

Gastrointestinal tract; Chronic wounds; Case reports

Abstract

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A 54-year-old male had undergone right hemicolectomy and ileo-colonic anastomosis for carcinoma-in-situ found at colonoscopy. Eighteen months later, he presented with a lower gastrointestinal bleed from an anastomotic ulcer, treated with resection of the anastomotic site and ileo-sigmoid anastomosis. In the ensuing 12 months, he had three episodes of haematochezia. Colonoscopy revealed a 12 mm anastomotic ulcer necessitating a further colonic resection and re-anastomosis. Two-years later, he presented with iron deficiency anaemia. He preferred expectant management and received ten iron-infusions over the subsequent four years. Thereafter, he developed painless haematochezia. Colonoscopy showed a 15 mm linear ulcer with mild ooze at the anastomosis. Histology was consistent with an ischaemic ulcer; there was no evidence of recurrence of carcinoma-in-situ. The ulcer remained refractory to endoscopic and medical treatment, as seen at three follow-up colonoscopies. Hyperbaric oxygen treatment (HBOT) was offered and he received 30 sessions over six weeks. Colonoscopy at HBOT completion revealed healing of the ulcer. The patient had no further overt bleeding and serum ferritin has continued to rise spontaneously over 12 months follow-up.

Introduction

Ulceration at ileo-colonic and colo-colonic anastomoses is a known complication of large bowel resection, described with variable frequencies ranging from 0.8 to 2.5%.[1–3] While the precise aetiology of such ulceration remains unknown, use of non-steroidal anti-inflammatory drugs (NSAIDs), development or recurrence of Crohn's disease and malignancy have been found in some cases. The incidence of anastomotic ulceration is more common in the paediatric population than in adults.[4,5] Anastomotic ulcers commonly present with iron deficiency and gastrointestinal bleeding.[1,2]

Many therapies, including sulfasalazine, sucralfate, 5-aminosalicylic acid (5-ASA), antacid therapy such as ranitidine and protein pump inhibitors have been described for management of anastomotic ulcers with variable success.[1–5] We report a case of recurrent anastomotic ulceration resistant to all medical and surgical intervention over several years but which healed with hyperbaric oxygen treatment (HBOT).

Case report

A 54-year-old male presented with abdominal pain and underwent colonoscopy which revealed a polyp and biopsies were consistent with carcinoma-in-situ. He was managed with right hemicolectomy and ileo-colonic anastomosis, from which he recovered well. Past medical history included basal cell carcinoma resection and solar keratosis. He was not on any regular medications. He denied intake of NSAIDs. He had adequate dietary intake of iron, was a non-smoker and drank 1–2 standard drinks of alcohol per day. There was no significant family history of gastrointestinal malignancy.

Eighteen months later, he presented with an acute lower gastrointestinal bleed. Colonoscopy showed bleeding from an anastamotic ulcer. A resection of the anastomotic site, transverse and left hemicolectomy and ileo-sigmoid anastomosis was performed. In the ensuing twelve months, he had three episodes of lower gastrointestinal bleeds and remained iron deficient requiring three iron infusions and one blood transfusion. He was investigated with capsule endoscopy, double balloon enteroscopy and colonoscopy. The investigations revealed a 12 mm ulcer at the anastomosis

Figure 1
15 mm, ischaemic, linear ulcer (white) at the ileo-colonic anastomosis with mild ooze

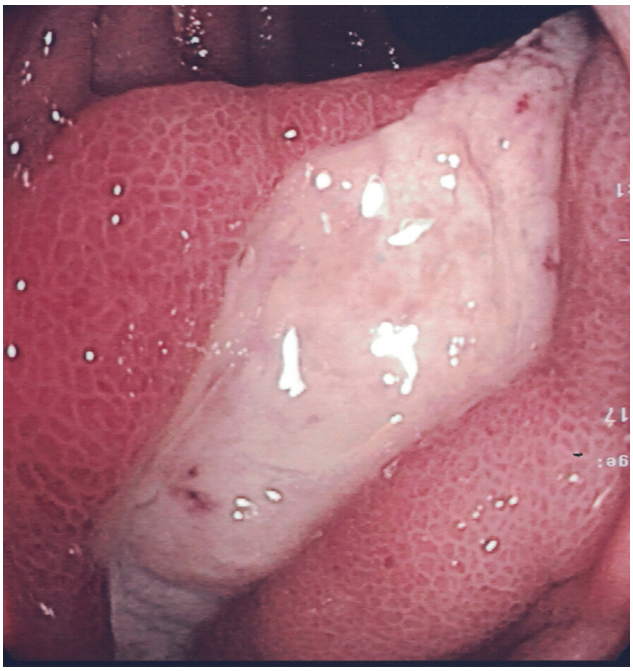


Figure 3
Healed ulcer, post 30 HBOT 90-minute sessions at 243 kPa, showing a healthy anastomosis with good vascular markings

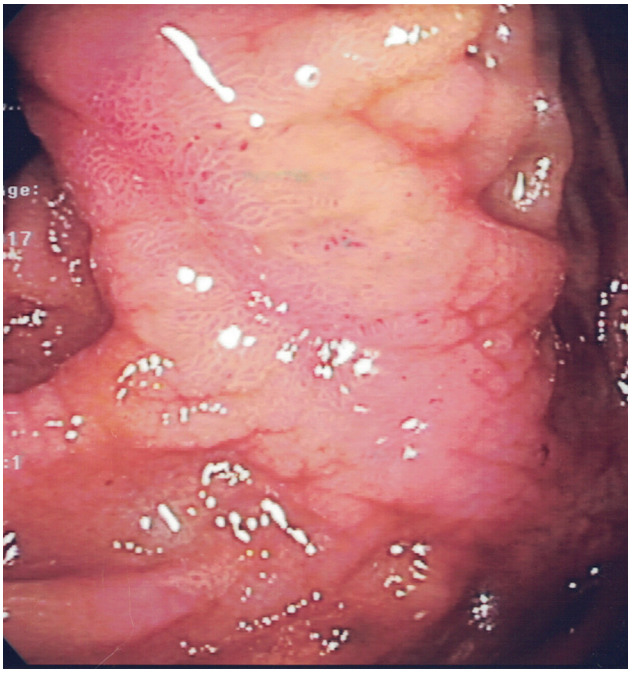
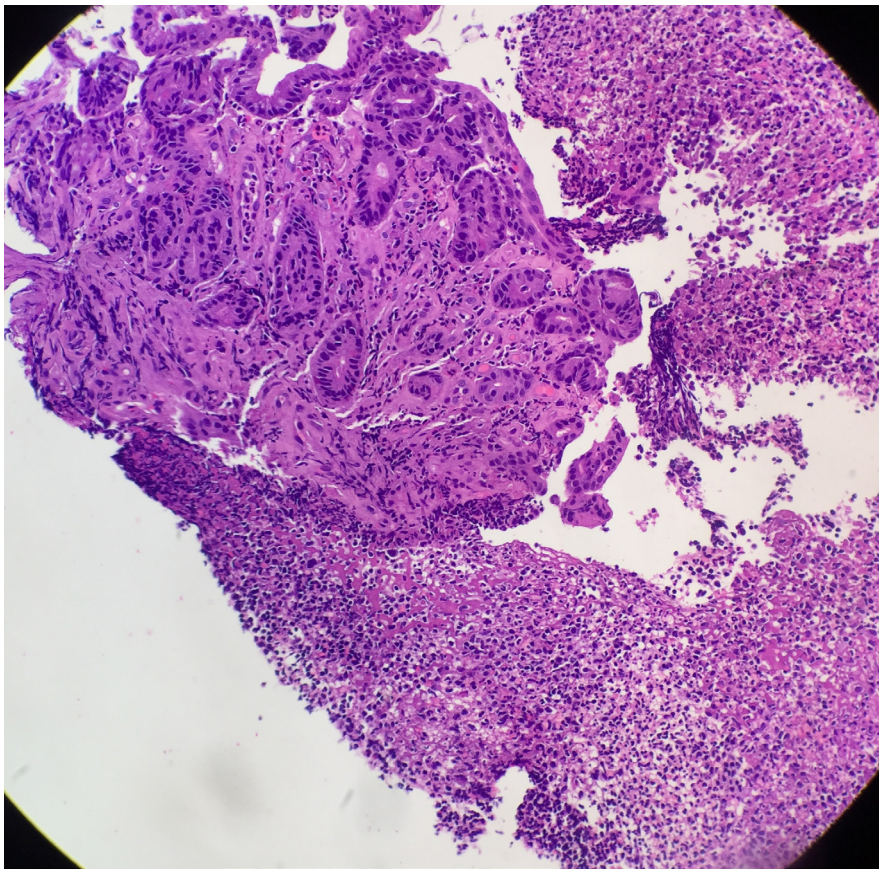


Figure 2
Ulcer biopsy with haematoxylin and eosin stain at 400 x magnification showing ulcerated mucosa and free-lying inflammatory slough with small crypts and partly fibrotic stroma suggestive of ischaemic ulcer prior to hyperbaric oxygen treatment



and no other source of bleeding was found. Subsequently he underwent his third colonic resection and ileo-colonic anastomosis.

He remained stable for the next two years before presenting with iron deficiency without overt bleeding. He did not have any episodes of epistaxis, melaena or haematuria. He was investigated with gastroscopy and duodenal biopsies to rule out other causes of iron deficiency. Gastroscopy was normal and biopsies did not show evidence of coeliac disease or *Helicobactor pylori* infection. He preferred expectant management and was managed with regular blood tests and iron infusions as required. He remained iron deficient and received 10 iron infusions over the subsequent four years.

Thereafter, he developed painless haematochezia prompting a colonoscopy. Repeat colonoscopy showed a 15 mm linear ulcer (Figure 1) with mild ooze at the anastomosis. The ulcer was treated endoscopically with argon plasma coagulation (APC). The anastomotic ulcer biopsies were consistent with an ischaemic ulcer (Figure 2). The ulcer remained refractory to endoscopic treatment with APC and medical treatment with ranitidine, pantoprazole and 5-ASA. The ulcer was non-healing, with persistent ooze, as seen at three follow-up colonoscopies.

This man presented a serious management dilemma. He had had multiple episodes of lower gastrointestinal bleeding secondary to his anastomotic ulcer, three major surgical procedures and endoscopic and medical management over a period of nine years. Despite all this, he continued to have a non-healing ulcer at the anastomosis. The options of continued conservative management with iron and blood transfusions, repeat surgery and hyperbaric oxygen treatment (HBOT) were discussed with him. Following review at a multidisciplinary team meeting and with due consideration of all of the treatment options, he opted for HBOT. He was treated with 30 x 90-minute HBOT sessions at 243 kPa over six weeks in a multiplace chamber, without any complications. Repeat colonoscopy at the completion of HBOT revealed healing of the ulcer, with no further ooze (Figure 3). The patient has not had any overt bleeding and serum ferritin has continued to rise spontaneously over 12 months follow-up.

Discussion

Large bowel anastomotic ulcers commonly present with iron deficiency and gastrointestinal bleeding as in this case.[1,2] Whilst such ulcers may be associated with the use of NSAIDs, development or recurrence of Crohn's disease and malignancy, none of these risk factors were present in this man. Many therapies, including sulfasalazine, sucralfate, 5-ASA, antacid therapy such as ranitidine and protein pump inhibitors have been described for management of anastomotic ulcers with variable success and had all been trialled before he underwent HBOT.[1–5]

HBOT has been shown to improve colorectal ischaemic anastomosis healing in animal models.[6] However, it does not appear to have been reported in the management of ileocolonic anastomotic ulcers in humans. This case demonstrates successful healing of an ileocolonic anastomotic ischaemic ulcer with HBOT after many years of failure of conventional surgical, endoscopic and medical treatment. As far as we are aware this is the first such case reported in the literature. Further studies are needed to assess the efficacy of HBOT for such ulcers.

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Letter to the Editor

Fatty diet, active hydrophobic spots, and decompression sickness

In a study of the effect of a fatty diet on decompression bubbles, based on the responses to a questionnaire regarding daily food consumption, the approximate fat intake for each diver was calculated, taking into account the maximum recommended intake for a person doing his type of work.[1] Following hyperbaric exposure, divers were divided into two groups: 'bubblers' (a minimum of the second level on the Kisman-Masurel scale[2]) and 'non-bubblers'. Bubblers had higher fat consumption than non-bubblers (146 ± 39% versus 92 ± 18%). There was only a small difference in body mass index between the two groups: 26.3 ± 3.3 $kg \cdot m^{-2}$ for bubblers and 24.9 ± 1.9 $kg \cdot m^{-2}$ for non-bubblers. Cholesterol and triglycerides in serum were high in the bubblers (211 ± 39 $mg \cdot dl^{-1}$ and 230 ± 129 $mg \cdot dl^{-1}$, respectively) compared with the non-bubblers (188 ± 34 $mg \cdot dl^{-1}$ and 153 ± 111 $mg \cdot dl^{-1}$, respectively).[1] The authors concluded that a high-fat diet significantly increased the severity of decompression stress in hyperbaric air exposures. However, their explanation that the increased amount of fat in the serum contained more dissolved nitrogen, and that this was the cause of the increase in bubble production, was challenged in a subsequent letter.[3]

Decompression bubbles can expand and develop only from pre-existing gas micronuclei. It is known that nanobubbles form spontaneously when a smooth hydrophobic surface is submerged in water containing dissolved gas. We have shown that these nanobubbles are the gas micronuclei underlying decompression bubbles and decompression sickness.[4] It has been suggested that hydrophobic multilayers of phospholipids on the luminal aspect of blood vessels, which we have termed active hydrophobic spots (AHS), were derived from lung surfactant.[5] The essential components of lung surfactant required to construct the surfactant films, namely dipalmitoylphosphatidylcholine (DPPC) and surfactant proteins B and C, were found in the plasma of man and sheep, while DPPC was also found at the AHS.[4] These findings have borne out the assumption that lung surfactants are the source of the AHS on the luminal aspect of blood vessels. These AHS seem to be stable, and their number and size increase with age as more DPPC settles. Bubbles may evolve at these AHS with decompression.[4] The nanobubbles so formed on the surface of these lamellar layers of phospholipids in divers will expand into venous bubbles on decompression.

The main surfactant in the lung is DPPC (40%), with the presence of additional components including other phospholipids, glycerides, and cholesterol. In the cited study,[1] only serum triglycerides and cholesterol were measured, whereas it may well be that other phospholipids and fatty acids were carried by proteins in the plasma. We suggest that, as with the different elements which compose the layers of surfactant in the lung, some of the additional fatty components carried by the blood will attach themselves to the AHS, thus contributing further to their enlargement. We hypothesise that divers who consume food that is high in fat, and as a result have more fatty components in their blood, will develop more and larger AHS, subsequently becoming bubblers with a higher risk of decompression illness.

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Ran Arieli, The Israel Naval Medical Institute, Israel Defence Forces Medical Corps, Haifa, Israel; Eliachar Research Laboratory, Western Galilee Medical Centre, Nahariya, Israel
Address for correspondence: 12 Klil-Hakhoresh, Rakefet, DN Misgav 2017500, Israel
arieli1940@gmail.com

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Bubbles; Cardiovascular; Surfactant; Risk; Hypothesis; Letters (to the Editor)

Baltic Diving and Hyperbaric Medicine Symposium 2018

The Baltic International Symposium on Diving and Hyperbaric Medicine was held this year at the end of May in Gdynia, a seaport on the Baltic Sea and home to Poland's National Centre for Hyperbaric Medicine. The small Hotel Nadmorski, where the meeting was held and most of the delegates were staying, is situated on the Bay of Gdansk.

The meeting, held in English, which for most attendees was a second language, covered a wide variety of topics in diving and hyperbaric medicine over three days. On the first day, two workshops were held; in the morning on bubble detection in divers and in the afternoon on intensive care in the hyperbaric unit. There were four hours of lectures in each session; eight speakers in the morning and six speakers in the hyperbaric medicine session. Both workshops were excellent.

There is still so much that is still unknown but assumed in the science of bubble detection in relation to the incidence of decompression illness (DCI). The workshop provided an overview of the concept of 'decompression stress' and how difficult it is to study, the use of both Doppler bubble detection and echocardiography, the role of right-to-left shunts in DCI and a good review of the 'life of a bubble'. It was a pity there were not more young scientists and physicians there to benefit from these excellent talks. Bubble detection is a small area that has numerous applications in diving science but has not been utilised to its full potential and it has been difficult for many to implement.

The session on intensive care under pressure was comprehensive and informative with regard to the varying ability of different units to care for ventilated, intubated patients inside a hyperbaric chamber. There are many units around the world that have limited experience and are often reticent to accept critically ill patients for hyperbaric treatment. Likewise, intensive care (ICU) specialists and surgeons are equally reticent to refer such patients for HBOT. Cardiovascular and respiratory problems that may be encountered in the intensive care patient in the chamber were well covered. Devices for patient monitoring and treatment that are safe and able to function under pressure were reviewed by the symposium's convener, Associate Professor Jacek Kot. It was interesting to learn that some European chambers are able to use the Corplus 3 defibrillator in their multiplace chamber. The 'elephant in the room' for many units is the cost of providing 24/7 cover for these emergency ICU patients.

The first day of the main Symposium covered diving medicine where many of the previous day's diving physicians and scientists spoke to a receptive audience on a wide range of topics. Of particular interest was a presentation on recreational diving fatalities in Finland, often technical divers on closed circuit rebreathers (CCR) found days later. Deep CCR diving deaths continue to generate considerable discussion including the cave diving fatality in South Africa from several years ago, and it was interesting to see again the video of this fatality.

The final day was on hyperbaric medicine. Amongst the various topics presented, there was a strong focus on hyperbaric oxygen (HBO) for severe sepsis, the view being expressed that more of these patients should be receiving HBO but this seems to not be getting through to intensive care doctors, otherwise we would be treating many more. The audience was reminded that the European Committee for Hyperbaric Medicine at its 2016 consensus conference promulgated a list of 'accepted' conditions for HBOT, including their rationale and level of evidence. These recommendations are to be found on the EUBS website and published in *Diving and Hyperbaric Medicine*.[1] Amongst conditions currently under study, the potential role of HBO for inflammatory bowel disease is the subject of a prospective, non-randomised European multicentre trial.

The National Centre for Hyperbaric Medicine is a large facility with clinical and interconnecting research chambers also used by the Polish Navy. Located outside the building are a number of old chambers that give an 'authentic' edge to this medical building in the middle of Gdynia. During the meeting, the local university rector, Professor Michal Gruchala, thanked Dr Zdzislaw Sicko, the former Head of the Hyperbaric Centre for his long-lasting work, whilst Dr Jarosław Pinkas, the former Polish Secretary of State for Health, received the traditional hard-hat diver's knife and a plaque for his support – an honour rarely deserved by a politician!

The Gdynia National Centre for Hyperbaric Medicine held an excellent symposium in a lovely location. Gdynia was easy to get to and an enjoyable place to visit. There are no excuses for not attending another unless you are concerned about your liver. You will need to be diligent though, as there were over 18 hours of talks if you planned to attend both the workshops and the main meeting. The informal exchanges during the social gatherings undoubtedly enhanced the overall experience for this 'rapporteur'.

Reference

1 Mathieu D, Marroni A, Kot J. Tenth European Consensus Conference on Hyperbaric Medicine: recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment. Diving Hyperb Med. 2017;47:24–32. PMID: 28357821. Kot J. Correction. Diving Hyperb Med. 2017;47:131–2. PMID: 28641327.

Fiona Sharp, Department of Diving and Hyperbaric Medicine, Fiona Stanley Hospital, Murdoch, WA, Australia
sharpief@doctors.org.uk

Book review

Gas bubble dynamics in the human body

Saul Goldman, J Manuel Solano-Altamirano, Kenneth M LeDez

320 pages
Academic Press, 2017
Available from: https://www.elsevier.com/books-and-journals
eBook ISBN: 9780128105207
Paperback ISBN: 9780128105191
Price: 105.00 USD

In vitro gas bubbles arise in decompression, barotrauma, and clinical accidents involving vascular interventions. Left untreated, bubbles eventually resolve but can cause ischaemia, infarction, pain, inflammation, and permanent disability. Consequences are well-known to divers and hyperbaric clinicians. This book provides an overview of gas bubble physics and medical applications. The authors are two physical chemists and a hyperbaric physician. About two-thirds is devoted to mathematical models and one-third to clinical applications and novel case reports. Effort has been made to appeal to a broad readership by including ten chapters that cover both theoretical and practical aspects. Although some of the bubble models may be found in scientific journals, much of the content is original and not available elsewhere.

The book begins with descriptions of the circulation, cell membranes, and microparticles. Covered are importance of bubble shape, blood flow in vessels, arterial bifurcations, shunts, and trans-pulmonary passage. Bubbles may be static or circulating, spherical or sausage-shaped, and subject to surface tension. There is discussion of microparticles and microbubbles that are the nuclei for gas phase formation; however, the major focus of the book is on how bubbles behave once they are present, and the physical principles underlying their management.

Next is a rigorous foundation of physical concepts. We see how Henry's law may be derived by equating chemical potentials for gas and dissolved phases, and that gas solutes move along the gradients of chemical potential for each species. Sections entitled "*Greater Detail*" or "*For Math Mavens*" go into more detail, but may be skipped by those less inclined. Sections entitled "*Medical Matters*" introduce relevant clinical applications, such as persistent (patent) foramen ovale, decompression sickness and arterial gas embolism.

A core application of bubble modelling is calculating rates of growth and resolution, and estimating bubble lifetimes, for example, in relation to circulation time of a bubble transiting from a right-to-left shunt to the brain or inner ear. The diffusion equation expressed in spherical coordinates, the Laplace-Young law of surface tension, and Fick's law are used to derive the Epstein-Plesset equation which tracks bubble growth and dissolution, allowing lifetimes to be estimated. This derivation is extended to account for convection and the shear modulus of an elastic medium. Solutions are obtained in a three-region geometry by matching gas fluxes at concentric boundaries. Including the shear modulus in the diffusion equation allows bubbles to persist despite local undersaturation, thus extending their lifetimes.

To deeply understand mathematical models, one has to work with them. A hands-on approach to modelling is encouraged by providing problems at the end of chapters with solutions at the back. Algebraic solutions dominate rather than numerical methods, although the latter are used in some examples, such as bubble growth in repetitive diving. The authors propose a model to explain inner ear decompression sickness and symptoms of Taravana in breath-hold divers, when a circulating bubble survives long enough to enter supersaturated brain tissue.

Dissolved gas transfer is discussed, including deterministic Haldane methods and M-values, followed by probabilistic risk functions derived from failure analysis. The complexity of gas movement in real tissues has led to many types of compartment models. Transfer coefficients between compartments give rise to coupled linear equations whose solutions may be obtained using matrix methods to extract eigenvalues. Multiple exchanging compartments may interface with a single risk-bearing compartment, such as when modelling pain-only bends in a joint capsule. These so-called mammillary models are used in pharmacokinetics, where peripheral compartments exchange solute with a single central compartment. Data fitting of outcomes to models using least-squares and maximum-likelihood methods to minimize error is then discussed, along with approaches to parameter optimization, including the Levenberg-Marquardt method and simulated annealing.

The book is rounded out by chapters covering the "*Evils that bubbles do*", such as vascular obstruction, gas emboli in cerebral and coronary arteries, spinal cord and inner ear ischaemia, and osteonecrosis. Also discussed are inflammatory mediators, ischaemia-reperfusion injury, and bubbles that arise during cardiac surgery. Treatment of gas bubbles is then discussed in conjunction with models illustrating the effect of hyperbaric oxygen on resolution, and handling diffusion of gases around sausage-shaped bubbles in a cul-de-sac zone.

This book will appeal to physical scientists and students interested in developing new approaches to modelling gas exchange in biological media, mathematicians interested

in expanding models and solution methods and clinicians interested in how gas models relate to clinical cases. Readers tasked with designing decompression schedules will not find specific algorithms here but will find approaches to modelling that may be adapted, with potential for new ideas and hybrid methods.

John Fitz-Clarke, Department of Emergency Medicine, Dalhousie University, Canada
jfitzclarke@eastlink.ca

Key words
Bubbles; Models; Diving tables; Decompression illness; Book reviews

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The **Diving and Hyperbaric Medicine Journal** website is at

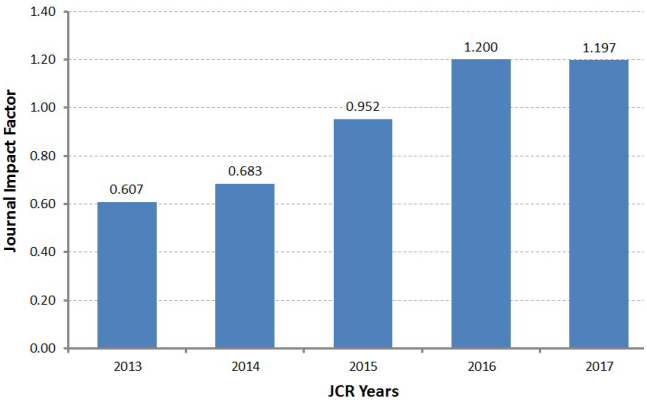
www.dhmjournal.com

The latest issues, embargoed for one year, are available for the personal use of society members only. Access is via your SPUMS or EUBS website log-in and password. Please respect that these are restricted access and to distribute their contents within one year of publication is a breach of copyright.

Older issues (from March 2007 to September 2017); articles for immediate release into the public domain; contents lists and the Abstracts of the most recent (embargoed) issues; information about submitting to the Journal; profiles of the Editorial Board and useful links are to be found on the site. This will be expanded progressively as resources allow.

Your membership ensures the continued publication of DHM – thank you for your support of SPUMS and EUBS.

Five-year Impact Factor report for *Diving and Hyperbaric Medicine*



The Diving and Hyperbaric Medicine Journal is now on Facebook

Like us at:
https://www.facebook.com/divingandhyperbaricmedicine/

Divers Emergency Service/DAN AP Telemedicine Scholarship 2018

It is with great pleasure we announce that the winner of the inaugural Telemedicine Scholarship is Dr Adrian Winsor, who will be presenting his work at the TRICON2018 in Durban in September.

The Telemedicine Scholarship is an award of AUD4,000 to support attendance of a trainee or recent trainee to the next SPUMS Annual Scientific Meeting. Please note that the 2019 SPUMS ASM will be in May 2019. Application and criteria for the 2019 Scholarship will be available from December 2018 and will be advertised.

The Divers Emergency Service (DES) telephone provides 24-hour advice for diving and underwater emergencies. Recognising that most doctors have no training in diving medicine, the volunteers providing this service have specific training and work in diving and hyperbaric facilities around Australia. This service has been in operation since 1986 and is supported by the Divers Alert Network Asia Pacific.

Enquiries to: david.wilkinson@sa.gov.au

Publications database of the German Diving and Hyperbaric Medical Society (GTÜeM)

EUBS and SPUMS members are able to access the German Society's large database of publications in diving and hyperbaric medicine. EUBS members have had this access for many years. SPUMS members should log onto the SPUMS website, click on "*Resources*" then on "*GTÜeM database*" in the pull-down menu. In the new window; click on the link provided and enter the user name and password listed on the page that appears in order to access the database.

Notices and news

SPUMS society information and news is to be found mainly on the society website: www.spums.org.au

ANZ Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine 2019

Dates: 18 February–01 March
Venue: Esplanade Hotel, Fremantle, Western Australia
Cost: AUD2,600 (inclusive of GST)

Course Conveners: Ian Gawthrope and Neil Banham
The Course content includes:
- History of diving medicine and hyperbaric oxygen
- Physics and physiology of diving and compressed gases
- Presentation, diagnosis and management of diving injuries
- Assessment of fitness to dive
- Visit to RFDS base for flying and diving workshop
- Accepted indications for hyperbaric oxygen treatment
- Hyperbaric oxygen evidence-based medicine
- Wound management and transcutaneous oximetry
- In-water rescue and management of a seriously ill diver
- Visit to HMAS Stirling
- Practical workshops
- Marine Envenomation

Contact for information:
Sue Conlon, Course Administrator
Phone: +61-(0)8-6152-5222
E-mail: fsh.hyperbaric@health.wa.gov.au

The SPUMS website is at
www.spums.org.au

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

The latest issues of *Diving and Hyperbaric Medicine* are via your society website login.

SPUMS Facebook page

Remember to 'like' us at:

http://www.facebook.com/pages/SPUMS-South-Pacific-Underwater-Medicine-Society/221855494509119

Australian and New Zealand College of Anaesthetists Diving and Hyperbaric Medicine Special Interest Group

The new Diploma of Advanced Diving and Hyperbaric Medicine was launched on 31 July 2017. Those interested in training are directed to the ANZCA website http://www.anzca.edu.au/training/diving-and-hyperbaric-medicine.

Training

Documents to be found at this site are:
- Regulation 36, which provides for the conduct of training leading to the ANZCA Dip Adv DHM, and the continuing professional development requirements for diplomats and holders of the ANZCA Certificate of DHM;
- ANZCA Advanced DHM Curriculum which defines the required learning, teaching and assessment of the diploma training programme; and
- ANZCA Handbook for Advanced DHM Training which sets out in detail the requirements expected of trainees and accredited units for training.

Accreditation

The ANZCA Handbook for Advanced DHM accreditation, which provides information for units seeking accreditation, is awaiting approval by Standards Australia and cannot yet be accessed online. Currently six units are accredited for DHM training and these can be found on the College website.

Transition to new qualification

Holders of the Certificate of DHM and highly experienced practitioners of DHM are eligible for recognition of prior experience towards the ANZCA Dip Adv DHM, as outlined in the guidelines for the transitional award of diploma in Regulation 36. Applications for credit must be made in writing to the ANZCA TA unit and must be submitted prior to 31 January 2019.

All enqueries should be submitted to dhm@anzca.edu.au.

Suzy Szekely, Chairperson, ANZCA DHM SIG
Suzy.Szekely@health.sa.gov.au

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: They must

1 be medically qualified, and remain a current financial member of the Society at least until they have completed all requirements of the Diploma;
2 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 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 submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval before commencing the research project;
5 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 (EO) 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 EO 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 and clinical research are acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis and if the subject is extensively researched 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, including case series, 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 proposal is approved prior to commencing research.

Projects will be deemed to have lapsed if:

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

For unforeseen delays where the project will exceed three years, candidates must explain to the EO by email why they wish their diploma project to remain active, and a three-year extension may be approved. If there are extenuating circumstances why a candidate is unable to maintain financial membership, then these must be advised by email to the EO 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 January 2016, the SPUMS Academic Board consists of:

Dr David Wilkinson, Education Officer, Adelaide;
Professor Simon Mitchell, Auckand;
Dr Denise Blake, Townsville.

All enquiries and applications should be addressed to:
David Wilkinson
education@spums.org.au

Key words
Qualifications; Underwater medicine; Hyperbaric oxygen; Research; Medical society

The SPUMS President's message

David Smart, President SPUMS

In June and July 2018 the world witnessed an incredible feat of international cooperation and logistics which culminated in the successful rescue of 12 trapped schoolboy soccer players and their coach from Tham Luang Nang Non Cave in Thailand. The actual circumstances were almost beyond belief and way outside the comfort zones of most people including divers. The whole process of the rescue was a remarkable achievement by an enormous number of people and organisations. A key role was played by Richard "*Harry*" Harris, who is a long-time SPUMS member and extraordinary cave diver, and his dive buddy Craig Challen, also an Associate member of SPUMS. Richard's combined skill and experience in anaesthesia and cave diving was crucial to the safe return of the trapped children and their coach to their loved ones. "*Harry*" is an outstanding high achiever in life and well known for his modesty and low key approach. On behalf of SPUMS we offer our congratulations to Richard and Craig. They were awarded the Star of Courage and Order of Australia Medal – richly deserved accolades for their selfless courage.

SPUMS also offers Richard our sincere condolences for the loss of his father at the very time he was selflessly working on the rescue. As SPUMS President, I am experiencing a similar loss at this point in time and I fully empathise with his need for some quiet private time in coming months. When and if the time is right, all of us in SPUMS are keen to hear Richard's story of the rescue. It is our privilege to have him as a member of SPUMS.

Key words
Medical society; General interest

SPUMS 48th Annual Scientific Meeting 20–26 May 2019 Solomon Kitano Mendana Hotel, Honiara, Solomon Islands

Theme

"***Old divers, bold divers but no old, bold divers***"
Cardiovascular health risk assessment and diving

Guest Speaker: A/Professor Nigel Stuart Jepson
Senior Staff Specialist and Director, Cardiac Catheterization Laboratories, Eastern Heart Clinic, Prince of Wales Hospital, Sydney, Australia

Scientific conveners: David Smart and Michael Bennett
Convener: Catherine Meehan
cmeehan@mcleodstmed.com.au
More information coming soon on the SPUMS website

SPUMS 49th Annual Scientific Meeting

Preliminary announcement

April 2020
Tutukaka, Northland, New Zealand

Guest Speaker: Richard Harris, Adelaide

Convenor: Greg van der Hulst

Honours for Richard Harris and Craig Challen

Richard Harris (left), an Adelaide anaesthetist and member of SPUMS, and Craig Challen (right), a retired Perth vet and associate member of SPUMS, were awarded a rare double honour at Government House, Canberra on 24 July 2018. The Star of Courage, the second highest Australian bravery decoration, recognises their courage in helping free the 12 boys and their soccer coach from the flooded cave in Thailand. They join an elite group of 162 Australians who have received the Star of Courage. They also received the Medal of the Order of Australia (OAM) for the specialist roles they played in the rescue mission.

In addition, a member of the Royal Australian Navy clearance diving team and six Australian Federal Police officers from the AFP Special Response Group were awarded the OAM and Bravery Medals for their work in the hazardous mission. Many other Thai military personnel and cave divers from around the world also contributed to this extraordinary, unique rescue, particularly the two British divers who found the children an incredible feat of cave diving in awful conditions.

EUBS Notices and news

EUBS notices and news and all other society information is now to be found mainly on the society's website: www.eubs.org

Jacek Kot
President, EUBS

I wanted to say one final thank you for the honour of serving as the 15th President of the EUBS. When I started in this role in 2015, the Society had already launched new projects to better suit the demands of the times, including formal cooperation with national societies in Europe and elsewhere, supporting hyperbaric 'newbies' with research grants, converting from paper to the electronic edition of the Journal and promoting inter-continental meetings.

I had then and I still have a vision of strong associations between different European bodies serving diving and hyperbaric medicine in order to strengthen our resources. And even if we think that we can change the world by ourselves and later on we are disappointed when the progress seems slow, we need to remember that any European society or organisation is not the project of just one person, but we all own it. On the one hand, it is a burden which needs great momentum to be moved along, on the other, when carried out by mutual agreement and cooperation, it will create new stability. This is still an open project...

In the meantime, I would express my sincere gratitude and appreciation to all Executive Committee members for their dedicated work for the Society. Special thanks go to Peter Germonpré, the Honorary Secretary, for his continued support – he is always there when needed.

I would like to conclude that the EUBS financial and membership situations are stable, research and travel grants are offered to youngsters, annual scientific conferences are planned several years ahead, the Executive Committee is reliable, affiliations are trusted and the Journal is shining. So we can continue our work and enjoy our EUBS membership. I look forward to continuing to serve the Society for another six years as Immediate Past President and Past President.

Key words
Medical society; General interest

EUBS 2019 – First Announcement

Even as TRICON2018 is finishing, it is already time to mark your calendars for the 2019 EUBS meeting! The dates will be 09–12 September, 2019 and it will take place in Tel Aviv, Israel, at the David Intercontinental Hotel. The conference will be jointly organised with the International Conference on Hyperbaric Oxygen and the Brain. This period has been chosen as it is after the summer break but just before the high holidays in Israel. Tel Aviv is an exciting hypermodern coastal city, with top-notch medical research and treatment facilities, but also bustling with beaches, restaurants and nightlife.

The conferences will be hosted by the Israeli Society for Hyperbaric and Diving Medicine, and the dedicated conference website www.eubs2019.com is already accessible (registrations and abstract submissions will start from October 2018).

EUBS Member at Large

As the EUBS Annual Scientific Meeting has just been held in South Africa, EUBS membership will now have elected a new Member-at-Large, and Karin Hasmiller will have left office as Member at Large 2015. However, she will remain working for EUBS as a member of the DHM Journal Governance Committee and in the Research & Education Committee. ExCom extends their thanks to Karin for her work for the Society thus far. We will also have elected a new Vice-President, and Ole Hyldegaard has taken over the role of President of EUBS from Jacek Kot. We thank Jacek for his excellent work and are happy that he will remain in the ExCom for some time to come.

The EUBS website is at <www.eubs.org>

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

The latest issues of *Diving and Hyperbaric Medicine* are via your society website login.

Scott Haldane Foundation

Dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 250 courses over the past 20 years, increasingly targeting an international audience with courses worldwide. The courses Medical Examiner of Diver (part I and II) and SHF in-depth courses, as modules of the level 2d Diving Medicine Physician course, fully comply with the ECHM/EDTC curriculum for Level 1 and 2d respectively and are accredited by the European College of Baromedicine (ECB).

SHF Course Calendar 2018
09–16 November: Medical Examiner of Divers (Level 2d), Kuror, Palau
16–23 November: Dangerous marine animals (Level 2d), Kuror, Palau
23–30 November: Dangerous marine animals (Level 2d), Kuror, Palau
SHF Course Calendar 2019
January: Refresher course, "*Organization diving medical*", The Netherlands
29-30 March: Medical Examiner of Divers part 1, Zeist , NL
4, 5 and 6 April: Medical Examiner of Divers part 2, AMC, Amsterdam, NL

On request: Internship different types of diving (DMP), NL
On request: Internship HBOT (DMP certification), NL/Belgium
The course calendar will be supplemented regularly.

For the latest information: www.scotthaldane.org

20th International Congress on Hyperbaric Medicine 2020

Dates: 13–16 September 2020
Venue: Rio de Janeiro, Brazil
For preliminary information contact:
Dr Mariza D'Agostino Dias
Email: mariza@hiperbarico.com.br

German Society for Diving and Hyperbaric Medicine (GTÜeM)

An overview of basic and refresher courses in diving and hyperbaric medicine, accredited by GTÜeM according to EDTC/ECHM curricula, can be found on the website:
http://www.gtuem.org/212/Kurse_/_Termine/Kurse.html

The Capita Selecta Diving Medicine annually offers symposia on diving medicine presented by speakers of national and international renown to a multinational audience of diving physicians, paramedics and highly educated diving instructors. The level of the presented material is advanced, i.e., Level 1 and 2d, and often beyond that. The lectures are in English.

27 October 2018: The ageing diver
Topics include the physiology of the healthy, ageing heart, lung and muscular system, clinical aspects of diving – cardiology, ageing of sensory system and the brain, DCI and age, the medical exam of the older diver.

Speakers include: Olga de Bakker, NL; Jacques Regnard, FR; Rienk Rienks, NL and Nico Schellart, NL

Register at: http://www.capitaselectaduikgeneeskunde.nl

30 March 2019: Diving medicine of women, children and divers with a disability

Speakers include: Selina Haas, AT, Ulrike Preiml, AT and Guy Vandenhoven (BE)

British Hyperbaric Association Annual Scientific Meeting 2018

Dates: 08–09 November
Venue: Danubius Hotel Regent's Park, London
07 November: Historical event
Hosts: London Diving Chamber
http://www.londondivingchamber.co.uk/
Further information:
http://www.ukhyperbaric.com/

Hyperbaric oxygen lectures

Welcome to: http://www.hyperbaricoxygen.se/
This site offers publications and high-quality lectures from leading investigators in hyperbaric medicine. Please register to obtain a password via email. Once registered, watch online, or download to your iPhone, smart device or computer for later viewing.
For information contact: folke.lind@gmail.se

Royal Australian Navy Medical Officers' Underwater Medicine Course 2018

Dates: 08–19 October
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. Emphasis is placed on the contraindications to diving and the diving medical assessment, together with the pathophysiology, diagnosis and management of common diving-related illnesses. The course includes scenario-based simulation focusing on the management of diving emergencies and workshops covering the key components of the diving medical.

Cost: AUD1,355 without accommodation
(tbc with accommodation and meals 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)2-9647-5117
E-mail: Rajeev.Karekar@defence.gov.au

The Science of Diving

Support EUBS by buying the PHYPODE book "*The science of diving*". Written for anyone with an interest in the latest research in diving physiology and pathology. The royalties from this book are being donated to the EUBS.
Available from: Morebooks https://www.morebooks.de/store/gb/book/the-science-of-diving/isbn/978-3-659-66233-1

The Historical Diving Society 2018 Annual Conference

Date: 03 November
Venue: RNLI College, Poole, UK

The HDS was formed in the UK in 1990 with the aim of preserving and protecting diving heritage. Since then the Society has grown into an international organisation with affiliated national societies across the world. It produces a newsletter, the *Historical Diving Times*, and the *International Journal of Diving History*. It publishes facsimile monographs of important works on diving.

Email: chairman@thehds.com
Websites: www.thehds.com
www.divingmuseum.co.uk

Royal Adelaide Hospital Medical Officers' Course, Diving and Hyperbaric Medicine 2018

Dates: 05–16 November
Venue: The Royal Adelaide Hospital, Adelaide
Cost: AUD2,500.00 (inclusive of GST)

Course Conveners: David Wilkinson and Suzy Szekely
Invited faculty includes: Professors Michael Bennett and Simon Mitchell
The course content includes:

- Physics and physiology of diving
- Recreational fitness-to-dive
- Occupational fitness-to-dive
- Decompression illness and non-dysbaric injuries
- Medical management and return to diving
- Technical and professional diving
- Marine envenomation
- Introduction to hyperbaric medicine

Contact for information:
Ms Lorna Mirabelli, Course Administrator
Phone: +61-(0)8-8222-5116
E-mail: Lorna.Mirabelli@sa.gov.au

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Companies and organisations within the diving, hyperbaric medicine and wound-care communities wishing to advertise their goods and services in *Diving and Hyperbaric Medicine* are welcome. The advertising policy of the parent societies appears on the journal website:
www.dhmjournal.com

Details of advertising rates and formatting requirements are available on request from:
E-mail: editorialassist@dhmjournal.com

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

Diving and Hyperbaric Medicine: Instructions for Authors

Diving and Hyperbaric Medicine (DHM) is the combined journal of the South Pacific Underwater Medicine Society (SPUMS) and the European Underwater and Baromedical Society (EUBS). It seeks to publish papers of high quality on all aspects of diving and hyperbaric medicine of interest to diving medical professionals, physicians of all specialties, members of the diving and hyperbaric industries, and divers. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing.

Address: The Editor, Diving and Hyperbaric Medicine, P O Box 35, Tai Tapu, Canterbury 7645, New Zealand
Email: editor@dhmjournal.com
Phone: +64-(0)3-329-6857
Mobile: +64-(0)27-433-2218
European Editor: euroeditor@dhmjournal.com
Editorial Assistant: editorialassist@dhmjournal.com
Information: info@dhmjournal.com

Contributions should be submitted electronically by following the link:
http://www.manuscriptmanager.net/dhm
There is on-screen help on the platform to assist authors as they assemble their submission. In order to submit, the corresponding author needs to create an 'account' with a user name and password (keep a record of these for subsequent use). The process of uploading the files related to the submission is simple and well described in the on-screen help, provided the instructions are followed carefully. The submitting author must remain the same throughout the peer review process.

Types of articles

DHM welcomes contributions of the following types:

Original articles, Technical reports and Case series: up to 3,000 words is preferred, and no more than 30 references (excluded from word count). Longer articles will be considered. These articles should be subdivided into the following sections: an **Abstract** (subdivided into Introduction, Methods, Results and Conclusions) of no more than 250 words (excluded from word count), **Introduction, Methods, Results, Discussion, Conclusions, References, Acknowledgements**, **Funding** sources and any **Conflicts of interest**. **Legends / captions** for illustrations, figures and tables should be placed at the end of the text file.

Review Articles: up to 5,000 words is preferred and a maximum of 50 references (excluded from word count); include an informative **Abstract** of no more than 300 words (excluded from word count); structure of the article and abstract is at the author(s)' discretion.

Case reports, Short communications, Work in progress reports, etc: maximum 1,500 words, and 20 references (excluded from word count); include an informative **Abstract** (structure at author's discretion) of no more than 200 words (excluded from word count).

Educational and historical articles, Commentaries, Consensus and other meeting reports, etc., for occasional sections may vary in format and length, but should generally be a maximum of 2,000 words and 15 references (excluded from word count); include an informative **Abstract** of no more than 200 words (excluded from word count).

Letters to the Editor: maximum 600 words, plus one figure or table and five references.

Formatting of manuscripts

All submissions must comply with the requirements set out in the full instructions on the DHM website. Non-compliant manuscripts will be suspended whilst the authors correct their submission. Guidance on the general structure for the different types of articles is given above.

The following pdf files are available on the DHM website to assist authors in preparing their submission:

- Instructions for authors
- DHM Key words 2018
- DHM Mandatory Submission Form 2018
- Trial design analysis and presentation
- EASE participation and conflict of interest statement
- English as a second language
- Guideline to authorship in DHM 2015
- Helsinki Declaration revised 2013
- Is ethics approval needed?

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+64-9-445-8454 (International)

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

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DAN ASIA-PACIFIC DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related incidents. 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 e-mail: research@danasiapacific.org

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

NFDIR is an ongoing study of diving incidents. 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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