Diving and Hyperbaric Medicine

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Extreme breath-hold diving - Part II

Perfluorocarbons – promise in decompression sickness?

Nitric oxide as a marker of inflammation in diving

Instrumenting the breath-hold diver

In-dive rehydration for the working diver

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PURPOSES OF THE SOCIETIES

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

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The Editor's offering

Since Peter Germonpré wrote his Presidential message, further tragedy has struck from earthquake in Chile, severe floods and storms, and, at the other extreme, drought and famine in many parts of the world. I am aware of a number of members of both societies who are involved in helping with the human suffering consequent on these natural disasters, and I am sure that I speak for all in wishing them success and safety in their emergency medical work.

Heparin, steroids, anti-platelet drugs, non-steroidal anti-inflammatories (NSAIDs) and lignocaine have all been investigated and enthusiastically advocated by some in the search for effective drug-based treatments of decompression illness (DCI). Only NSAIDs, in a single randomised controlled study (RCT) that has not been replicated, have shown any potential benefit as an adjunctive therapy to hyperbaric oxygen, and then only in terms of reducing the number of hyperbaric treatments required (by at least one, NNT of five patients) to reach a plateau of recovery, rather than any improvement in recovery itself. Even fluid resuscitation has no convincing evidence to support its use, though this is a bit like saying that mechanical ventilation in the critically ill patient with acute respiratory failure has not proved to be life-saving! Other interventions have almost no clinical outcome evidence of any sort. Adjunctive therapy has been reviewed recently for us by Richard Moon, and in a Cochrane review by Michael Bennett et al.[1,2]

The remarkable gas-carrying properties of perfluorocarbons offer new and exciting prospects for acute medicine in several clinical situations. The long history of their research development and current theoretical and experimental evidence for their use in DCI are provided by Bruce Spiess in two articles in this issue. What is not mentioned are the very considerable difficulties that present themselves in developing a meaningful, multi-centre, prospective RCT protocol to assess their role in treating DCI, just as was the case with lignocaine.[3] These agents have only been tested in DCI in animal models and may carry a potential for an increased risk of acute oxygen toxicity with hyperbaric oxygen therapy. Nevertheless, we now have the prospect of a useful adjunctive therapy for the future, particularly in severe cases and for first aid treatment in remote sites.

Predicting the future is a risky pastime. In her first review of extreme breath-hold diving, Professor Schagatay calculated that the likely limit to static apnea duration was probably around 11 minutes plus, but cautioned that not all the potential contributing factors had been accounted for in this estimate.[4] Soon after, the static apnea world record was pushed out to 11:35 min, with elite divers predicting that the final limit may be in excess of 13 minutes! Her second article probes the physiology of breath-hold swimming exercise (without diving to depth), and reviews the safety procedures in place during these swimming-pool-based competitions. Linked to this work and reported in this issue, is the development of miniaturised, computer-based, pressure-resistant technology that will allow physiological monitoring of the free-swimming diver. Those of you in the Antipodes who follow cricket will have been entertained by the 'Heart Tracker' monitoring on TV of individual players during international matches and marvelled at the ability of young athletes to take their heart rate into the 200s during play, especially if they are a fast bowler! Such technology, of course, has a far more important role in advancing our understanding in both physiological research and clinical medicine, and is likely to become increasingly sophisticated in the future.

In editing this journal, we draw satisfaction from helping authors to clarify their writing. Now that we are receiving a steadily increasing number of submissions from authors whose first language is not English, this has assumed greater importance. Even if you are not confident with written English, this office is happy to assist once a paper has been provisionally accepted for publication. Often, the reviewers, as well as critiquing the content of papers, contribute to this. Language is a dynamic, constantly changing beast, and English is no exception. A recent example for me is having to abandon (with much heartache!) "apnoea" in favour of the Americanised spelling, "apnea", which is now used in well over 80% of all publications in this field.

The executive committees of EUBS and SPUMS have recently reached agreement on their continued cooperation in the publication of this journal. The writer has been appointed as Editor for a further three years through 2012. I hope to serve the societies well and to the best of my abilities, and I look forward to hearing the views of any of our readers.

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Michael Davis

Front page photo of a diver performing a static apnea at the 2008 World Championships was taken by Mr Kimmo Lahtinen of Finland. Kimmo is the current President of AIDA International and has a photographic website <www. freediving.pictures.fi> well worth viewing.

The Presidents' pages

Peter Germonpré
President, EUBS

Dear friends,

As I write this column, the full extent of the human and material damage caused by the earthquake in Haiti is just beginning to unfold. Relief forces and goods are being rushed into a devastated country, struggling with the unforeseeable chaos and misery. The loss of tens of thousands of lives seems only the beginning of a tragedy that will leave its mark for years to come.

As in the rest of the world, all of us in Europe are making a financial effort to support the aid organisations in providing medical and sanitation help. The expressions of solidarity are heart-warming: small kids donating part of our luxury to help children at the other end of the globe, politicians donating part of our luxury to help. It is all most of us can do from here. Will it be enough? Bad question to ask. Of course it will not ever be enough, but it will help make a start to the rebuild. Friends and colleagues who flew out to Haiti as part of aid teams speak about the hope and courage that live among the despair and grief.

Catastrophes like these may help us to realise how well off we are, how lucky that we do not have to struggle for the basic necessities of human existence. They may help us to realise that our "Best wishes for the New Year" better be sincere and not stay hollow words, forgotten when our fellow man/woman/child is in need.
Even drops of water on a hot plate cool it down a bit.

At the end of 2009, both the EUBS and SPUMS Executive Committees have reviewed the two-year trial period of our joint journal *Diving and Hyperbaric Medicine*, and I am happy to confirm that our general appreciation is more than positive. We are currently consolidating the merger and will continue to operate the Journal as a product of both scientific societies. Spanning the entire globe, our Journal has the potential to become a true 'global journal of diving and hyperbaric medicine' (we might need a new title in a couple of years…).

No news yet about the final outcome of our Medline application – a quick poll among ExCom members yields about a 50-50 positive/negative expectation. Possibly when you read this, the decision will have been made. Regardless of the outcome, DHM will have an Impact Factor as from 2010 – I am sure our Editor in Chief will inform you of this in his editorial. Regardless of the outcome, our efforts in producing good papers should continue and even increase. Do you feel you are not up to the task of writing a paper? Have you never written a scientific paper? I am not aware of any statistics that show how large a proportion of any one group of medical specialists actually has written a scientific paper, but surely 100% could make the effort? You will find that, once you have written one, it is easier to write the second. Just look over the contributions in this and previous issues and convince yourself that you can do this too.
Even small grains of sand finally make up a mountain.

Our next Annual Scientific Meeting, in Istanbul, Turkey, is coming ahead at full speed; mark and block the different dates and deadlines now. The organising and scientific committees are making a huge effort to compose a great scientific and social programme, but need your help and input. Our ASM is a unique occasion to practise face-to-face information exchange (also called 'talk over a drink'), renew or make new friendships and increase your knowledge and that of your colleagues. Encouragements to participate are many: nice city, good climate, reasonable price, good science. Please do not forget to renew your membership in time, and encourage your colleagues to become a member of EUBS as well. You can easily do so by visiting the EUBS website. If the informatics let you down, just ask for help. Send us an e-mail. Sometimes computer programmes just mess up – they can be mended.
Many individual people are needed to forge a league.

As our membership year nears its end, it is also time to recruit a new Member-at-Large to assist in the work of the Executive Committee. Here again (and for the last time in this column) I appeal to you, EUBS members, to nominate someone you feel is capable of contributing to the continuing development of EUBS as a society, and its interactions with other societies. We need candidates for a new three-year term. Look around you (even if it is in the mirror) and give us a name. How to proceed is outlined in our newsletter section, later in this journal.
Every long journey starts with a first small step forward.

All the best to everyone,

Peter Germonpré

Michael Bennett
President, SPUMS

Another year has passed in the life of SPUMS and, as so many before, it has been a year of change. Four big things have captured the attention of your committee since I last wrote – and all will have a direct effect on many of our members. I will touch on each of these below, but equally important and exciting is the news about one member in particular – Commodore Robyn Walker. Robyn was one of our most popular and effective presidents until we could persuade her no longer, and has now been recognised for the great qualities she brings to her 'real' job with the RAN. CMDR Walker is currently serving as the Director General Garrison Health Support within Joint Health Command and was named in the Australia Day honours list as a Member of the Order of Australia (AM) - Military Division for exceptional service as a medical officer in the Australian Defence Force. You are thoroughly deserving, Robyn, and congratulations on behalf of the members and friends of SPUMS.

To more general matters, you will all be aware that this year we will hold our 39th ASM at the Berjaya Resort on Redang Island in Malaysia in conjunction with the Asian Diving and Hyperbaric Medicine Association. Our convenor, Glen Hawkins, has been working tirelessly on this one. No-one will have failed to notice that arrangements have been a bit late coming out this year. The delays have been well and truly outside Glen's control and include the resort dumping us due to a major renovation that would make the resort unsuitable for us, and extensive problems with the new web site (vide infra). An alternative site suitable for both organisations was proving a real headache and when Berjaya agreed to delay their plans until after our meeting it seemed the prudent thing to accept – on our terms.

So, as I write, registrations and travel/accommodation bookings are open on the web site. Several aspects make this a particularly attractive meeting. The collaboration with ADHMA allows us to offer two outstanding guest speakers and a chance to widen our horizons, while the diving will be in pristine waters where the fish have had to put up with a lot fewer divers than at many of our previous venues. Two innovations this year are the two update sessions (one each in diving and hyperbaric medicine), open to anyone who would like to get a more rounded grasp on these fields than is possible in a scientific meeting, and a totally flexible dive package – dive as much or as little as you want (OK – within reason). Both these are designed to attract some new people along to join the old lags. Get in contact with Glen for any further details and to submit your abstracts.

The second 'big thing' this year is the upgrading of our web site to allow on-line membership payments, ASM bookings and open forums on matters of interest to the membership. In addition, the Diving Doctors List has been substantially upgraded with details, location maps and whether or not the individuals listed will consider dive medicals in those less than 16 years old. The changes to the list are a response to many complaints from potential divers about the difficulty finding an appropriate physician. This should be a thing of the past with the information now available. There are lots of other things to explore, including the new SPUMS guide to the pre-diving medical with all the recent changes accepted at last year's ASM (coming soon and available to members), and the membership promotion package I alluded to in my last column is also available for download. Try using it in a presentation to your hospital or GP division.

While we hope you can now use the site to its best potential, it has to be said that the development did not go completely without incident! In particular, the secure payment system proved to be a stumbling block, as did getting a smooth link to the Berjaya Resort for bookings. In retrospect, it was asking too much of one individual to manage both the new web site and the ASM in the same year, but Glen has battled away at it and has now beaten the problems into submission. The Executive is grateful for his efforts and hopes he has some time off for good behaviour once the meeting is concluded. We hope to see many of you there.

The third great project is the cementing of our relationship with the EUBS. An agreement to continue joint ownership of this journal is accepted in principle (and enthusiastically) by both societies, and the present Editor, Mike Davis, EUBS and SPUMS have signed a contract for him to continue in this role for the next three years. He continues to produce a quality publication of which members of both societies should be proud. We are all waiting anxiously for word from the National Library of Medicine about the indexing of DHM on Medline to add to that on EMBASE and ISI.

Finally, I hope some of you enjoy this issue's CME package. The previous package seemed of use to some of you and we will continue this feature for the time being. No doubt the Editor will let me know if there are better uses for the space!

I look forward to seeing many of you in Redang. If not there, then perhaps next year, when rumour has it we will be returning to one of our favourite destinations. Get to the ASM this year to find out where…

Michael Bennett

Short communications

Variations in exhaled nitric oxide concentration after three types of dive

Pieter-Jan van Ooij, Antoinette Houtkooper and Rob van Hulst

Key words
Diving, hyperbaric, hyperoxia, expired nitric oxide, F_ENO

Abstract
(van Ooij P-J, Houtkooper A, van Hulst R. Variations in exhaled nitric oxide concentration after three types of dive. *Diving and Hyperbaric Medicine*. 2010;40(1):4-7.)
Introduction: An increase in exhaled nitric oxide concentration (F_ENO) occurs during an exacerbation of chronic obstructive lung disease or other inflammatory processes of the airway. Raised F_ENO levels are also observed during normobaric, mild hyperoxic exposures, whereas after hyperbaric hyperoxic exposure the F_ENO level is reduced. This study investigated the variations of F_ENO after three different types of dive.
Methods: Military divers participated in either a closed circuit rebreather dive (CCR, n = 17, pO_2 = 130 kPa), semi-closed circuit rebreather dive (S-CCR, n = 12, pO_2 = 180 kPa) or a compressed air dive (scuba, n = 17, pO_2 = 126 or attendant, n = 12, pO_2 = 118 kPa). Before and after each dive, the F_ENO was measured using a hand-held electrochemical analyser (Niox Mino®).
Results: All values for F_ENO fell within the normal range (5–25 ppb). A small decrease in F_ENO level was found after all dives. After CCR dives, F_ENO fell from 16.4 (± 8.0) pre-dive to 13.6 (± 7.5) ppb, after S-CCR from 16.2 (± 7.2) to 13.6 (± 6.3) ppb, scuba from 17.1 (± 5.6) to 16.1 (± 5.2) ppb and attendants from 17.7 (± 9.8) to 17.3 (± 9.1) ppb. Only after a CCR or S-CCR dive was this decrease statistically significant ($P < 0.05$).
Conclusion: In our divers, hyperbaric hyperoxia up to 180 kPa led to a small decrease in F_ENO in the conductive compartment of the lungs, the biological importance of which is unknown.

Introduction

Nitric oxide (NO) is a small molecule with the qualities of a free radical. In the human body, NO is made under the influence of NO-synthase (NOS) from the oxidation of L-arginine by NOS to L-citrulline with the release of NO.[1] Three forms of NOS are described: neuronal NOS (nNOS), endothelial NOS (eNOS) and inducible NOS (iNOS); nNOS and eNOS are often termed constitutional NOS (cNOS).[1,2] While cNOS is constantly available and produces low quantities of NO, iNOS will produce NO in large quantities under more extreme circumstances, such as an inflammatory process.[2] In the lung, cNOS is found in the epithelium, endothelium and neurons, while iNOS is found in the epithelium and macrophages. Despite its radical qualities, the half-life of NO in air can be tens of seconds, which allows us to measure NO in the exhaled breath.[1]

NO, in reaction with oxygen, can be metabolized to nitrite (NO_2^-), nitrate (NO_3^-), and peroxynitrite ($ONOO^-$). Peroxynitrite has cell- and tissue-damaging activity which causes inflammation. This kind of inflammation plays an important role in the exacerbation of asthma or chronic obstructive pulmonary disease (COPD).[1] In 1899, Lorrain-Smith demonstrated that breathing oxygen at a partial pressure (pO_2) higher than 50 kPa can cause pulmonary damage, leading to pulmonary oedema and airway inflammation.[3] In view of this inflammation, one would expect an increase in the exhaled nitric oxide concentration (F_ENO) after exposure to a breathing gas with a pO_2 of more than 50 kPa. However, earlier studies showed inconsistent results. Raised F_ENO levels were observed during normobaric, mild hyperoxic exposures,[4,5] whereas after hyperbaric hyperoxic exposure the F_ENO level was reduced.[2,6] The aim of this study was to investigate the effect on F_ENO of three different types of dive, each using a different breathing gas with a pO_2 of more than 100 kPa.

Methods

STUDY POPULATION

All participating divers and attendants (n = 58) were professional military divers and fit to dive. All dives used to measure F_ENO were made as part of their daily routine or as part of their training. Each diver or attendant participated in only one type of dive. All participants were male and were informed about the aims of the study during a general meeting. It was explained that they could withdraw from the study at any time and the F_ENO results would not be put in their personal medical file. Before F_ENO measurement, they were asked again if they had any questions or objections regarding the study. Table 1 presents demographic data on the divers and attendants.

Table 1
Demographic data, mean (SD) for the participating divers and attendants (*n* = 58)

	CCR divers (*n* = 17)	S-CCR divers (*n* = 12)	Scuba divers (*n* = 17)	Attendants (*n* = 12)
Height (cm)	181.4 (6.2)	184.8 (5.4)	183.8 (5.9)	181.5 (7.9)
Weight (kg)	85.6 (8.7)	91.7 (10.8)	89.5 (11.6)	88.3 (13.5)
Age (years)	24.5 (2.6)*	34.3 (8.5)*	40.2 (6.5)†	43.3 (4.8)†
Smoking (pack-years)	0	0	0.9 (2.6)	0

* − $P < 0.05$ between all groups; † − $P < 0.05$ between all groups except between scuba divers and attendants

THE DIVES

Closed circuit rebreather (CCR) dive: this was a wet dive where the divers (*n* = 17) used a CCR (LAR VII, Draeger®) and breathed 100% oxygen. Maximal pressure at depth was 130 kPa (3 msw, pO_2 approaching 130 kPa) and the dive time was 60 min.

Semi-closed circuit rebreather (S-CCR) dive: this was a wet chamber dive where the divers (*n* = 12) used a S-CCR (SIVA 55, Carleton®) and breathed 60% oxygen and 40% nitrogen. Maximal pressure at depth was 300 kPa (20 msw, pO_2 approaching 180 kPa) and bottom time was 47 min. Decompression was done according to the Canadian diving tables (DCIEM Table 1: 21 msw/50 min). Total dive time was 58 min.

Scuba dive: this was a partial wet, partial dry dive where the divers (*n* = 17) used scuba (Mk 25/S550, Scubapro®) while breathing compressed air. Maximal pressure at depth was 600 kPa (50 msw, pO_2 approaching 126 kPa) with a bottom time of 14 min. Diving was done in the wet compartment of our pressure chamber; for both S-CCR and scuba dives, the water temperature in the wet chamber was 13–15°C. Decompression according to an adapted DCIEM Table 1 (51 msw/25 min) was done in the dry compartment where the divers breathed chamber air. The total dive time was 71 min. During this study, we also measured F_ENO of the attendants (*n* = 12) who stayed inside the dry compartment during this whole dive. Their maximal pressure at depth was 560 kPa (46 msw, pO_2 approaching 118 kPa).

MEASUREMENT OF F_ENO

Before and directly after every dive, the F_ENO was measured using an electrochemical hand-held NO analyser (Niox Mino®, Aerocrine AB, Sweden). Compared to on-line NO analysers, the measured F_ENO values using the Niox Mino® are statistically the same.[7] All measurements were done according to the ATS/ERS guidelines with an expiratory flow rate of 50 ± 5 $ml \cdot s^{-1}$.[8] The divers and attendants were not allowed to drink coffee, eat or smoke within 1 h before any measurement.

STATISTICAL ANALYSES

The results are presented as mean and standard deviation (SD). Regarding the F_ENO data, the Shapiro-Wilk (S-W) test (STATA Manual Reference G-M; 2003. p. 231) showed a non-normal distribution for one of the groups (chamber attendants). Therefore, we log transformed the F_ENO data after which the S-W test showed a normal distribution for all groups. Differences between the log transformed pre- and post-dive F_ENO values were analysed using the paired Student's t-test. A *P*-value < 0.05 was considered statistically significant. Analyses were performed using Stata SE software (StataCorp, version 9.2).

RESULTS

All F_ENO measurements fell within the normal range (5–25 ppb).We found no differences between the three dive groups regarding height, weight and smoking history (i.e., pack-years). There was a significant difference in age between the groups, except between scuba divers and attendants. The CCR divers were the youngest divers and the attendants the oldest (Table 1).

All dives produced a small decrease in F_ENO, with the greatest decrease after a CCR dive (-2.8 ppb) and the smallest

Table 2
F_ENO (ppb) pre- and post-diving, mean (SD) shown;
CCR – closed circuit rebreather; S-CCR – semi-closed circuit rebreather; * *P* < 0.01

	CCR divers (*n* = 17)		S-CCR divers (*n* = 12)		Scuba divers (*n* = 17)		Attendants (*n* = 12)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
F_ENO	16.4 (8.0)	13.6(7.5)*	16.2 (7.2)	13.6(6.3)*	17.1 (5.6)	16.1 (5.2)	17.7 (9.8)	17.3 (9.1)

in the attendants (-0.4 ppb). Only after a CCR or S-CCR dive did this decrease in F_ENO become statistically significant at the $P < 0.05$ level (Table 2).

Discussion

The results of the CCR and S-CCR groups, with a small decrease in F_ENO, are in line with results from earlier studies.[2,6] As the CCR and S-CCR divers were the only ones who performed a totally wet dive, submersion could play a role in this reduction. During submersion there is a central pooling of blood within the thoracic cavity. This thoracic pooling results in an increased pulmonary blood flow which leads to an increase in pulmonary artery pressure.[9] It is known that pulmonary arterial hypertension results in decreased F_ENO, so it is conceivable that increased pulmonary artery pressure could cause a drop in F_ENO.[10] Secondly thoracic pooling leads to an improved ventilation-perfusion relationship.[9] This improvement could result in a higher level of NO diffusion and, therefore, to a lower net F_ENO. Eventually, both mechanisms could cause a more pronounced decrease in F_ENO in a wet dive compared to a dry hyperbaric chamber dive.

However, compared to earlier studies, in which only dry chamber dives were performed, our findings showed a smaller decrease in F_ENO.[2,6] Therefore, submersion alone cannot fully explain the decrease in F_ENO we found. More plausible explanations for this decrease were given by Puthucheary et al who stated that hyperbaric oxygen inhibits iNOS which leads to a decrease in F_ENO.[2] Also, the presence of reactive oxygen species could scavenge NO, resulting in decreased F_ENO.[2]

As F_ENO is a biological marker, normal deviations play a role and must be taken into account. A difference of up to 2 ppb between two measurements of F_ENO can be found, so a difference of more than 4 ppb is considered to be of biological significance.[11] Regarding the Niox Mino®, which has an accuracy of +/- 2.5 ppb, a difference of more than 5 ppb is regarded as biologically significant.[7] In view of this, we conclude that, although we found a statistically significant reduction of F_ENO in the CCR and S-CCR groups, these minor changes are not bio-medically relevant.

Finally a limitation of the present study should be mentioned. We measured the F_ENO with an expiratory flow rate of 50 ± 5 $ml \cdot sec^{-1}$, according to the ATS/ERS guidelines.[8] At these flow rates, one measures the F_ENO from the bronchus down to the alveolus, but not the alveolus itself.[12] To measure the alveolar compartment F_ENO, the subject should exhale in a controlled fashion over 8–10 sec at a flow rate of at least 250 $ml \cdot sec^{-1}$, and sidestream sampling (of alveolar gases) occurs during the final part of exhalation.[10,13] As we used a flow rate of 50 $ml \cdot sec^{-1}$, we measured changes in the conductive compartment of the lungs only and not in the alveolar compartment (see Appendix). Earlier studies used flow rates of up to 100 $ml \cdot sec^{-1}$, implying that they also only measured changes of F_ENO in the conductive compartment.[2,4–6] To differentiate between the alveolar and conductive compartments, the multiple exhalation flow technique (MEFT) should be used.[14] Since hyperbaric hyperoxia produces alveolar damage, one should use expiratory flow rates of at least 250 $ml \cdot sec^{-1}$, and we strongly recommend that future studies use the MEFT to differentiate between F_ENO changes in these two compartments after exposure to an increased level of pO_2.

Appendix

The F_ENO pathway is often visualized as a two-compartment model with a conductive (airway to generation 17) and alveolar compartment (generation 18 to alveolus). F_ENO is a net result of flux and diffusion of NO in these compartments.[13] Based on this idea it is possible to calculate the F_ENO using the formula of George et al.[13]

$$F_ENO = C_{aw,no} + (C_{alv,no} - C_{aw,no}) * \exp(-D_{aw,no}/V)$$

where $C_{aw,no}$ is the airway wall concentration of NO, $C_{alv,no}$ the steady-state alveolar concentration of NO, $D_{aw,no}$ diffusing capacity of NO, and V is the exhalation flow rate.

- Healthy persons have a F_ENO between 5 and 25 ppb.[7,11,12]
- Values above 50 ppb can be found in exacerbation of asthma and chronic obstructive pulmonary disease or an acute eosinophilic airway inflammation.[12]
- Values below 5 ppb can be found in smokers or after strenuous efforts.[8]

F_ENO is not influenced by age, day-to-day or within-day variations but is reduced in females.[8,11]

Acknowledgements

We would like to thank our participating military divers and Willard van Ooij, MSc, for his additional statistical advice.

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A subjective evaluation of a drinking system for saturation divers

Arvid Hope and Rudolf Brekken

Key words
Saturation diving, fluid balance, thermal problems (hypothermia and hyperthermia)

Abstract

(Hope A, Brekken R. A subjective evaluation of a drinking system for saturation divers. *Diving and Hyperbaric Medicine.* 2010;40(1):8-10.)
Studies have shown that divers may lose large volumes of body fluids in hot-water-suit (HWS) dives lasting for four hours or longer, and that this dehydration is mainly caused by sweating. Body fluid balance may be impaired and the diver's alertness and power of judgement could be influenced by such imbalance. The main objective of the present study was to obtain a subjective judgement of a drinking system for divers (DSFD) and to obtain information related to body fluid loss during long saturation lock-out dives. Via a suction pipe imbedded in the microphone unit in the oronasal mask, the DSFD makes it possible for the diver to drink while in the water. Ten divers tested the drinking system during 12 saturation lock-out dives lasting an average of 5.5 h. A questionnaire was answered after each dive. The divers drank 21 times (range 5–30 times) during the dives, and the average drinking volume was 1.4 litre (range 1.0–1.5 litre), but drank only 0.04 litre (range 0–0.3 litre) in the bell after diving. The system was easy to operate and preparation and clothing did not cause any delay. The suction pipe did not intrude and the microphone performed excellently. The work in water was not hindered by DSFD and all divers were very satisfied with the drinking system. It was obvious that the need for fluid intake after a dive with DSFD was markedly reduced – another good indication of maintained body fluid balance.

Introduction

Occupational diving is an activity that may influence body fluid balance and induce non-homeostatic conditions. With the open hot-water-suit (HWS) technique, cooling of the diver is avoided by the surface-heated seawater delivered via an umbilical to perforated hoses sewn into the suit material. Warm seawater with a temperature of about 38°C will thereby continuously flood the skin's surface. The hot water enters the suit through an on/off valve at the diver's waist making it possible for the diver to control the supply. However, it is a fact that most (or all) divers regulate the hot water flow in order to be warm and comfortable rather than thermoneutral or cold (personal communications). Since the diver's skin is at 100% humidity the normal evaporative heat loss does not occur and hyperthermia may develop. If the diver loses body fluids equivalent to more than 3% of his body weight, both his physical and mental performance may be impaired by the end of the dive.[1,2] Thus, if a critical situation should occur at the end of a dive, a dehydrated and hyperthermic diver might not react adequately and thereby might endanger his safety.

The effect of water immersion and weightlessness on the circulatory system and urine production (immersion diuresis) is well established.[3] In addition to this fluid loss, we have previously shown that divers may lose large volumes of body fluids in HWS dives lasting for four hours or longer, and that this dehydration is mainly caused by sweating.[4,5] In support for this view, body core temperature increased by 0.6°C, from a predive value of 37.4°C, during operational saturation diving.[6] Also, fluid intakes of more than 1.5–2 litres have been reported shortly after diving (information from a diving company). It should be emphasised that during a normal working dive lasting for 4–6 hours the diver does not drink any fluid. To maintain body fluid balance, a drinking system for divers (DSFD) has been developed.[7] The DSFD may, therefore, add to both comfort and safety during long-lasting HWS dives.

The main objective of the present study was to obtain a subjective assessment of DSFD and to obtain information related to body fluid loss during long-lasting, saturation lock-out dives.

Methods

A bag containing the drinking fluid is located on the diver's back. The fluid flows via a supply hose, a manually operated valve penetrator on the side of the helmet, and a suction pipe with a mouthpiece imbedded in the microphone unit in the oronasal mask (Figures 1 and 2). When the diver requires a drink he opens the valve and sucks on the mouthpiece.

Ten divers tested the drinking system during 12 saturation lock-out dives with an average lock-out duration of 5.5 hours (range 3–6 h 50 min). A questionnaire (Table 1) was answered after each dive.

Results

The detailed results from the subjective evaluation are presented in Table 1 (one diver did not answer questions 3 and 8, and two divers did not answer questions 12 and 13).

Figure 1
The drinking system dismantled into its constituent parts: a fluid bag (A), supply hose (B), valve (C) and microphone with cable and suction pipe (D)

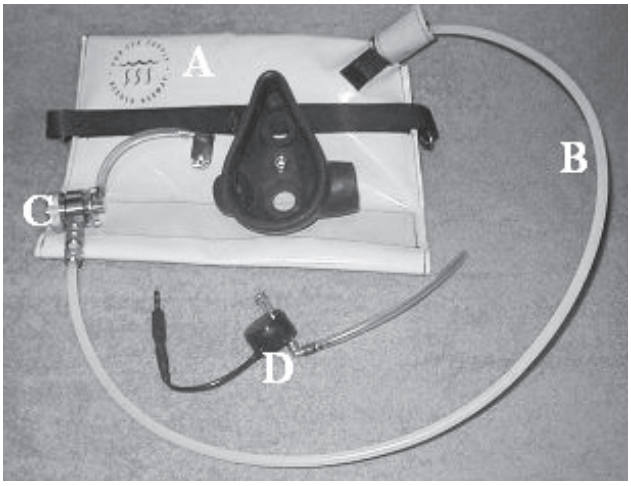

Figure 2
A diving helmet showing the supply hose (B) and the valve (C) in position

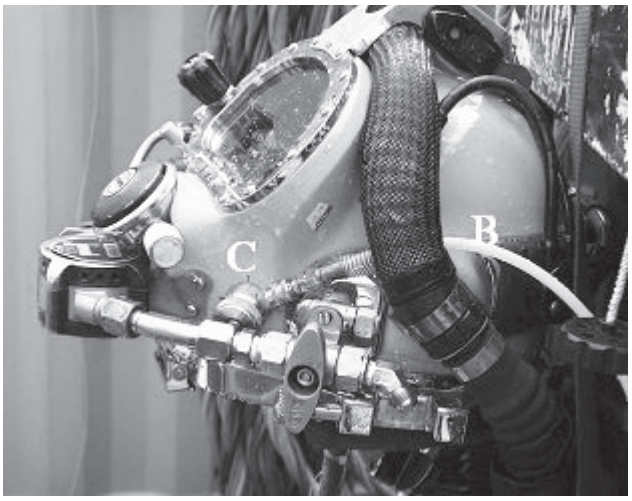

Table 1
Subjective evaluation of a drinking system for divers from 12 trials with ten divers; average lock-out duration was 5.5 hours

Questions	Responses
Physiological aspects	
1. What did you drink?	9 dives: Juice and water (50/50) 2 dives: Water 1 dive: Orange juice
2. How often did you drink during the dive?	Average: 21 times (range 5–30)
3. Did you drink every time you felt the urge?	Yes: 11 No: 0
4. How much did you drink in the bell after the dive?	Average: 0.04 L (range 0–0.3)
5. Would you consider using the drinking system if offered on subsequent occasions?	Yes: 12 No: 0
6. On a scale from 1 to 5, how would you characterise the use of DSFD? (1 = unuseable, 5 = very useful)	Average: 4.5 (range 4–5)
7. Do you believe the need for a break during a long lock-out would diminish with DSFD?	Yes: 10 No: 2
Technical aspects	
8. Was the dressing delayed due to the DSFD?	Yes: 1 No: 10
9. If yes, indicate approximately the time delay	5 min
10. Was it difficult to operate the DSFD valve?	Yes: 0 No: 12
11. Did the mouthpiece cause any problems with:	
Communication?	Yes: 0 No: 12
Breathing?	Yes: 0 No: 12
Comfort?	Yes: 0 No: 12
12. Are you satisfied with the shape of the microphone?	Yes: 10 No: 0
13. Are you satisfied with the position of the microphone hole in the oral nasal mask?	Yes: 10 No: 0
To be answered by the Diving Supervisor (n = 5)	
14. How would you characterise the sound quality of the microphone compared to other microphones?	Excellent: 1 Good: 4

On average, the divers drank 21 times (range 5–30 times) during a dive. The drinking volume using the DSFD was 1.4 L (range 1.0–1.5 L). Fluid intake in the bell after the dive was only 0.04 L (range 0–0.3 L) when using the DSFD.

Discussion

The drinking system was easy to operate and preparation and clothing did not cause any delay. The suction pipe did not intrude and the microphone performed well (Table 1). Thus, the work in water was not hindered by DSFD. All divers were very satisfied with the drinking system and indicated that they would use the system if available in future diving operations. By using the DSFD it was obvious that the need for fluid intake after a dive was markedly reduced – a good indication of maintained body fluid balance. Under normal operational conditions, divers drink about 1–1.5 L in the bell after finishing the dive. Possible negative effects of dehydration on the diver's physical performance, alertness and power of judgment will thereby be avoided.[1,2]

It is also well established that body temperature regulation is impaired by dehydration.[8] The observed core temperature increase in saturation divers may partly be caused by the sweat fluid loss and dehydration previously described.[4–6] Furthermore, dehydration may increase the risk for decompression sickness (DCS). In a review of 68 recreational divers with spinal cord decompression sickness, risk factors were fatigue, circumstances suggesting dehydration, and extreme physical effort.[9] A study on pigs dived on similar tables showed that dehydration significantly increased the risk of severe decompression sickness.[10] These findings are relevant to saturation diving and the in-water decompression during excursions. The importance of maintained fluid balance is further indicated by the recommendation of the International Marine Contractors Association that a diver spending over two hours out of a closed bell should be offered the opportunity to return to the bell for a drink or other refreshments.[11] Thus, we maintain that the DSFD described here will add to the safety of divers during long HWS dives.

Acknowledgements

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Review articles

Predicting performance in competitive apnea diving. Part II: dynamic apnea

Erika Schagatay

Key words
Breath-hold diving, hypoxia, exercise, cardiovascular, respiratory, physiology, safety, review article

Abstract

(Schagatay E. Predicting performance in competitive apnea diving. Part II: dynamic apnea. *Diving and Hyperbaric Medicine*. 2010;40(1):11-22.)
Part I of this series of articles identified the main physiological factors defining the limits of static apnea, while this paper reviews the factors involved when physical work is added in the dynamic distance disciplines, performed in shallow water in a swimming pool. Little scientific work has been done concerning the prerequisites and limitations of swimming with or without fins whilst breath-holding to extreme limits. Apneic duration influences all competitive apnea disciplines, and can be prolonged by any means that increase gas storage or tolerance to asphyxia, or reduce metabolic rate, as reviewed in the first article. For horizontal underwater distance swimming, the main challenge is to restrict metabolism despite the work, and to direct blood flow only to areas where demand is greatest, to allow sustained function. Here, work economy, local tissue energy and oxygen stores and the anaerobic capacity of the muscles are key components. Improvements in swimming techniques and, especially in swimming with fins, equipment have already contributed to enhanced performance and may do so further. High lactate levels observed after dynamic competition dives suggest a high anaerobic component, and muscle hypoxia could ultimately limit muscle work and swimming distance. However, the frequency of syncope, especially in swimming without fins, suggests that cerebral oxygenation may often be compromised before this occurs. In these pool disciplines, safety is high and the dive can be interrupted by the competitor or safety diver within seconds. The safety routines in place during pool competitions are described.

Introduction

This series of articles deals exclusively with competitive apnea diving. This is fundamentally different from the repeated foraging and hunting diving activities undertaken by the Ama of Japan, or in spearfishing and team sports activities involving high levels of exertion during short dives, e.g., underwater hockey or rugby.[1–3] In competitive diving the aim is to perform one dive of maximal duration, distance or depth, whilst avoiding hypoxic syncope. The factors determining the limits of apneic duration at rest (static apnea, STA), a major prerequisite for performance in all competitive apnea disciplines, were summarised as:

- total body gas storage capacity in lungs, blood and tissues;
- tolerance to asphyxia;
- metabolic rate.[4]

Distance swimming, where physical work is added to the stressors, and diving to depth, where pressure effects are superimposed, each impose new, potentially limiting factors. The focus in this second article will be on the limiting factors of working apneas for maximal distance swimming in shallow water in dynamic apnea with (DYN) and without (DNF) fins. Few scientific studies directly concerning these disciplines have been done, and no previous review in this field exists. The main factors determining performance in the dynamic disciplines will be presented, but calculation of the relative influence of these factors is not possible given our present knowledge. A model of the relationships between several of the factors involving calculations of gas exchange during deep working dives has been published,[5] yet several conditions differ in horizontal diving.

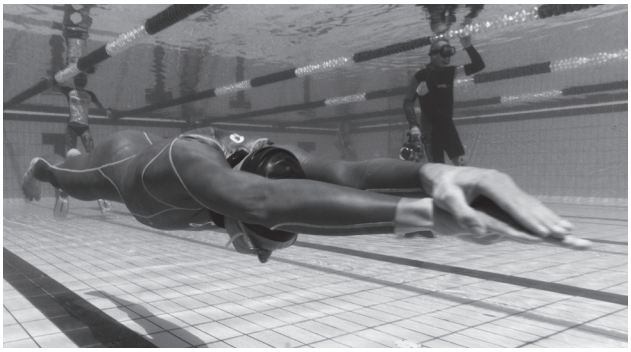
In the past two decades, performance in competitive apnea diving has shown a surprising and escalating development: of 12 male and female world records in the six main competition disciplines in January 2009, only three remain unchanged one year later. A central issue is: can the safety measures taken during training and competition keep pace when records approach the human limits in duration, distance and depth? The thorough risk-management safety systems developed amongst elite divers and during competitions will be described here. The risks and pathological effects of apneic diving have been well reviewed recently.[6,7] It is important to emphasize at the outset that record attempts and training for apnea sports should never be done without proper knowledge of the risks involved and how to prevent them. Without doubt, the beginner apneist is at higher risk than the elite free diver.

Working without breathing – dynamic apnea

Competitive dynamic apnea is performed in a pool, the aim being to cover the longest possible distance, without any time limitation. Distance swimming is initiated from a resting state and physical work starts simultaneously with

Figure 1
Record-holder Natalia Molchanova during DYN at the World Championships 2008

Figure 2
Record-holder William Trubridge during DNF at the World Championships 2008



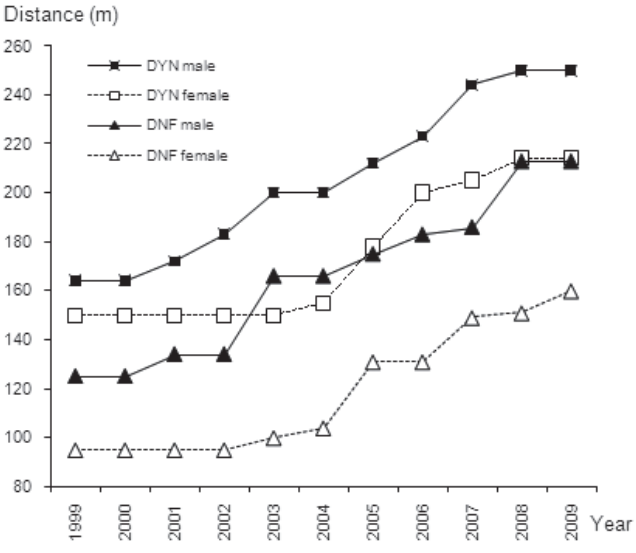
the dive. The two dynamic apnea disciplines, DYN and DNF differ in the presence of fins for propulsion (Figures 1 and 2). The physiological consequence is that in DYN, which is performed on the elite level almost exclusively with a monofin, the work is restricted to the legs, pelvis, lower abdomen and back, while in DNF the propulsion resembles breast stroke swimming, i.e., the whole body is at work, but with little dorsoventral flexion of the back compared to the dolphin-style kick used with a monofin. While there is one glide phase in DYN, there are two in DNF, one after each of the arm and leg strokes, which are performed separately. The record distances tell us something about the differences in energy requirements: the current world record in DYN is 250 m for men and 214 m for women, while in DNF the records are 213 m for males and 160 m for females (Figure 3). Thus, distances are 15–25% shorter in DNF than in DYN, whilst the dive times are similar, reflecting the greater energy cost when swimming without fins.

Anthropometrics may have a greater impact on results in DNF. Just as in swimming, there are benefits to having big hands and feet and a long arm reach.[8,9] The smaller difference between DNF and DYN records among males could be because of their relatively larger hands and feet, and more powerful upper-body pull phase than females. Technical skill and work economy may also be more important in DNF, where energy wasted is greater due to thrust being smaller. As swimming propulsion is required over the entire horizontal distance attempted, except that obtained from the push-off from the pool sides involving explosive work, DNF is likely the energetically most demanding of all the apnea disciplines, with the 'constant weight, no fins' (CNF) depth discipline, involving passive sinking during the free-fall phase, second. The two major differences from deep diving are that in dynamic apnea work for propulsion is required during the entire dive, and no initial period of hyperoxia precedes the development of hypoxia. Apnea exceeding ten minutes at rest in STA may be impressive; yet swimming 250 m underwater on one breath indicates that the maximal human aerobic dive limit and anaerobic work capacity may exceed that formerly considered possible.

Blood flow distribution: priority of regions versus overall shortage

Local hypoxia occurs in many sports and distribution of blood flow is the main problem, but in dynamic apnea, the challenge is to sustain propulsive work during progressive systemic hypoxia. During resting apneas, the diving response reduces heart rate and cardiac output, and directs the blood flow mainly towards the brain and heart, while the rest of the organism receives a more limited blood flow.[4,10] The response is initiated at apnea onset and is thereby 'hypoxia preventive'.[11] It is enhanced by facial chilling and has priority over other homeostatic responses, e.g., related to body chilling and immersion.[12,13] The response develops and conserves oxygen even if the apnea is preceded by work,[14–16] or starts simultaneously with work.[17] It has been suggested that during swimming dives, working muscles will also receive part of the blood flow.[18] In experiments simulating DYN, with apnea and work initiated at the same time, heart

Figure 3
Record development for DYN and DNF during the past decade



rate falls to a level intermediate between that of rest and of eupneic work, and apneic oxygen consumption ($\dot{V}O_2$) was reduced by 25% during exercise, and by 40% during resting apneas.[19] The diving response will thus favor the central circulation and to some extent working muscle. However, when blood oxygen content is depleted, the main problem is the tolerance of the brain to hypoxia. While brain hypoxia will eventually lead to syncope, muscle asphyxia may lead to lost muscle force and loss of power in propulsion. The central question is which will occur first.

There are at least two mechanisms that prioritise brain oxygenation during immersed apnea: centralization of blood flow by peripheral vasoconstriction via the diving response, and CO_2-induced cerebral vasodilation.[10,20,21] During non-immersed, maximal resting apnea, skeletal muscle oxygenation decreased earlier than cerebral oxygenation.[22] A study involving short resting and working apneas, showed that brain blood flow increased in both situations.[23] In the face of diminishing blood oxygen supply to working muscle, local muscle oxygen stores as well as hypoxia tolerance must be maximized in the dynamic apnea disciplines.

The efficiency of the diving response to conserve oxygen depends on both the rate of response development and the extent of reduction in cardiac output. It has been suggested that the onset of the response is affected by hypoxia and work, but not its final level of adjustment.[24] However, at least in trained divers, bradycardia is enhanced toward the end of the apnea.[25,26] Some divers claim that by starting a dynamic apnea with a passive apneic phase, the diving response will be allowed to restrict peripheral blood flow more efficiently than if work starts immediately (personal communications from divers, 2008). This has not been validated yet and, although this is not a technique used in competition today, any such changes could enhance performance as long as peripheral muscle hypoxia is not the main limiting factor.

Myoglobin

Myoglobin is a heme protein involved in both oxygen transport and storage within the muscle cell. With enhanced local oxygen stores in the form of increased myoglobin, the environment in working muscles would be greatly improved in the diver, leaving elevated circulating oxygen levels for the brain and heart. High myoglobin concentration is regarded as an important adaptation to apneic diving in mammals, extending the time the diver can rely mainly on stored oxygen to sustain aerobic metabolism.[27,28] In marine mammals, a difference in myoglobin concentration between swimming and other muscles develops in association with their start in diving activity, suggesting that this could be a result of training.[29] More myoglobin is present in the swimming muscles of species that perform long, deep, dives than those that make shorter, shallow dives.[28] Myoglobin has a much higher affinity for oxygen than haemoglobin, and for the stored oxygen in myoglobin to become available for aerobic metabolism, the partial pressure of oxygen has to be low. If the diving response shuts off circulation at an early stage, the switch to myoglobin-derived oxygen would presumably occur earlier.[30] Swimming muscle mitochondrial density was found to be increased but capillary density much lower in seals compared to in dogs, suggesting that oxygen is mainly derived from within the muscle cells.[30]

In humans, myoglobin may be mainly an oxygen diffusion facilitator.[31] High-altitude dwellers have a higher muscle myoglobin concentration than lowlanders,[32] but data on the effect of endurance training during hypoxia on myoglobin concentration are conflicting.[33,34] Short-term altitude exposure (7–9 days at 4,500 m) leads to hypoxia-induced erythropoiesis but, at the same time, a down-regulation of myoglobin may occur in order to release iron for enhanced erythropoiesis.[35] This could be different during long-term exposure to altitude hypoxia, when iron availability is not limiting. Apnea training has been shown to elevate erythropoietin levels, which could possibly conflict in the short term with myoglobin elevation.[36] Thus, the situation in human divers remains unclear, as neither the presence of efficient vasoconstriction in working muscle during diving, nor their levels of myoglobin have been established. As will be seen in the next section, post-dive lactate levels are increased in DYN and DNF, indicating the contribution of anaerobic metabolism. Lactate accumulation will decrease the affinity of myoglobin for O_2, thus facilitating diffusion of O_2 to mitochondria for sustained oxidative phosphorylation during apnea.[37] Thus, with lactate development in dynamic disciplines, increased oxygen supplies may be made available, leading to prolongation of aerobic metabolism in parallel with the anaerobic one.

Anaerobic metabolism and performance

Ama divers at Hegura, Japan, sustain repeated diving of limited depth and duration for several uninterrupted hours, with surface intervals of similar duration to the dives, spending nearly half of their working time underwater (Schagatay E and Lodin-Sundström A, unpublished observations, 2009). This suggests that these dives are within aerobic dive limits. In Korean Ama, pH was observed to decrease only slightly.[38] On the other hand, elite apnea divers may require both enhanced aerobic and anaerobic capacity to perform maximal competition dives. Aside from a small contribution from energy-rich phosphates, the production of lactate from muscle glycogen is the most important anaerobic process for energy production. Lactate increases after both resting apneas and apneas involving exercise, showing that a net production occurs during apnea.[11,16,39,40] During eupneic work, part of the lactate produced in working muscles is catabolised by the less active muscles or used during recovery to resynthetize glycogen.[41] However, during apneic diving, lactate removal from working muscles may be a significant problem due to selective vasoconstriction, and restricted blood flow may lead to considerable regional differences in lactate concentration.

To our knowledge there are no published studies on lactate levels after competition DNF and CNF dives. Preliminary data have been reported from experimental pool dives, showing in STA an elevation from 1.2 to 2.2 mmol·L^{-1}, and from 1.8 to 6.8 mmol·L^{-1} in DYN, but information concerning the exact protocol, sampling site and times was lacking.[42] We recently measured capillary lactate two to four minutes after competition dives during a world championship. In the same eight subjects, DYN and DNF resulted in similar levels of approximately 10 mmol·L^{-1} (normal resting range: 1–2 mmol·L^{-1}; Schagatay E and Lodin-Sundström A, unpublished observations, 2009; Figure 4). The mean distances covered were 173 m in DYN and 135 m in DNF ($P < 0.001$), suggesting that the workload was significantly reduced by using fins. Capillary blood lactate levels were also elevated to over 5 mmol·L^{-1} in two competition STA dives exceeding eight minutes. This demonstrates that the diving response effectively shuts down some areas from circulation, causing anaerobic metabolism even under resting conditions. These lactate levels are substantially higher than previously reported after both resting and working experimental apneas, and clearly show the importance of anaerobic pathways during maximal competition apneas. Interestingly the levels after dynamic apneas were in the same range as values seen after 200-yard, maximal-effort freestyle swims, despite only comparatively moderate levels of physical exertion in the apneic swims.[43]

Hypoxia impairs endurance performance by causing premature muscle fatigue,[44] which could limit performance in dynamic apnea. The causes of muscle fatigue are complex, and only partially understood, and may be central or peripheral in origin.[44–48] The traditional view, that acidification will be limiting to performance by disrupting the contractile processes,[49] has been challenged by more recent research, suggesting several possible mechanisms such as ATP depletion that results in intracellular accumulation of potassium, inorganic phosphate accumulation when creatine phosphate is hydrolysed, and negative effects on calcium availability and its binding to troponin.[45,50] Acidosis-induced discomfort may, in itself, limit performance even before muscle functions are inhibited.[45] However, cerebral hypoxia may be the more acute problem in DNF, evidenced by the observation that one out of six of the DNF dives at a recent world championship resulted in syncope, while other disciplines had only a few cases (Schagatay E and Lodin-Sundström A, unpublished observations, 2009).

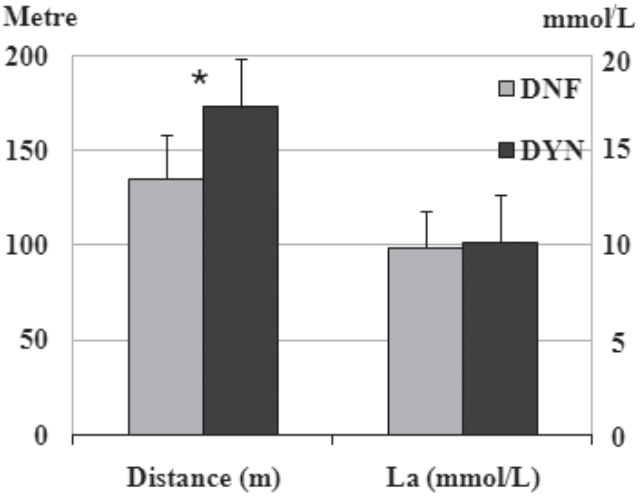
The high lactate levels recorded after competition dives in elite apneists in our studies seem to contrast with the findings of a fall in post-apneic lactate after long-term apnea training in triathletes.[51] The authors suggested that this reduction, which could be a result of either reduced cellular production or increased catabolism, or both, is an adaptive response to hypoxic work. The increase in lactate in that study was low compared to our observations,[51] suggesting that apneas were far from maximal. The lowered lactate levels seen in such voluntary non-competition dives after training may indicate that maximal dives could be significantly extended before performance-limiting hypoxia develops.

During competitive dives, accumulation of factors leading to fatigue cannot be allowed to reach levels intolerable to the working muscles, or the dive will be terminated due to loss of propulsion. Directly after an in-competition dynamic apnea, the athlete is occasionally unable to walk and may feel the effects of the dive in the legs for some time, being unable to perform another productive competition dive in the same discipline for hours or even days. During the first minutes after a maximal competitive dive, there is also an oxygen debt that must be paid back, and blood and tissue CO_2 accumulation, which has to be released.[52] However, normal arterial oxygen saturation was recorded within two minutes even after dynamic competition dives leading to syncope (Schagatay E, unpublished observations, 2009). Apneic swimming may be a good model for the study of the mechanisms behind hypoxic limitation of work in the presence of hypercapnia as compared to hypocapnic or eupneic hypoxia.

Preparations before diving

Preparations for sports competition are often as important to the outcome as events taking place during the actual competitive performance. This section deals with some preparatory methods explored by divers to enhance performance. Different strategies may be used and there is little consensus among divers as to what works best. While the general rule a few years ago was to perform a warm-up protocol including dives, as well as long periods of various breathing techniques, more recently no-warm-up and no-warm-up/no-breathe-up approaches have been adopted by some top divers; the physiological background to these approaches is largely unknown.

Figure 4
Mean (SD) distance covered (m) and blood lactate concentration (mmol·L^{-1}) after DNF and DYN competition dives in 8 divers; * $P < 0.001$



BREATHE UP, OR NOT?

Just as in STA, specialized breathing techniques are often used before dynamic disciplines to enhance performance.[4] The effect of 'yoga breathing' is to lower the large, 'slow' tissue stores of CO_2 and maximize O_2 storage, allowing longer apneas before asphyxia develops.[52,53] Yoga breathing is also used to improve relaxation and mental focus. Some divers also perform 'classical' hyperventilation just before diving, mainly to lower lung and blood CO_2 levels and delay the onset of involuntary breathing movements.[4,54] Finally, as in STA, lung packing is used to increase the O_2 stores at the start of the dive.[4,55,56] In dynamic apnea, however, lung packing has to be balanced against other performance requirements. Air volume for achieving neutral buoyancy at the optimal swimming depth is pre-set by the placement of neck and hip weights, and a horizontal position across the entire arm- and leg-stroke cycle is essential for reduced drag. Lung packing must also allow for efficient swimming and for turning at each pool end, but hydrostatic pressure effects may allow more packing than in STA. Dry STA and immersed DNF performance were both increased by approximately 12% after packing in trained divers, despite more packing in DNF.[57] An important aspect is to avoid 'packing blackout', caused by brain hypoxia, which may occur when the diver does not submerge quickly or deep enough after packing and the high intrathoracic pressure, by impeding venous return, causes a dramatic fall in blood pressure.[56] Excessive lung packing may even cause temporary asystole, leading to syncope, and air embolism.[58,59]

While nearly all divers use lung packing to some extent, an increasing number of divers use a 'no-breath-up' approach without yoga breathing or hyperventilation, as they believe that the consequent rapid CO_2 accumulation and massive contractions of the diaphragm will lead to improved performance once they can overcome the associated discomfort. The rising pCO_2 does not appear to positively affect the diving response, but other explanations may be that the hypercapnia-induced cerebral vasodilatation enhances brain oxygenation,[20,21] and possibly that the mechanical effects of the involuntary breathing movements enhance brain oxygenation by restoring cardiac output.[60] However, the relative proportion of the 'struggle phase' does not seem to affect the total apneic time achieved, implying that a longer struggle phase does not by necessity lead to prolonged apnea.[60] Differences in individual predisposition may affect the outcome of different strategies, and it appears that the preferred method in one diver may even limit performance in another. Such a lack of a common physiologically logical approach is demonstrated in other sports as well, but the effects in apnea may be greater as the sport is rapidly developing and no general consensus concerning productive methods has yet been reached, and few studies exist.

WARM UP, OR NOT?

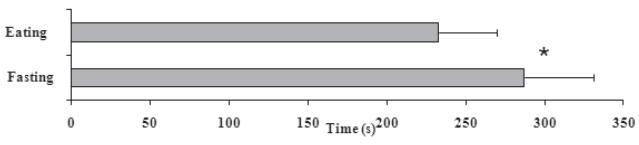
Repeated apnea, sometimes called short-term training, leads to prolongation of apnea times.[60–62] This increase in apneic duration has, at least in non-divers, both psychological and physiological components.[61] The prolongation occurs without changes in pre-apneic pCO_2, lung volume, or the diving response, most likely from the release of erythrocytes from the spleen, which enhances blood O_2 storage capacity.[62,63] These erythrocytes remain in circulation for several minutes after a warm-up apnea and would be fully saturated and contribute to oxygenation at the onset of the competition dive.[64] They may also enhance CO_2 transport out of tissues before the start of apnea and buffering capacity during the apnea. Another plausible contributor to the increased apneic duration across serial apneas could be reduced CO_2 sensitivity. A long-term reduction of the hypercapnic ventilatory response with apnea training has been noted;[65] however, there was no reduction in CO_2 sensitivity with repeated apneas at short intervals, ruling out this effect.[66]

Despite the apparent physiological logic behind warm-up dives, many top divers claim they perform better without and both approaches have their advocates, across all disciplines. The no-warm-up approach suggests the possibility that a shock response is part of the most powerful protective responses against hypoxia, quite similar to the massive diving response observed during involuntary experimental dives in seals wherein the human experimenter controls the duration.[67] However, the voluntary nature of human competitive apnea would speak against such an influence. Since apnea duration is inversely proportional to the metabolic rate,[54,68] any physical activity elevating oxygen consumption just before a dive would be counterproductive. The build up of hypoxia-related factors with incomplete recovery between warm-up dives could also limit performance in the dynamic disciplines, and the cooling effect of water on the face, maximizing the diving response, may be lost with time spent in the water.[69]

EATING OR FASTING?

The benefits of fasting before maximal-duration performance in STA may be obvious as metabolism is restricted when energy intake is low.[4] Elite apneists fasting overnight showed an increase of 23% in apneic duration compared to apneas 1.5 h after a meal (Schagatay E and Lodin-Sundström A, unpublished observations, 2009; Figure 5), twice the prolongation previously reported in inexperienced subjects.[70] Fasting before active swimming disciplines

Figure 5
Mean (SD) apneic duration after eating and after 12 h fasting in 10 divers; * $P < 0.001$



with higher energy requirements for muscle work may be more questionable, as lowering of blood glucose and liver glycogen might limit performance. While most divers practise overnight fasting before STA, many take some form of energy supplementation, but avoid large meals, before dynamic events. This discipline-specific difference in strategy was supported by the findings of lower blood glucose values before STA events (range 4.3–5.0 $mmol \cdot L^{-1}$), compared to before dynamic and depth disciplines (range 4.5–6.5 $mmol \cdot L^{-1}$; Schagatay E, unpublished observations, 2008). It has been suggested that breath-holding may be safer after carbohydrate ingestion as the more rapid build up of CO_2 will force the diver to interrupt the dive earlier,[70] but this is questionable in elite divers where hypoxia may be the main limiting factor.

Work economy

When pre-dive O_2 stores and CO_2 buffering capacity have been maximized, and baseline starting conditions have been optimized, the next task is to limit energy expenditure during swimming. Work economy is important in most sports but it is a crucial factor in apnea, as the limited oxygen stores set the outer limits for performance. A balance between restricted use of the stored oxygen reserves and the metabolic cost of swimming is essential. Several factors affect work economy during swimming.[71] The metabolic power used to create thrust for propulsion through the water should be applied with utmost efficiency, and here the position in the water, buoyancy and anthropometrics determining biomechanical efficiency are all important factors. Correct weighting is essential for performance in the dynamic disciplines. Without proper weighting, energy is expended for maintaining the appropriate depth, such that both over- and under-weighting may cause problems. The position in the water also depends on individual lung volume and pre-dive lung packing, though final adjustments can be achieved by altering swimming depth; final weighting is often done in test runs just before competition. A challenge in DNF is to be correctly weighted and remain horizontal during the two glide phases despite the shift in the centre of buoyancy.

Swimming speed must not be too slow, as basic body functions will continue to consume oxygen at a steady rate, while the rate of oxygen consumption used for swimming should be kept within limits that allow sustained muscle function without developing performance-limiting hypoxia. Water resistance rises exponentially with increasing speed, thus increasing the work of swimming. Trained swimmers use less oxygen than untrained swimmers at a given swim speed and swim faster at a given $\dot{V}O_2$.[71] A relatively high proportion of former competitive swimmers is found among the top athletes in the dynamic disciplines compared to other apneic disciplines, confirming that swimming technique and work economy are likely essential factors for performance in these disciplines. The greater distances reached in DYN, despite similar lactate accumulation and durations, show that the main difference lies in the faster speed and greater efficiency with fins. In a study comparing crawl kick with kick using small bi-fins at the same metabolic power, speed was increased by 0.2 $m \cdot s^{-1}$. At similar speeds, the energy cost of fin swimming was about 40% lower than without fins because of a 40% lower kick frequency; mechanical efficiency improved by about 10%.[72] The work economy using a monofin is likely further improved compared to small bi-fins.

Efficient turning is essential in both dynamic disciplines, and in DNF, which is usually performed in a 25 m long pool, it accounts for a considerable proportion of the propulsive force for each length of the pool. In DYN, which is mostly done in a 50 m pool, it is more likely a hindrance, as the sinusoidal stroke and relatively higher propulsive speed have to be interrupted at each turn.

Physical fitness and apnea performance

While technical skill is of great advantage in the swimming apnea disciplines, it is less obvious how general physical fitness relates to performance in all apnea disciplines. A high haemoglobin (Hb), for example, will lead to both enhanced $\dot{V}O_2$ max and increased oxygen storage, but high $\dot{V}O_2$ max is not in itself related to performance, as the diver needs to minimize $\dot{V}O_2$ during apnea. It is the oxygen storage aspect of high Hb which is important. Although breath-hold duration has been reported to be correlated to $\dot{V}O_2$ max,[73,74] two months of physical training leading to enhanced $\dot{V}O_2$ max did not increase the physiologically determined easy-going phase, showing that the two are not causally connected.[74]

Earlier studies on the effects of physical fitness on the diving response have been conflicting with a positive effect, no effect, or a negative effect reported.[75–77] We found no enhancement of the cardiovascular diving response after long-term physical training,[74] nor a correlation between competition results and hours of general physical training during two months preceding a major apnea competition (Schagatay E, unpublished observations, 2006). While swimming-muscle strength is necessary for performance, excessive muscle is costly in terms of oxygen consumption even when inactive. On the other hand, increased blood volume associated with high lean body mass may contribute to apneic duration, so the net effect is difficult to assess.[4] The morphological characteristics of competitive apneists have not been studied, but the impression is of greater variation in body composition compared to athletes in other sports.

While the benefits of general physical fitness for apnea performance may be uncertain, apnea-specific training has clear positive effects.[4,74] Some of the performance-related effects reported are:

- increased total lung volume;
- enhanced erythropoiesis;
- enhanced conscious tolerance of hypoxia;
- decreased sensitivity to hypercapnia;
- later occurrence of diaphragm contractions;

- greater psychological tolerance of diaphragm contractions;
- more pronounced diving response;
- decreased arterial desaturation rate reflecting decreased apneic metabolic rate;
- slower depletion of pulmonary oxygen and
- longer apnea times.[4]

Thus, a specific 'apnea fitness' may be reached only by apnea-related training. Additional effects are suggested by inter-group comparisons, e.g., larger spleens and higher Hb, but these differences could also reflect pre-selection and such factors need to be studied further.

Equipment

Although apneic diving is far less technology-dependent than other diving, and limiting energy expenditure is more important than maximising speed, part of the improvement in performance in the swimming disciplines has been the result of technological development. The thrust caused by human hands and feet for propulsion in water is poor compared to that by flippers of marine mammals. Only a few years ago, in both DYN and CWT, bi-fins were the main means of propulsion, whilst now, at elite levels, monofins predominate. Blade size and flexibility modifications (allowing customization), increasing the angle between foot and blade for biomechanically more efficient kicking, and drag reduction are essential in this ongoing development. While most fins used are still made of fiberglass, new materials with better mechanical properties, e.g., carbon fibre, are being introduced; but the experience of the swimmer is also essential to fin-swimming efficiency.[78]

As any failure to be correctly weighted will cause unnecessary energy expenditure to remain horizontal and at the right depth, the diver uses hip, neck and sometimes leg weights for basic buoyancy adjustments and better density distribution, depending on regional density differences due to body composition, lung volume and suit used. Divers are better weighted than previously, with well-designed weights, adjusted carefully for the predicted lung volume.

The use of low-drag suits developed for swimmers has improved hydrodynamics further, with gains in distance per stroke by 5% compared to swimming trunks.[79] Whether the recent ban on such suits in swimming competitions will be extended to AIDA* competitions remains to be seen. However, the drag of naked human skin is low with values comparable to those of slender fish such as eels.[80] To further reduce drag, masks are often replaced by goggles and a nose clip in distance disciplines. Divers may also shave head and body, or use swim caps to reduce drag. In swimmers, body shaving increased distance per stroke by 5–10% thereby reducing the physiological cost of swimming.[81,82]

Footnote: AIDA – The International Association for the Development of Freediving was established in 1992. It is the official sports body for the administration of international freediving competitions and the recognition of world records, and sets the standards for freediving education. The AIDA Assembly is made up of representatives of the increasing number of affiliated national freediving associations, <www.aida-international.org>.

Figure 6
Weine Gustavsson being coached during his preparation prior to a DYN competition dive

Psychological requirements

Once gas-storage, anaerobic capacity and work economy have been maximized, remaining paths to increased performance are to improve the diver's tolerance to asphyxia and the mental performance during hypoxia. While the biological tolerance of brain cells to hypoxia may be improved by training,[83] psychological effects of training may also help to improve tolerance to the discomfort of the urge to breathe and the capacity to focus and perform during hypoxic conditions. The capacity to cope with aching muscles and respiratory distress, and the alertness to end the dive in time are essential. Apneists use a variety of relaxation techniques in their mental preparation, both in training and in the immediate lead-up to a competition dive, but these have been little studied to date.[4] A coach may have an important role in this process during competition before dives and in the critical phase following a dive, when instructions to breathe and focus are often given (Figure 6). While in deep diving there is no way to interrupt a dive once at depth – it is an all-or-nothing event, as there is no return once commenced except back to a surface that may be far away – in static and dynamic apnea, mental stamina will have a strong influence on apnea duration and results.

Recovery from an apnea dive

After the performance of a competitive dynamic dive there is unlimited time to rest, thus accumulation of hypoxia-related factors leading to fatigue are acceptable consequences, but the hypoxic threat to consciousness is a main obstacle. There is a delay in the nadir of arterial oxygen saturation after apnea

Figure 7
Safety diver accompanying Martin Stepanek during a DYN swim, World Championships 2006

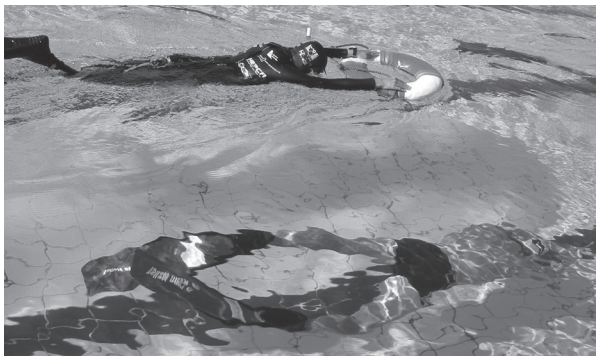


Figure 8
Per Westin surfacing from a DYN competition dive; note the safety diver immediately available to assist

Figure 9
The surface safety protocol: the diver removes his goggles, signals OK after which he says he is OK

termination, because of the circulation time from the lung to the brain, especially with vasoconstriction.[17] Therefore, the recovery and safety procedures are particularly focused on this period.

To avoid syncope after surfacing, most divers use 'hook breathing', in which the breathing cycle is interrupted on inspiration and ends with a Valsalva-like manoeuvre, the subsequent expiration being done against some resistance. This technique, which awaits scientific evaluation, resembles the post-dive breathing pattern used by Ama divers who exhale against resistance, making a characteristic whistling or moaning sound (Schagatay E, unpublished observations, 2009). Ama divers state that the technique speeds up their recovery. This may be especially important after deep diving, where it would probably also aid in reversing the blood shift into the thorax, but it is used in all apnea disciplines.

There is, at present, no evidence that repeated syncopes cause any long-term effects on cognitive functions.[84] Elevation of S100B, a marker of brain damage, occurs after long, experimental apneas, but only to levels not normally associated with brain damage.[85]

Safety procedures during AIDA competitions

While distance swimming in a pool is an occasional cause of death in recreational breath-hold divers, pool competitions under AIDA rules have been comparatively safe because of the rigorous safety systems put in place. Well-trained safety divers follow the competitor at arm's length throughout the swim to provide immediate assistance in the water at the first signs of hypoxia (Figures 7 and 8). In all apnea disciplines, whether in a swimming pool or open water, a surface protocol must be performed by the diver within 15 s of surfacing. This involves removing the facial equipment, showing an 'OK' hand-signal, and stating verbally "*I am OK*", in that order (Figure 9). Any failure to perform this procedure, in the correct order and without overlap between the three maneouvres will lead to disqualification. The diver is not allowed any physical help within 30 s after a dive if it is to be registered as successful, but may receive instructions from the coach. Other causes for disqualification are dipping of the airways underwater during this period, or so-called 'post-blackout mechanical movements' involving involuntary repeated nodding of the head suggesting 'micro syncopes', or hypoxic loss of motor control (known amongst the divers as "sambas") compromising the surface protocol.

Should syncope occur, the safety diver will first do a 'blow, tap, talk' procedure (similar to the shake and call at the start of basic life support – BLS), with which, in over 90% of cases, the diver resumes breathing within seconds. If further support is needed, rescue breathing is initiated and, in extreme cases, full BLS. Trained medical personnel and equipment for oxygen treatment are immediately available on site. To date, there have been no fatalities during AIDA-

organized competitive apnea events. Competition regulations require competitors to have an annual medical assessment, and medical personnel at the competition site can remove an athlete from competition should a medical problem arise.

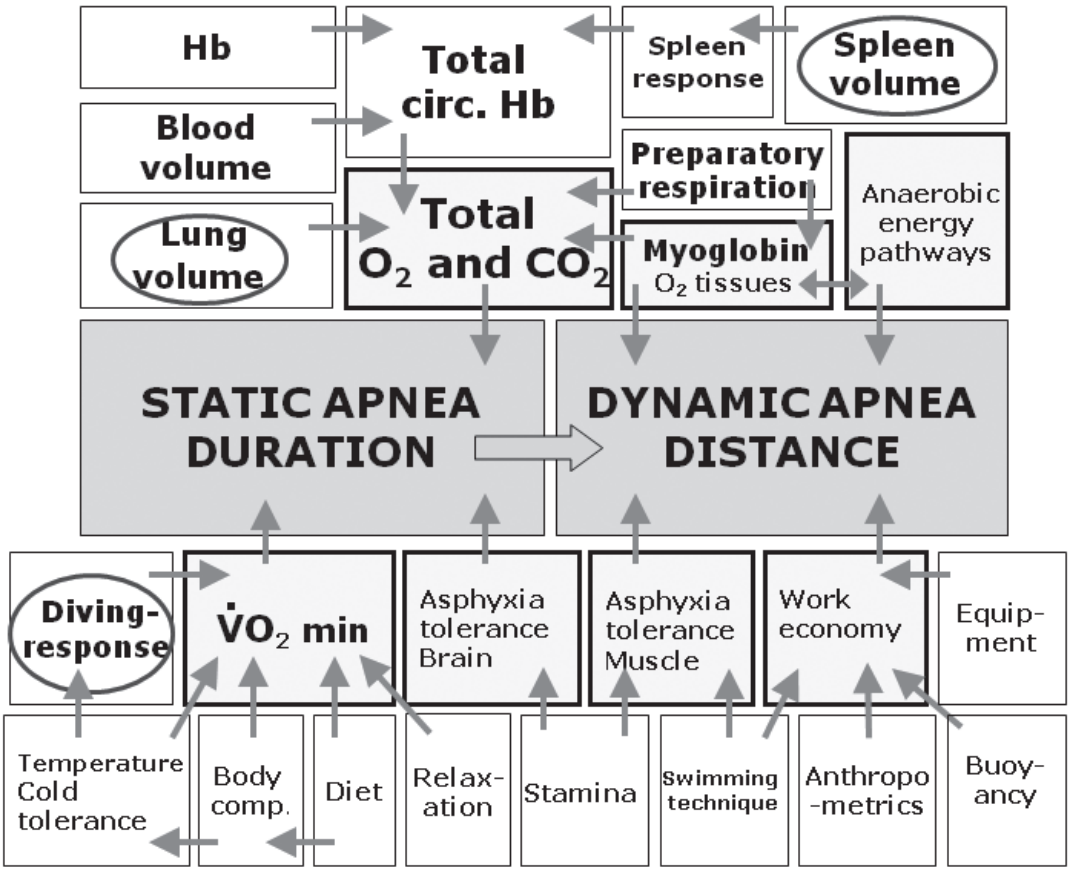
The safety measures adopted during competitions make these events much safer than the training done by many apnea athletes, or diving by spear fishermen and recreational freedivers without proper safety education and training. Freediving on any level should only be done with a dive buddy with enough knowledge to assist in an adverse event. Hyperventilation should not be practised by recreational free divers and diving time, depth and resting intervals should be kept to sensible limits based on individual ability. It cannot be overemphasized how important it is to contact diving schools that provide proper apnea diving education, whether one wishes to start freediving for pleasure, food gathering or competition. Any activity involving maximal apnea attempts will always carry a great risk of syncope, and should only be practised in the presence of trained safety divers. The goal with serious dive training should not be to get closer to one's existing limits, but rather to move these forward by proper training, in order to maintain the same safety margins. This is possible as many factors essential for performance can be improved by training. The safety systems developed by competition divers will likely keep pace with increased results at least in the pool disciplines. These measures could be imported to other diving activities, a process which is currently taking place in Sweden, where recreational freediving education within the Swedish Sports Diving Federation is developing similar safety routines.

Competitive apnea is here to stay, no matter what view is adopted by the medical community. As in politics, dialogue is the key to maximising safety, rather than a wall of silence or demands to ban the sport.[86] Clearly we best serve the cause of safety by providing sound advice to athletes on how to avoid injury, based on knowledge of the competition conditions associated with the specific apnea disciplines and data from physiological laboratory and field studies.

Predicting performance

By adding new factors to the figure used for static apnea in the first article,[4] we obtain an overview of the major physiological, psychological and physical factors predicting performance in the dynamic disciplines (Figure 10). The main factors have arrows directly into the discipline boxes, and these in turn are determined by a number of factors placed further out (arrows indicating interactions between

Figure 10
Factors predicting performance in the competitive apnea pool disciplines
(arrows indicating interactions between non-adjacent boxes have been omitted for clarity)



non-adjacent boxes have been omitted for clarity). All factors determining static apneic duration also affect the dynamic disciplines, as indicated by the horizontal arrow between the two, but a new set of factors related to work is added by DYN and DNF. Most biological factors show both a component of inherent individual variation and a component of variation induced by training. Ideally, when the detailed interdependencies among these factors have been determined, and these can be reliably measured, the model could be used to predict individual performance at a given state of training, but such predictions are premature due to the limited data available from dynamic apnea disciplines. Individual differences in predisposition for apneic diving are remarkable, and increased recruitment of athletes to the sport and improved training methods may forward these limits beyond those considered realistic today. In a survey of 17 world-championship participants in 2008, the divers predicted a future limit for DYN to be around 325 m. This could physiologically be within reach with improvements in work economy, gas storage capacity, anaerobic capacity and further technological developments, and in these pool disciplines safety can be kept high as the dive can be interrupted by the competitor or safety diver within seconds. Further improvements in the current world records in DYN and DNF ultimately depend on the balance between total gas storage, anaerobic capacity and the metabolic requirements at the optimal swim speed, individual stamina and brain tolerance to asphyxia, without resulting in syncope.

Conclusions

A successful diver in the dynamic apnea disciplines is likely to possess superior swimming skill and excellent work economy, balancing speed and restricted energy expenditure to achieve maximum distance within the limits set by maximized gas storage capacity and the tolerance of the brain to asphyxia. Anthropometrically for DNF, a strong upper body, long arm reach and big hands and feet may be beneficial. For DYN, the diver should, instead, have a good fin-swimming technique, flexible back and pelvic region, and a powerful diving response, shutting off the circulation to non-working areas. High lactate values after competition dives indicate that the anaerobic capacity of muscle is important to maximal performance. Enhanced myoglobin concentration in swimming muscles could contribute to prolonging the aerobic dive limit, and high local tolerance to hypoxia is important for successful performance. These factors all affect the metabolic part of the model but, as in other apnea disciplines, the highest possible gas storage and minimum oxygen saturation requirements for alert consciousness at apnea termination set the outer limits for dynamic performance. Just as in static apnea, the diver can spontaneously terminate the dive at any time and breathe, and psychological 'stamina' (self discipline) as well as good judgement is essential for performance. Future physiological research should be directed towards determining myoglobin levels as well as local anaerobic capacity in these highly specialized athletes. Studies of blood flow redistribution by the diving response in laboratory models of dynamic apnea will determine which areas are 'sacrificed' and which are prioritized, an aspect central to defining performance.

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Basic mechanisms of gas transport and past research using perfluorocarbons

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Key words
Perfluorocarbons, gas solubility, oxygen, nitrogen, carbon dioxide, nitric oxide, solubility, review article

Abstract

(Spiess BD. Basic mechanisms of gas transport and past research using perfluorocarbons. *Diving and Hyperbaric Medicine*. 2010;40(1):23-8.)
Perfluorocarbon compounds have been utilized either in pure (neat) form or as emulsions suspended in aqueous fluids. These man-made chemicals possess a unique physical property allowing them to dissolve much more of the respired gases (oxygen, nitrogen and carbon dioxide) than any water-based system. Understanding the basic physical chemistry surrounding these emerging medical technologies will assure they are utilized to maximum benefit for mankind. It is clear they should not simply be viewed as 'blood substitutes' but rather as enhanced gas-transport pharmaceuticals.

Introduction

Carbon fluoride chemistry had its early beginnings in the 1930s–40s, during which time a unique feature of carbon halide bonds was discovered – a very high-energy ionic attachment. When a carbon chain or cyclic structure is completely substituted with halogens the ability of other carbon compounds to attack or change the parent structure is greatly limited.[1,2] They become, to a very great extent, inert and unable to be changed. The carbon-fluoride bond is unique in having the highest energy of any organic bond, 120 kcal·mole^{-1}. Solid perfluorocarbon (PFC) coatings are utilized to make non-stick pans. Liquid perfluorocarbon oils have other very useful properties.

During the Manhattan Project, it was discovered that such pure PFC oils were inert insulators. Uranium and plutonium could be stored safely in containers of PFC oils without fear of degradation and/or reaction. One would suppose unplanned reactions between uranium and plutonium could be a bad thing! A serendipitous observation occurred during such storage of radioactive material when it was noted that a tremendous amount of oxygen (O_2) dissolved in the oil. That observation went uninvestigated until the late 1960s and early 1970s, when a group of physiologists suggested that perhaps such oils could be used for medical purposes. LC Clarke, the famous physiologist and inventor of electrodes for pressure and biochemical measurements, along with Geyer and Galon, began experimenting with such PFC liquids.[1–4] They quickly found that tremendous amounts of dissolved O_2 and other respiratory gases could be harbored in equilibrium in such PFC oils. The now classic demonstration of rodents spontaneously breathing oxygenated PFC created outcries both of animal cruelty and fascination. Goldfish could swim in the water above the PFC and, as long as the PFC was in contact with 100% O_2, it seemed animals could live with liquid-PFC breathing for long periods, emerge and survive. This 'scientific trick' was picked up by Hollywood in the movie *The Abyss*. This was not just science fiction, as the producer had turned to advice from several excellent scientists such as Thomas Shaffer and Marla Wolfson, who were in liquid PFC-breathing research.[5–8] *The Abyss* did, however, spawn conjecture that perhaps such human liquid-PFC breathing could either be used as a way to escape from a disabled submarine or work as a new technology for deep sea exploration. Today, science fiction may be yet closer than we had previously believed.

Physiology of PFC usage

This article, however, is intended to discuss the physiology and physical chemistry whereby PFCs, either as pure oils or in intravenous emulsions, can enhance and change mammalian gas exchange.[9] The understanding of how these technologies work may well soon change medicine and mankind's future; but it is only through a careful understanding of their capabilities and limitations that science, not science fiction, will move them to technological reality.

Respiratory gases are transported both by active chemical binding and by passive solubility in fluids.[10] O_2 and CO_2 bind to haemoglobin as well as other metalloproteins throughout the body. The reactions are complex and widely reviewed elsewhere. What is important with regards to PFCs is the fact that, although erythrocytes carry vast amounts of O_2 within them, it is dissolved O_2 that ultimately fuels the energy production in target mitochondria. The red cells create a microenvironment of high-pressure O_2 immediately outside of their cellular membrane. Within Angstroms from their surface, the dissolved O_2 levels drop.[11] A popular myth exists that somehow cells pull O_2 from erythrocytes. It is rather that red cells, through biochemical changes in their cellular pH and 2,3 diphosphoglycerate (2,3-DPG), chloride ion, etc, release more O_2 thereby increasing dissolved O_2 in the local environment. The closer a red cell approaches the wall of a blood vessel the higher the local O_2 concentration gradient for cellular uptake. Total

Table 1
Oxygen content equations with and without perfluorocarbons present; note the dramatic increase in dissolved O_2 when PFC is present[9] (reproduced with permission with minor modification)

O_2 content equation	$Cao_2 = [1.36 \times Hb_{(conc)} \times Hb\ Sat] + [0.0031 \times PaO_2]$	Cao_2: total oxygen content; Hb_{conc}: haemoglobin concentration; Sat: saturation; Pao_2: arterial partial pressure of oxygen
O_2 content equation with PFC present	$Cao_2 = [1.36 \times Hb_{(conc)} \times Hb\ Sat] + (0.0031 \times PaO_2) + (0.1432 \times PaO_2 \times \beta)$	β: Fluorocrit (percentage of whole blood taken up by PFC particles – Oxycyte)
O_2 content equation done a different way with a second-generation PFC – Perflubron	O_2 blood $= (Y \times O_{2max}) + (4.7 \times 10^{-3} \times V_{RBC} \times P) + (2.9 \times 10^{-3} \times V_{plasma} \times P)$	Y: relative saturation of Hb; O_{2max}:. maximum O_2-carrying capacity of Hb (100% saturation; ml O_2.100 ml^{-1} blood) and equals 0.45 × %haematocrit; V_{RBC}: fractional volume of the red blood cell; V_{plasma}: fractional volume of the plasma; P: total ambient pressure. A 1 g.PFC per kg BW dose added to the blood produces a 30% increase in total O_2 in the blood (all present and available for metabolism, since it is dependent only on Henry's law)
Gradient of O_2 from an erythrocyte (Hb) to the mitochondria	$VO_2 – DO_2\ (PcO_2 – PmitO_2)$	VO_2: O_2 uptake; DO_2: O_2 diffusing capacity; PcO_2: average capillary partial pressure O_2; $PmitO_2$: average mitochondrial partial pressure O_2

O_2-carrying capacity can be calculated from a standard equation (Table 1).[9] That equation takes into account, but downplays, dissolved O_2 in plasma. Indeed, in most medical teaching the content of dissolved O_2 is disregarded, yet it is dissolved O_2 that the mitochondria actually utilize. Therefore, erythrocytes function as a bank of stored O_2 that continuously overpressurizes the aqueous plasma fluid such that the net flow of O_2 is to the mitochondria of metabolizing cells. As blood courses through tissues, it exchanges O_2 between venous and arterial blood, as well as driving it into tissues. The levels of various gases within tissues is noted in Table 2.[12] The plasma not only is a conduit for gas movement but a resistor, as its capacity for gas solubility is quite limited (Figure 1).[13]

Understanding that the plasma gap functions as a resistor brings to light how PFCs may well be utilized for the future.[13] First, one has to understand the physics of gas solubility in PFCs. Henry's Law states that "*at a constant temperature, the amount of gas dissolved in a liquid is directly proportional to the partial pressure of that gas in equilibrium with that liquid*."[9] Every fluid has an inherent solubility coefficient for every gas, dependent upon relative molecular polarity and molecular size. Water is a highly polar molecule, whereas lipids tend to be considerably less polar. Most fats, however, still have a large number of protons (hydrogen atoms) as side chains and, therefore, are relatively polar. Once a hydrocarbon molecule has all its available valences substituted with halogens (preferably fluoride), then the resultant carbon-based oil becomes highly non-polar. PFC gas solubility is noted in Table 3.[14]

Pure PFC can carry large amounts of O_2 dissolved at 101.3 kPa.[14] Even more soluble than O_2 is carbon dioxide (CO_2), and nitrogen (N_2) is somewhat less soluble in PFC than is O_2. However, N_2 is highly insoluble in water. Remember the O_2 solubility in plasma is 0.0031 ml·100ml^{-1}, whereas for a PFC emulsion (not pure PFC) the solubility of O_2 is 50-fold higher (Table 1). These facts can be utilized in making gas solubility and content equations (Table 1).

Figure 1
Representation of a tissue bore with a capillary running through it; the cylinders inside the capillary represent red blood cells separated by plasma gaps; various resistances to O_2 movement are indicated, with corresponding PO_2 within the plasma, vessel wall and tissue[13] (adapted with permission)

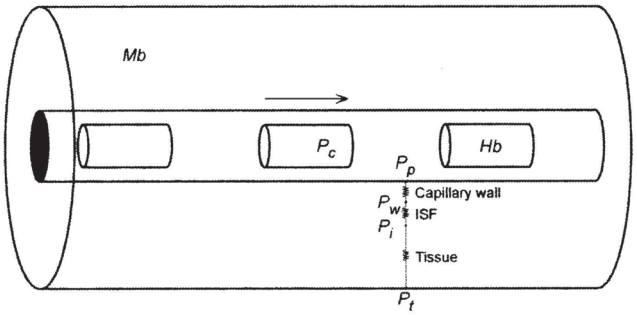


Table 2
Gas partial pressures in various parts of the human body; note the low tissue PO_2 and that all tissues are saturated with N_2 at sea level

Sample site	O_2	CO_2	N_2	H_2O	Total
Inspired air	158	0.3	596	5.7	760
Expired air	116	32	565	47	760
Alveolar air	100	40	573	47	760
Arterial air	100	40	573	47	760
Venous blood	40	46	573	47	706
Tissues	≤30	≥50	573	47	700

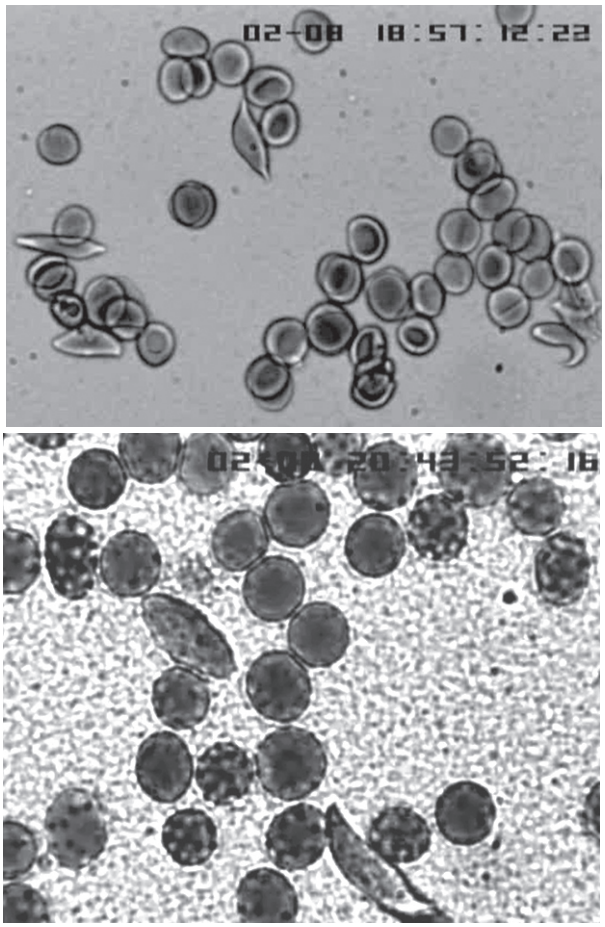
Table 3
O_2 solubility at 101.3 kPa in different pure perfluorocarbons; note that these PFC emulsions are between 20–60% PFC whereas modern emulsions are 40–60% PFC; one is limited in usage of the products to approximately 2–5% fluorocrit[9]

Compound	Solubility ($mlO_2 \cdot 100ml^{-1}$ of compound)
Perfluorodihexyl ether	55.42
Perfluorodibutyl sulfur tetrafluoride	48.02
Perfluorotriisobutylamine	44.37
Perfluoro-(*N*-ethylmorpholine)	50.10
Perfluoro-*N*,*N*-dipropylmethylamine	52.60
Perfluorotriethylamine	53.86
Perfluoro-*N*-methylpiperidine	41.33
Perfluoro-*N*-methylmorpholine	37.57
Perfluoro-*N*,*N*-dimethyl-*N*-hexylamine	51.51
Perfluoro-*N*-butylmorpholine	50.59
Perfluoro-4-(*N*,*N*-dimethyl-2-aminoethyl)-morpholine	45.07
F-Tertbutylperfluorocyclohexane	43.00

Pure PFC has been utilized to enhance O_2 delivery in the lungs.[5–8] Small amounts have been nebulized, causing a coating of PFC in the alveoli and terminal bronchi. These experiments seemed to use the PFC as a surfactant, although the enhanced O_2 solubility may have enhanced gas delivery. Also, fully filling the tracheo-bronchial tree with pure PFC and creating liquid-PFC breathing has been accomplished in both animal and human studies. This works to enhance gas transport, and data from respiratory distress syndromes suggest that it may have a clinical application there in the future. Some studies have suggested that by using liquid-PFC breathing, possibly in conjunction with intravenous PFC, decompression sickness could be averted.

Most PFC utilizations in commercial development have focused upon intravenous emulsion technology.[9] PFC, an oil, is immiscible with plasma, therefore, micro-particles of fat emulsions (micelles) have been created.[15–17] At least one technology utilizes perfluorododecapentane, which has a boiling point close to body temperature.[18] This is injected in liquid form and flashes instantly into micro-gas particles, leading to enhanced gas transport within the particles. However, the rest of this article will focus upon the biophysics and chemistry of the emulsions in preparation today.[19–21] Modern-day emulsions are made from egg yolk phospholipids, quite similar to propofol and intralipid. The particles closely range around 0.2 microns in diameter, in comparison to an erythrocyte which is 5–8 microns. The micelles are considerably denser than other formed elements of the blood, and they will separate out in low-flow or standing fluids. However, with normal blood flow the PFC micelles are dispersed between red cells and also pushed to the walls of the blood vessels. The plasma gap essentially is replaced with micelles forming a gas-conduit bridge from

Figure 2
Human sickle red cells in an artificial capillary; A: normal plasma, B: PFC has been added; note the granular appearance of the PFC micelles. The dramatically increased solubility of O_2 in these micro particles overcomes the resistance of the plasma gap when PFC is added to whole blood[9] (reproduced with permission)



red cells to endothelium and vice versa (Figures 1 and 2). If one considers that the solubility of respiratory gases is up to 50 times higher than in plasma, then this bridge of microparticles allows for rapid gas transfer. Diffusion speed appears to be enhanced by PFC micelle presence, but in reality it is simply a function of enhanced solubility of gas within each micelle and the close proximity of each micelle that makes gas movement appear to speed up.[9]

Because gas molecules in PFC micelles are not chemically bound but are held through enhanced solubility, every molecule of O_2 is available for metabolic utilization. In whole blood, haemoglobin has a complex interaction between each added O_2 molecule in the four haem moieties, as well as being modulated by pH, chloride ion, and 2,3–DPG. Therefore, under normal physiologic conditions, the maximum 21 volumes per cent of O_2-carrying capacity can only release from haemoglobin approximately 5–6 volumes per cent of O_2 (tissue demand). PFC micelles are

rapidly in equilibrium with any microenvironment they inhabit. It is gas solubility according to Henry's Law and the relative gas partial pressures in those tissue/blood or lung microenvironments that determine the content of gases dissolved within them at any one time. PFC can be effective as a third compartment of gas-carrying capacity within the blood stream. The amount of added potential gas-carrying capacity can be calculated (Table 1).

However, gas delivery from the red cells should be thought of as the most important physiologic contribution of PFC. Using normal and low haematocrit blood, adding PFC emulsions increases the mass transfer coefficient by 14% or more. Convective gas movement (forward propulsion of blood) and diffusive gas movement are considerably different.[13] With normal whole blood, convective movement must be present, otherwise tissues become hypoxic very quickly and withdraw all available O_2 from the microcirculation. In several recent studies, it has been shown that, with PFC present in the microcirculation at low or no-flow convective states, tissue O_2 delivery remains present. This must be due to the massively enhanced diffusion effects.

O_2 diffusion is important for normal metabolic function. The first generation PFC that garnered approval for treatment of myocardial ischaemia during balloon angioplasty was not easy to use commercially and was withdrawn from the market. Today, a third-generation compound, (Oxycyte™, Oxygen Biotherapeutics Inc, USA) is being tested for a wide range of tissue ischaemia indications, including traumatic brain and spinal cord injury, organ preservation, carbon monoxide poisoning and cardiopulmonary resuscitation.

N_2 is highly insoluble in whole blood and tissue. Rapid changes in ambient pressure can cause supersaturation leading to formation of a gas phase in blood and tissues. PFC has been shown to increase xenon (another highly insoluble gas) movement out of striated muscle by well over 100%. In multiple experiments using PFC infusions, air embolism effects have been reduced.[17,20,22–30] These studies show dramatic reductions in organ effects. The speed of bubble resolution has been shown to be increased by PFC presence, but the entire story is not simply the increased solubility of N_2 in PFC.[30] Bubbles sticking and interacting with endothelial cells are decreased, perhaps by the surfactant effects of the PFC as well as its emulsifiers. PFC also has under-investigated, independent anti-inflammatory effects and may preserve endothelial cell function. It is entirely possible that PFC may change the stress induced upon endothelial cells when a bubble is present in the microcirculation and that the glycocalyx itself is better preserved.[31] Whilst PFCs speed up the dissolving of N_2, perhaps the more important effect is on O_2 delivery to tissues that would otherwise become hypoxic from the blood vessel blockade due to bubble formation.

CO_2 is far more soluble in PFC than in either whole blood or plasma, and, when PFC is present, cerebral blood flow increases, perhaps 10% or more. Whether this is due to fluxes in CO_2 or whether other mechanisms are at play is not established.[9] Either way, in models of neurologic ischaemia, traumatic brain and spinal cord injury and stroke, PFC appears to have a unique neurological protective effect.

Another important gas in the microcirculation is nitric oxide (NO). NO exerts so many different cellular function-controlling events that it is hard to generalize everything it does. NO is probably very highly soluble in PFC micelles. Only a small number of studies to date have looked at NO effects in the presence of PFC. It appears that PFC at first works as a NO sink, but once it is equilibrated, it then may act as a NO donor.[32] To date, no one has tried attaching NO donors within the micelle itself or pre-equilibrating the emulsions with NO before infusion. Just as PFC micelles enhance movement of O_2 from erythrocytes, so might such micelles enhance the movement of NO from endothelial cells to erythrocytes. Normally haemoglobin is a NO binder and, being encased in the red cells streaming through the centre of capillaries, it is held away from endothelial cells. If PFC enhances NO movement from endothelial cells to haem proteins, one might expect to see hypertension, but this is not described as a side effect of PFC infusions.

Conclusion

In the future, it is likely that an intravenous PFC will become approved as a treatment for tissue ischaemia. Today, a phase IIb double-blind, placebo-controlled, large (128 patients), dose-escalation trial of PFC for the treatment of civilian closed-head injury is underway. Animal studies in blast-induced traumatic brain injury are showing efficacy, and that programme is expanding quickly due to its military importance. The use of PFC infusions for sickle cell crisis and carbon monoxide poisoning is being researched as are other indications. In a second article, the use of PFCs for decompression illness will be reviewed. Suffice it to say that success in such a treatment is dependent upon understanding of the physico-chemical means by which PFC emulsions can carry both O_2 and N_2. The future investigation of enhanced delivery/removal of respiratory gases by PFC will almost certainly encompass a more basic understanding of the physiology of CO_2 and NO fluxes when PFC is present. To look way into the future, the use of liquid-inhaled PFC may yet find a medical usage. It does offer the possibility of being used as a method to create or enhance suspended animation as well as for individual organ preservation.[33] Work and discussions are on-going with space agencies to understand how this might be possible.[34] PFC as a tool for medical application, temporarily changing the way that respiratory gases are transferred within the body, is very exciting.

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The potential role of perfluorocarbon emulsions in decompression illness

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Key words
Perfluorocarbons, decompression sickness, decompression illness, air embolism, treatment, research, review article

Abstract

(Spiess BD. The potential role of perfluorocarbon emulsions in decompression illness. *Diving and Hyperbaric Medicine*. 2010;40:28-33.)
Decompression illness (DCI) is an occasional occurrence in sport, professional, and military diving as well as a potential catastrophe in high-altitude flight, space exploration, mining, and caisson bridge construction. DCI theoretically could be a success-limiting problem in escape from a disabled submarine. Perfluorocarbon emulsions (PFCs) have previously been investigated as 'blood substitutes' with one approved by the United States Food and Drug Administration for the treatment of myocardial ischaemia. PFCs possess enhanced (as compared to plasma) respiratory gas solubility characteristics, including oxygen, nitrogen and carbon dioxide. This review examines approximately 30 years of research regarding the utilization of PFCs in gas embolism as well as experimental DCI. To date, no humans have been treated with PFCs for DCI.

Introduction

Decompression illness (DCI) is an incompletely defined clinicopathological diagnosis in humans with a wide spectrum of presenting signs and symptoms.[1,2] The disease is caused by gas bubble formation/movement in tissues and within the vascular tree or by gas forced into the circulation from pulmonary barotrauma. These gas bubbles cause either primary direct tissue destruction or secondary events from decreased blood flow (oxygen delivery), endothelial cell dysfunction, inflammation, coagulopathy/thrombosis and many other effects. The readership is familiar with many of the manifestations and difficulties with the diagnosis of DCI and is referred elsewhere for review.[1–3]

Mankind lives and works most often in a narrow range of ambient gas pressures. The gas column above us functions as a fluid, exerting continuous equal pressure to all parts of the body. Gases are soluble in tissues and blood, based upon Henry's law. At 101.3 kPa (1 bar) the human body is saturated, with all respiratory gases in equilibrium with the partial pressures of each gas. Seventy per cent of the body is made up of water, therefore the relative solubility coefficients for respiratory gases in water versus fat (oils) determine the total amount of gas dissolved in aqueous media or tissues at any one time.[4,5] It is through a sudden decrease in ambient pressure that tissues and blood potentially become supersaturated with gases. Supersaturation leads to bubble formation. The respiratory gases leave their dissolved state when some, as yet undefined, parameter allows for a small nidus of micro-bubble formation to occur.[6] It has been suspected that micro-particles allow for the original formation of micro-bubbles.[6] Once formed the micro-bubbles grow, potentially rapidly, as local, supersaturated gases move from tissue and blood into the gaseous phase of the micro-bubble.

Growth of a bubble is dependent upon gas composition, internal/external pressures and the surface tension of the bubble itself.[6] Bubble dynamics is an entire study unto itself, but, suffice to say, particularly within the blood stream, bubbles are rapidly coated with proteins that themselves then have complex interactions with tissues, cells and the micro-environment.[6–7] DCI is often thought to be a disease of diving, with inadequate times for gas equilibration between various faster and slower equilibrating tissue categories. However, any rapid reduction in ambient pressure may cause DCI and as man ventures into ever more unique

environments (deep ocean, high altitude and space) the potential for DCI will be ever more challenging.

To date, the standard of care has been to treat suspected DCI with high inspired oxygen (O_2) and, as rapidly as possible, with recompression therapy.[2,8] Recompression therapy requires specialized, expensive, bulky, not easily transported equipment, as well as trained personnel. Transport from remote accident/dive sites to an available treatment chamber may require many hours, involve difficult logistics and therein further delay treatment, as well as altitude exposure during air transport. A robust, easily administered, effective, non-toxic therapy that could begin as soon as DCI was suspected would, therefore, represent a dramatic advance in treatment. The remainder of this article will explore work performed to offer a potential alternative/supplement to existing recompression therapy.

Prior PFC studies

Perfluorocarbons are organic-based oils that have complete substitution of the carbon hydrogen bonds with fluoride.[9,10] Respiratory gases are non-polar molecules whereas water is a highly polar molecule. The gas solubility coefficient for gases is dependent upon relative polarity between the gas and the solution as well as molecular size. Organic fluids, for example fats such as membranes, have a higher solubility coefficient for respiratory gases than water. Plasma (60% of blood) is water-based, with some proteins and fat micelles. Blood supplies O_2 to cells through a complex interaction of erythrocytes (acting as an O_2 bank) and dissolved O_2 in plasma. Nitrogen (N_2) and other insoluble gases have a limited capability of being carried in blood and plasma. PFCs, because of their change in molecular polarity, change gas solubility dramatically. PFCs have a 50- to 60-fold enhanced O_2 solubility as compared to aqueous media. Their ability to dissolve N_2 may be even greater. The use of PFC emulsions (stable intravenous solutions) offers an attractive possibility for the treatment/prevention of DCI. The preceeding article in this issue describes how PFCs act as gas transporters.[11]

Early work with PFC emulsions utilized a relatively weak (less concentrated) set of emulsions. FC-43 and Fluosol-DA 20% were 10% v-v of PFC as compared to solutes and emulsifying agents. Fluosol-DA 20%, a Japanese-manufactured emulsion, was the first 'blood substitute' FDA-approved PFC for human usage.[12] FC-43 had a longer half-life and was utilized for animal studies only. These were investigated in the 1970s and 80s as potential 'artificial blood' compounds. A series of cases of use in humans of Fluosol-DA 20% for patients refusing blood transfusion was heralded and its usage for coronary ischemia made it commercially available.[12] The emulsion itself was cumbersome (supplied frozen and not completely emulsified) and for that reason it was withdrawn from the market in 1994.

In DCI, the earliest work was in rodent models.[13–15] Work in hamsters and rats showed that the use of these agents could potentially prolong life after usually lethal experimental dives. Neither of the groups that did the early work in these rodent models followed up with larger animal models or proposed going to human trials. This initial success did spur other work, but the focus was more on arterial gas embolism (AGE) in heart surgery than on the treatment of DCI in dive accidents.

Treatment of venous (femoral vein) gas embolism (VGE) in rabbits was investigated as a model of surgical gas embolism prevention, and provided early experimental evidence that PFCs could perhaps absorb air.[16] In both continuous ($0.25 ml \cdot kg^{-1} \cdot min^{-1}$) and bolus air embolism models, the instillation of FC-43 dramatically prolonged life in 100% O_2-breathing animals. Both venous and arterial partial pressures of O_2 were better in the PFC groups, whilst central venous pressure was lower in the PFC, O_2-breathing group, suggesting partial resolution of bubble size. In the PFC group breathing room air, the average time to death was the same as in those who did not receive PFC. Of interest, that particular PFC emulsion was stabilized by a unique emulsifier. The use of emulsifier alone did not enhance prolongation of survival.[16] Other studies using larger animal AGE and VGE models showed promise in terms of prevention of organ damage (stroke and myocardial infarction as well as death).[17–20]

An awake-rat, DCI survival study with compression to 690 kPa (6.8 bar) for a short bottom time also demonstrated that PFC infusion immediately after surfacing, but prior to the full development of DCI, dramatically decreased lethality by 24 hours (control 11/12 versus PFC 4/12).[21] The numerical reduction in lethality is not the entire story. Those that died of DCI in the untreated group did so very quickly whereas even those that did die of DCI after PFC treatment and 100% oxygen breathing did so much later in their time course. These first rodent experiments were focused on the efficacy of PFCs for a much more prevalent problem, AGE and VGE in surgery, particularly cardiac surgery, rather than on DCI.

A model of gas saturation in muscle tissue examined radioactive xenon washout. Xenon is an inert and relatively insoluble gas.[22] Dogs were allowed to breathe radioactive xenon until such time as they had saturated all their striated muscle tissues. They were then given either a saline control volume expander or an equal volume of PFC emulsion and the speeds of off-gassing of the xenon and muscle washout were examined. It was calculated that, given the dose of the PFC utilized, its relative amount in a stable emulsion, volume of distribution, etc, that the speed of muscle removal of xenon would be increased by 77%. In actuality the speed of xenon washout exceeded that and was increased 109%. It should be noted that this did not come anywhere near a 30- to 50-fold increase in solubility that could be calculated for xenon in pure PFC alone. However, because the amounts of PFC in stable emulsions are limited and the amount of

emulsion added to the circulating volume is again limited, the washout speeds achieved are more modest.

The findings of PFC-enhanced survival after experimental air-dive DCI went under-appreciated for about a decade. In the late 1990s, the PFC literature was reviewed and a large animal (swine) saturation dive model with >85% lethality perfected. These investigators had used this dive profile as a standardized tool with which to investigate a number of interventions. The United States Navy, in developing this large swine model, has utilized it as a more reliable reflection of human physiology than rodent models. In a now landmark study, 20–25 kg swine were compressed to 485 kPa (4.8 bar) for 22 hours (thought to be a N_2 saturation dive).[23] From that depth, they were rapidly decompressed (202.6 kPa·min^{-1}). The day prior to diving, the swine had an indwelling catheter placed for intravenous or PFC infusion after surfacing. Upon surfacing, the animals were immediately removed from the chamber and placed in plastic cages breathing either room air or ≥ 95% O_2. Three groups were studied: a control group which received volume expansion but breathed room air; another group with volume expansion and enhanced O_2 and the experimental group which received 6 ml·kg^{-1} of PFC (Oxygent™, Alliance Pharmaceuticals Inc, San Diego, California) within 10 minutes of emergence. The results were dramatic, with a reduction in lethality from 85% to 15%. Of interest, this reduction of lethality was similar to that seen in the rodent experiments. Of great interest was the observation that any animals that did succumb in the PFC-treated group died a sudden death. Such animals were up and around their enclosures walking, eating and drinking normally then suddenly collapsed and died as though an air embolism had obstructed a coronary artery. Those who breathed room air or O_2 succumbed to neurological (ataxia and paralysis) and cardiopulmonary DCS (pulmonary oedema and tachypnoea). Thus, not only was there a quantitative difference in lethality, but also a clear difference in the physiological path leading to death.

Other work in AGE had shown that PFCs infused to a cardiopulmonary bypass (CPB) machine prior to either routine CPB or a massive cerebral AGE could dramatically reduce the effects of embolism. AGE is a near-universal event in CPB for heart surgery. Massive AGE, however, is a rare and devastating event, whereas micro air emboli happen in every case. Retinal angiography has shown that temporary occlusion of arterioles occurs in most CPB cases and that by 45 minutes after surgery these occlusions have passed.[24] With a first-generation PFC infusion in dog models of CPB, up to 95% of the microvascular obstructions from AGE could be prevented.[25] In the massive air embolism model, PFC infusion prior to AGE prevented cerebral strokes, attenuated electroencephalographic insult and actually increased brain blood flow. Furthermore, in some unique studies of retinal endothelial permeability after AGE, it was shown that, by using PFC prior to an AGE, endothelial integrity could be maintained.[26,27] Clearly PFC pre-treatment protected the vasculature from the effects of transiting air. Later work examining present-day PFCs and isolated, cultured endothelial cells has supported this earlier CPB work.[28] Other research has shown that PFC given prior to AGE decreases a bubble's dwell time in the pre-capillary arterioles. Furthermore, they have shown that air bubbles, when touched to the cell membrane of cultured endothelial cells, will cause a programmed cell death for those cells. In the presence of PFC, this effect of killing endothelial cells is largely prevented for reasons that are still not clear. Eckmann has also shown that bubble dissolution time is sped up when PFC is present in the microcirculation. This work was conducted using a Russian PFC formulation very similar to the weak, first-generation FC-43 and Fluosol-DA 20%.[29]

Essentially the work by Dromsky et al could be viewed as either pre-treatment or very early treatment/prevention of DCI. The swine, when surfaced immediately, received intravenous PFC. The AGE and VGE experiments were carried out with the same idea of pre-treatment. In a series of studies trying to understand the physiology of PFC, VGE and DCI models in rabbits and then in swine were utilized. In these experiments, animals were highly instrumented and mechanically ventilated. The respiratory exhaled gases were continuously monitored with a mass spectrometer recording breath by breath end-tidal N_2 concentrations. In the rabbit study with PFC pre-treatment and VGE carried out in exactly the same manner as the work from the early 1980s, those animals that had PFC introduced into their circulation had a higher peak exhaled N_2 and a quicker return to baseline (no detectable N_2, Figure 1).[30] It is interesting to note that, with PFC present, prior to femoral vein air embolism, there exists a small but noticeable amount of N_2 coming out of the animal. This may be due to enhanced N_2 tissue washout because the PFC is stored in its vials with argon gas, not N_2. The conclusion was that PFC partially increased the speed of N_2 elimination through the lungs.

In a swine DCI project, highly instrumented anesthetized, spontaneously breathing animals were dry dived to 608 kPa (6 bar) for 30 minutes bottom time.[31] On surfacing, severe cardiopulmonary DCI developed. Animals were treated with PFC immediately upon removal from the dive chamber and then underwent controlled ventilation with 100% O_2. Although outwardly this study had some similarities to the earlier US Navy work (swine and treatment at surface), it had major differences (spontaneous versus controlled ventilation, anesthetized versus awake, and fully instrumented) and was pursued in an attempt to further understand the physiology of PFC in DCI. Those animals which received PFC had a slightly faster N_2 washout. At necropsy, most of the animals had large bubble loads still present in their pulmonary arteries and right heart, suggesting that even though the mass spectrometer had detected washout of N_2 DCI was very much present.[30] The swine study showed that the creation of bubbles in the venous circulation was greatly reduced when PFC was present

Figure 1
End-tidal N_2 (mmHg) over time for each breath with and without infused PFC after experimental venous air embolism[30] (with permission)

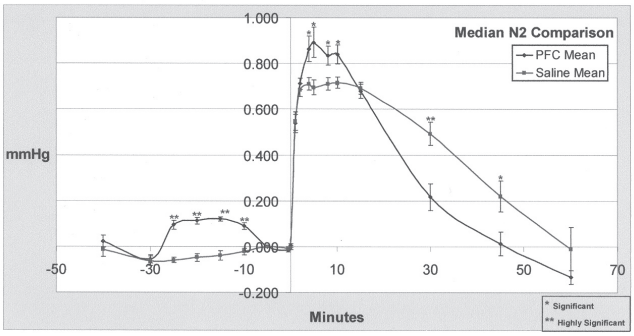
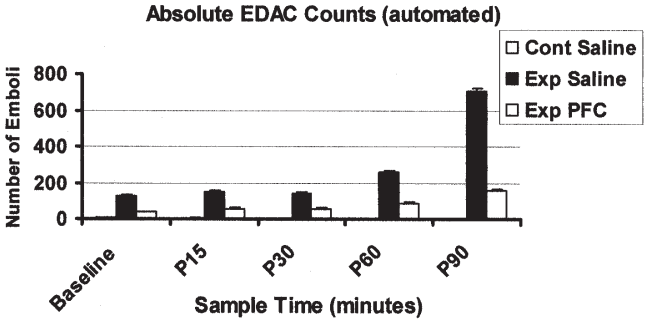


Figure 2
The automated embolic counts per minute in the internal jugular vein of swine[31] (with permission)



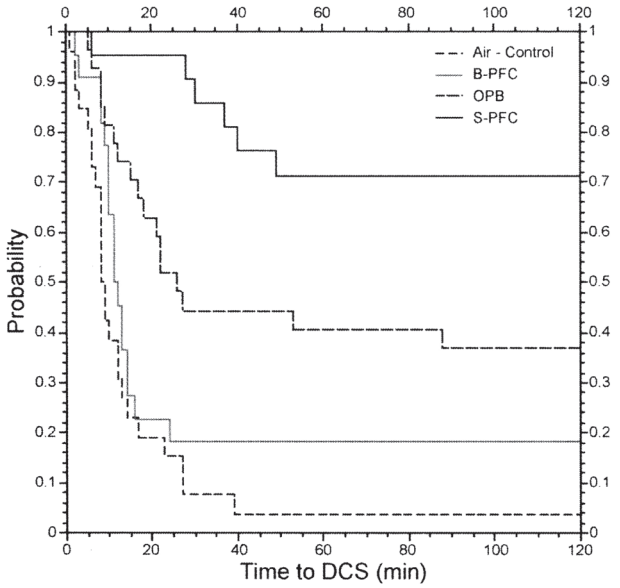
(Figure 2). Control saline is simply a measure of background noise, in that these animals did not undergo DCI. A new bubble-counting technology (EDAC, Luna Technologies, Hampton Roads, VA, USA) had been applied to the internal jugular vein. Using active sonar, this technology counts and can potentially characterize bubble size as emboli move past the sensor. This provides more quantitative information than standard ultrasound detectors. So, although both mass spectroscopy and the bubble-counting device showed promise in terms of the physiologic effects of PFC to blunt the response, the cardiac output and pulmonary artery pressures rose in the animals that received PFC. Swine have a species-specific pulmonary hypertensive response to the micro-particles of the PFC emulsion. This had been well described elsewhere but considerably confused the data. Even with that effect, there was a positive survival effect of PFC as has been the case in all the other, both large and small, animal studies.

Because of the pulmonary vascular effect in swine, the next series of experiments switched to sheep, as they had little or certainly much less of a pulmonary vascular hypertensive response. Again, changes in exhaled N_2 were seen, but more important seemed to be the effects of whole-body O_2 delivery and utilization. Although N_2 washout curves were similar, the plateau that the animals reached showed more total N_2 removed in the PFC group. In animals that received the intravenous emulsion and breathed a helium-O_2 mixture, there was a slightly increased removal of N_2. The most important finding of this work was not the amount or speed of N_2 washout, but that animals with PFC had an increased O_2 delivery to tissues and an increased O_2 utilization.[32] The conclusion was that there is a combined effect of PFC in DCI, both decreasing bubble effects and, perhaps more importantly, increasing O_2 delivery to tissues at risk for ischemia.[32]

The US Navy laboratories have continued working with their awake-swine, saturation-dive model of DCI. In an effort to 'prevent' DCI, PFC was given at depth ten minutes prior to surfacing.[33] Results for PFC given at depth were compared to those with PFC given on surfacing, as well as for a 10-minute period of 100% O_2 pre-breathing prior to rapid surfacing (Figure 3) . Both O_2 pre-breathing and PFC infusion at depth were better than no treatment, but the best was treatment at the surface with PFC and 100% O_2. What was missing from this study was a combination of O_2 pre-breathing and PFC at depth as well as O_2 breathing during the surfacing time period.

Breathing a high O_2 partial pressure combined with an enhanced O_2-carrying capacity and tissue delivery of O_2 carries the possibility of seizures at depth. It appears that concern is warranted. When the same group investigated grand mal seizure activity at 507 kPa (5 bar) with control versus saline infusion versus PFC infusion, they noted 0/26 seizures in the controls, 1/16 in the saline-infusion group and 7/16 in the PFC-infusion group.[34] In this study, all animals

Figure 3
The Kaplan-Meier curves for development of decompression sickness[33] (with permission)



breathed an enhanced O_2 mixture (46% N_2, 54% O_2). Even with these problems, there was no increase in death rate due to PFC. Indeed, PFC animals had the best survival statistics. Does this mean that DCI cannot be prevented with PFC at depth? To date, there has been no trial using other gas mixtures at depth with controlled levels of O_2, and perhaps helium as an inert gas for pre-breathing prior to assent along with PFC infusion. Also, it cannot be assumed that DCI victims who have previously received PFC infusions could not be recompressed.

Future research directions

Some key pieces of information remain to be investigated before PFC infusion, in conjunction with high FiO_2 breathing, can be recommended as an adjunctive or non-recompression treatment for DCI. If PFC were to be utilized, it is most likely that it would be utilized first in a group of victims who have a delayed or prolonged travel time to definitive recompressive therapy. That would make sense, in that the PFC treatment is portable and easy to administer, as is O_2 therapy. However, a major question remains in terms of how long after symptoms develop does PFC therapy retain efficacy? That question is now being investigated by the US Navy and our laboratory. If, for example, spinal cord AGE was encountered and symptoms had been present for 12–24 hours, would PFC infusion still be able to provide any central neurologic salvage? If a victim receives PFC and then either does not have complete resolution of their symptoms or develops symptoms later, could they safely be recompressed using a standard 284 kPa compression dive table? This question, and any increased risk in seizure activity, is presently being investigated.

A major challenge to regulatory approval of PFC therapy for DCI is the fact that it is essentially unethical to create experimental DCI in humans for the purpose of a randomized treatment trial. The groups working in this area have held discussions with the US FDA to invoke a 'two-animal rule' for approval of a new drug indication. This rule allows for proving efficacy with more than one species using the prescribed treatment, i.e., PFC intravenous infusion at the surface with enhanced O_2 breathing. Still, safety needs to be proven. If a DCI indication is allowed by regulatory agencies, the safety of the drug infusion will have to be proven both in human volunteers (divers at surface without DCI) and other human trials (traumatic brain injury trials, for example, wherein PFC treatment is underway).

Conclusions

There is considerable literature and scientific support for the use of a PFC intravenous infusion in conjunction with 100% FiO_2 breathing to treat/prevent DCI. Although the work has spanned nearly 30 years of animal experimentation, no human has yet to be treated for DCI with PFC. To do so safely today remains as yet unethical, since we do not know the consequences of such treatment in terms of limiting standard treatment options. However, groups are working today on answering the remaining questions and the data from such studies look promising in terms of being able to recompress victims without undue risk of seizures. Perhaps in the not too distant future, PFC infusions could be utilized at the site of first contact/rescue when an intravenous line is placed to begin treatment prior to transport and recompression therapy.

It may well be possible and ethically appropriate to design a human trial of such therapy in a sub-group of patients for whom lengthy transport or delays to treatment are expected. Only once more is known about recompression with PFC circulating will it be possible to push the possibilities of human trials and/or change recompression tables depending upon N_2 off-gassing and alternative gas-mixture breathing. One motivation for developing PFC treatment of DCI is disabled submarine rescue, wherein a potential mass casualty event with DCI is at least a theoretical fear. PFC appears promising as a therapeutic option in such a disaster scenario, wherein it would be logistically difficult to have enough chambers and trained personnel on site at a DISSUB rescue mission. This is most likely to happen in remote parts of the world with politically difficult or hostile environments meaning that quick movement of medical teams and rescue vehicles into position may be difficult or impossible. Therefore PFC as a pre-treatment or early intervention at 101.3 kPa appears, at least scientifically, at this point to hold great promise.

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Technical report

A novel wearable apnea dive computer for continuous plethysmographic monitoring of oxygen saturation and heart rate

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Key words
Diving, breath-hold diving, transcutaneous oximetry, hypoxia, diving research, physiology

Abstract

(Kuch B, Koss B, Dujic Z, Buttazzo G, Sieber A. A novel wearable apnea dive computer for continuous plethysmographic monitoring of oxygen saturation and heart rate. *Diving and Hyperbaric Medicine*. 2010;40(1):34-40.)
We describe the development of a novel, wrist-mounted apnea dive computer. The device is able to measure and display transcutaneous oxygen saturation, heart rate, plethysmographic pulse waveform, depth, time and temperature during breath-hold dives. All measurements are stored in an external memory chip. The data-processing software reads from the chip and writes the processed data into a comma-separated-values file that can be analysed by applications such as Microsoft Excel™ or Open Office™. The housing is waterproof and pressure-resistant to more than 2.026 MPa (20 bar) (breath-hold divers have already exceeded 200 metres' sea water depth). It is compact, lightweight, has low power requirements and is easy to use.

Introduction

Medical concerns about professional and recreational diving safety stem, in part, from lack of field studies monitoring physiological parameters. This deficiency is primarily because of the lack of instrumentation suitable for underwater measurements of simple but important parameters such as heart rate and arterial blood pressure. With the lack of direct measurements, results from 'models' of underwater diving as well as inferences from the clinical world are commonly adopted in diving medicine. Unfortunately, both processes are intrinsically uncertain and may not be scientifically valid. Thus, the transfer to the underwater environment of routine clinical instrumentation would represent a useful advance, just as it has in space medicine. This task requires the development of novel underwater diagnostic and monitoring instrumentation as well as the elaboration of ad hoc support infrastructure.

The physiological signals that can currently be measured underwater are: heart electrical activity by continuous electrocardiogram (ECG), heart anatomy and function by echocardiography, blood pressure by sphygmomanometer and transcutaneous oxygen saturation ($S_{tc}O_2$).[1–7] However, with the underwater instrumentation currently available, the diver is unable to utilize directly and in real time the information on his physiological status, as the acquisition and communication of physiological data requires the use of more or less complicated devices that are operator-mediated.

The basic idea of the present work was to develop a convenient, small user-friendly apnea diving computer (ADC), which is able to:

- provide continuous recording of two vital parameters such as $S_{tc}O_2$ and heart rate together with water temperature and hydrostatic pressure;
- display the information in real time on a graphical display; and
- store data for at least four hours.

Originality of the device stems from the use of a single sensor for the detection of both $S_{tc}O_2$ and heart rate, which at the same time overcomes the important technical limitations of current fingertip oximeters and continuous underwater ECG recording. Existing finger plethysmographic devices have serious limitations to their underwater use and the recording of the ECG is challenging because of the difficulty encountered in the electrical insulation of the electrodes and the dimensions of the recorder. Without suitable electrical insulation, ECG signal recording on an immersed body is difficult (fresh water) or impossible (sea water), even if the diver uses a neoprene wetsuit, and the recorder is too bulky and uncomfortable to be portable underwater.

Clinical $S_{tc}O_2$ meters (pulse oximeters) are generally based on measurement of the absorption of transmitted light at specific red and near-infra-red wavelengths. The transducer probe is usually placed on the ear lobe or on the finger tip.[8,9] Measurement of $S_{tc}O_2$ in divers has been attempted using standard, finger-transmission pulse oximetry.[10] However, this approach did not produce reliable results probably because of the peripheral vasoconstriction associated with the diving reflex, which is further enhanced in cold water, reducing finger blood flow and preventing correct estimation of $S_{tc}O_2$.

An alternative to transmission pulse oximetry is reflectance pulse oximetry. Using this approach, the light transmitter and receiver are situated a short distance from each other (around 8 mm) in the same probe. Light is transmitted into the underlying tissue and the reflected light is received and measured. In this situation the spectral intensity of the reflected light depends on the O_2 saturation of the arterial blood in the underlying tissues. Importantly, a reflectance transducer probe can be placed on any part of the body surface, in particular on the temple or forehead, a region less affected by vasoconstriction and consequent blood hypoperfusion compared to the fingertip. Additionally it can easily be protected from cold water (e.g., the glabellum or temple by the diving mask). As the pulse oximeter provides a pulsatile waveform synchronous with cardiac contraction, intervals equivalent to the ECG RR interval could possibly be estimated.

Reflectance pulse oximetry probes placed on the forehead were shown to have acceptable agreement with transmittance probes for pulse oximetry within a typical range of $S_{tc}O_2$ in patients undergoing peripheral vascular surgery.[11] $S_{tc}O_2$ values from these methods were compared with oxygen saturation (S_aO_2) measurements of simultaneously collected arterial blood, and S_aO_2 closely matched both $S_{tc}O_2$ probe values. Similar conclusions were reached recently for the use of forehead reflectance oximetry probes versus conventional digit sensors in paediatric patients.[12] The utility of reflectance pulse oximetry beyond the simple measurement of arterial oxygen saturation from the finger or earlobe was recently expanded for use at internal sites such as the oesophagus and bowel; analysis of the photoplethysmographic waveforms produced by these sensors proved useful in providing new physiological data.[13]

Methods

HARDWARE

The core component of the ADC is a low-power 8-Bit RISC microprocessor (Atmega644p, Atmel) with the following specifications:

- 64 kbytes Flash Program Memory
- 4 kbyte SRAM
- 2 kbyte EEPROM
- 8 MIPS @ 8 MHz

The Atmega644p is operated at 7.3 MHz (internal clock). The real-time clock is based on a 32.768 KHz crystal. A combined digital 16-bit temperature/pressure sensor (MS5541B, Intersema) is integrated in the design for depth measurement. It is specified for a maximum pressure of 14 bar (1 bar = 101.3 kPa). In the range 0–5 bar it has an accuracy of +/-20 mbar. A 128x64 matrix display (EA DOG-M, Electronic Assembly) is used to visualize all dive-relevant parameters plus the plethysmography waveform, heart rate and $S_{tc}O_2$. The dive profile and plethysmography waveform are continuously tracked and stored in an external 32-Mbit memory chip (AT25DF321, Atmel), which allows continuous recording and storage of five hours' data (with a plethysmographic sampling frequency of 75 Hz). The pressure sensor, display and external memory are connected to the serial peripheral interface (SPI) of the microcontroller; the SPI is a bus system for serial synchronous data transmission.

In order to provide the $S_{tc}O_2$ signal and heart rate, a commercial pulse oximeter module (OEM III, Nonin) is used. A reflectance probe (8000R, Nonin) was chosen that can be placed on the forehead or on the temple ($S_{tc}O_2$ accuracy +/-3 per cent saturation; heart rate accuracy +/-3 beats per min). It is interfaced to the microcontroller via the universal asynchronous receiver/transmitter (USART1) at 9,600 bits per second.

Two piezo-buttons allow user input. They are connected to the external interrupt INT0. PC communication is done via a serial interface to USB converter (TTL-232, FTDI). The interaction of all hardware parts is shown in Figure 1. The overall low power consumption and the integrated step-up converter (MAX1724, Maxim) allow powering of the whole ADC via a single 1.5V AAA battery.

SOFTWARE

The firmware of the device was developed in the programming language C. As Integrated Development Environment, the IAR Embedded Workbench (IAR Systems) was chosen. It is a set of development tools for building and debugging embedded applications using assembler, C and C++ in Windows 9x/NT/2000/XP/Vista™ environments. The firmware rests upon two major parts. The first part is devoted to measurements, and data pre-processing for storage, and their display on the computer screen. The second part stores all the measurements into the external memory chip.

Continuous tracking of the physiological condition of the diver (including plethysmography) plus parsing, measuring

Figure 1
Hardware diagram for the apnea dive computer

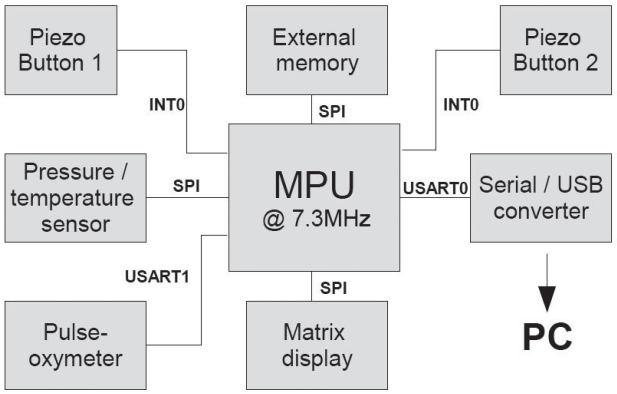
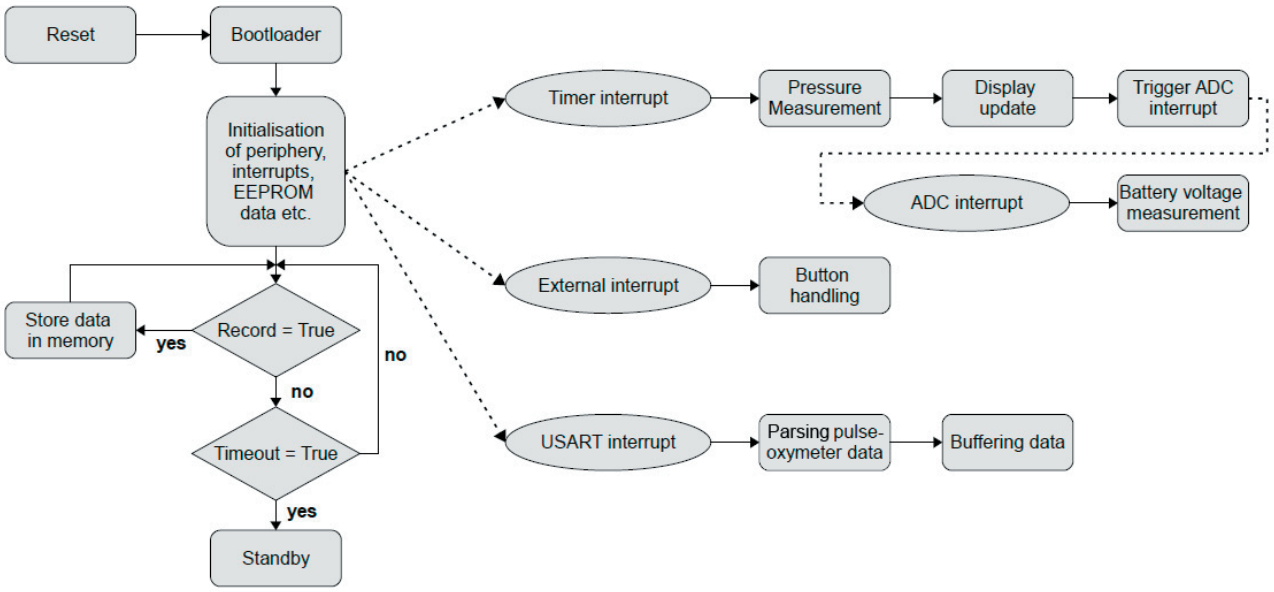


Figure 2
Programme scheduling in the apnea diving computer



and displaying data is time-intensive on a low-power 8-bit microcontroller. Thus a pre-emptive scheduling algorithm with fixed priorities is implemented, which controls everything quasi in parallel. The Nonin OEM III pulse oximeter provides heart rate and $S_{tc}O_2$ (4-beat average values) and the plethysmographic waveform in 25 data blocks, 3 times per second via USART1. Each block has a size of 5 bytes. These data are parsed and stored in a ring buffer whenever an USART1 interrupt occurs. In addition, every 250 ms, depth and temperature are measured and displayed together with $S_{tc}O_2$, heart rate, time and the plethysmographic waveform. To achieve precise timing, this is done using the real-time clock of the Atmega and the timer interrupt. Additionally the battery voltage is measured using the internal analogue-to-digital converter. To prevent data packet loss during USART1 communication, the USART1 interrupt has a higher priority than the timer interrupt. The main loop has lowest priority; it reads the ring buffer and stores its data together with depth, temperature and time into the external memory. The whole programme flow is detailed in Figure 2.

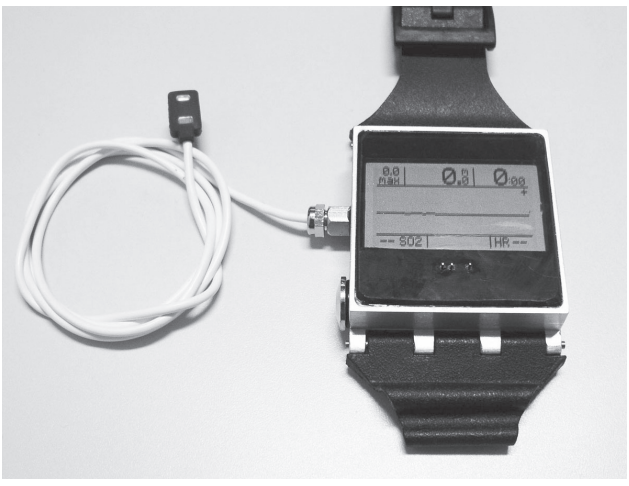
DATA PROCESSING

For visualization and analysis of the recorded data, software was developed under the Eclipse SDK 3.4.1 in Java 1.6 and the Standard Widget Toolkit. The Standard Widget Toolkit is an open-source widget toolkit for Java that provides efficient, portable access to the user-interface facilities of the operating systems. For serial communication with the diving computer, RXTX was chosen. It is a native library, which provides serial and parallel communication for the Java Development Toolkit under the GNU LGPL license. The software reads out the external memory of the ADC at 230400 Baud.s-1 and stores the data in one comma-separated-values (CSV) file per dive. Thus, data can be easily analysed within arbitrary applications like Microsoft Excel™ or Open Office™. After successful data transmission, the memory of the ADC can be erased.

PROTOTYPE

The prototype ADC is wrist-mounted in a square housing measuring 60x60x25 mm (similar in size to some wrist-mounted decompression computers, Figure 3). A single 1.5V AAA battery serves as power supply. The overall power consumption is 60 mA at 7.3 MHz system clock. In sleep mode, the power consumption is reduced to 70 µA. Instead of developing a water- and pressure-proof housing, the internal space of the device is simply encapsulated in

Figure 3
The prototype wrist-mounted apnea diving computer



silicone gel (SilGel 612, Wacker Chemie AG). Further, this measure allows installation of the digital pressure sensor directly on the electronic board, as the ambient pressure is transduced via the soft silicone gel to the membrane of the pressure sensor. Only the battery is housed in a water- and pressure-proof compartment.

VALIDATION AND TESTING

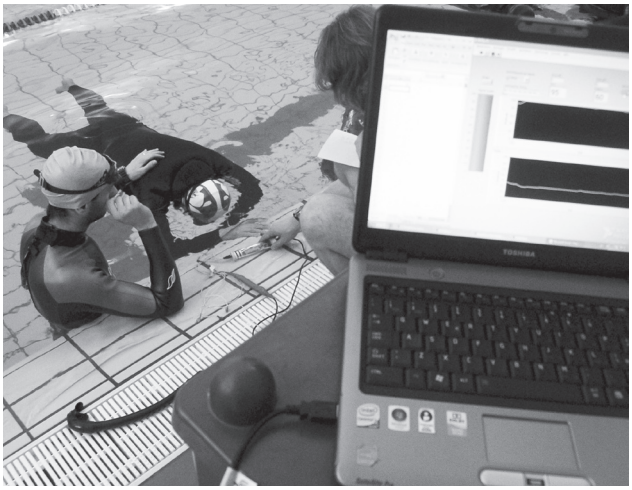
In the first stage of testing, the performance of the ADC was studied on five volunteer apnea divers (height 160–185 cm; weight 50–80 kg; age 27–35 yo) in a swimming pool. All gave written, informed consent for the study, which was approved by the local ethical committee. Measurements obtained with the ADC were compared with measurements from a reference pulse oximeter (ChipOx, Weinmann Medical Technology; accuracy +/- 3 per cent saturation over the physiological range of $S_{tc}O_2$ and +/- 3 beats per min heart rate). Since the reference pulse oximeter uses a transmission sensor on the finger, this is affected by vasoconstriction, and so comparisons were made in warm water with the diver wearing a thin wetsuit. Since the fingertip sensor is only pressure- and water-resistant to a maximum of 1 metre fresh water (mfw), comparative tests were performed during static apnea at the edge of the pool. For direct comparison of the two devices, they were connected to a poolside computer via a USB port and analysed in parallel using LabView software developed ad hoc with a 1 Hz sampling rate.

The ADC was first tested with the subject breath-holding whilst seated poolside, with the two sensors placed on a finger each; the aim of this test was to assess whether the ADC delivered values comparable to those from the reference transducer. Then, with the reference transducer finger-mounted and the ADC placed on the temple, the diver breath-held whilst floating horizontally in the water with the face immersed (Figure 4). During the tests, water temperature was 26°C and air temperature 30°C.

In the second phase, which was carried out in a 10.5 mfw deep research pool, one elite breath-hold diver (male, height 160 cm, weight 55 kg, age 26 yo) and one untrained subject (male, height 185 cm, weight 75 kg, age 28 yo) who nevertheless was able to sustain breath-holds for more than two minutes, were studied. The ADC probe was fixed on the temple with tape, and kept in position by the hood of the diving suit. This arrangement resulted in a clear signal in all cases. Divers performed four breath-hold dives to 1.5 or 10 mfw depth at various times of the day.

Statistical analysis of the collected data was not performed, since it has already been shown that reflectance pulse oximetry probes placed on the forehead have acceptable agreement with transmittance probes for pulse oximetry within typical ranges of $S_{tc}O_2$.[11] Both pulse oximeters used in this study are certified OEM products, and should work accurately according to their specifications. The aim of the current paper is to present the developed system and sample measurement data; a physiological study on apnea divers is ongoing and detailed results, including statistical analysis, are expected later in 2010.

Figure 4
A subject performing an immersion apnea whilst being monitored with the apnea dive computer



Results

In all the tests, the plethysmographic signal was visualized and heart rate and $S_{tc}O_2$ detected. Table 1 and Figure 5 show $S_{tc}O_2$ values obtained with the ADC and the reference transducer during dry apneas with both sensors placed on the finger. The mean differences in $S_{tc}O_2$ between the reference pulse oximeter and the ADC were less than the quoted accuracies of the two devices.

The results during immersed static apnea were similar, except that at the end of the apnea phase, the fall in $S_{tc}O_2$ recorded with the ADC probe on the temple was greater than that recorded by the reference transducer and recovered slightly more slowly (Table 2 and Figure 6).

In phase two, $S_{tc}O_2$ and heart-rate values were successfully obtained during repeated breath-hold dives at 1.5 or 10 mfw in both divers. Table 3 shows that the fall in $S_{tc}O_2$ was more marked in the untrained diver compared to the elite diver despite apneas of shorter duration. In a typical dive to 10 mfw depth (Figure 7), $S_{tc}O_2$ remained constant up to approximately two minutes of apnea and then started to drop slightly. During the ascent desaturation accelerates rapidly and $S_{tc}O_2$ reached the lowest value (nearly 70%) upon surfacing.

Discussion

This study describes the research and development of a novel wrist-mounted apnea dive computer (ADC), able to provide continuous measurement of $S_{tc}O_2$, heart rate and plethysmographic pulse waveform, water temperature

Table 1
Comparison of the simultaneous measurements of $S_{tc}O_2$ using the apnea dive computer and a reference plethysmograph during dry land breath-holds

Diver	Sex	Apnea time (min)	max $S_{tc}O_2$ Ref	max $S_{tc}O_2$ ADC	min $S_{tc}O_2$ Ref	min $S_{tc}O_2$ ADC	Diff min/max Ref	Diff min/max ADC	Mean diff Ref/ADC
1	m	02:30	98	100	79	81	19	19	3.2
2	f	03:03	99	99	64	66	35	33	1.9
3	m	02:45	99	99	81	79	18	20	2.3
4	m	02:57	100	98	78	82	22	16	3.8
5	m	03:22	97	97	69	71	28	26	3.5

Table 2
Comparison of the simultaneous measurements of $S_{tc}O_2$ using the apnea dive computer and a reference plethysmograph during immersed static apnea with face immersed

Diver	Sex	Apnea time (min)	max $S_{tc}O_2$ Ref	max $S_{tc}O_2$ ADC	min $S_{tc}O_2$ Ref	min $S_{tc}O_2$ ADC	Diff min/max Ref	Diff min/max ADC	Mean diff Ref/ADC
1	m	04:15	100	100	82	63	18	37	3.7
2	f	03:23	100	100	66	53	34	47	6.1
3	m	03:48	98	100	81	69	17	31	4.9
4	m	04:20	100	100	79	65	21	35	4.5
5	m	04:35	100	99	72	59	28	40	5.1

and depth in a simple, user-friendly way. Oxygen is the most essential element to life, its lack having immediate consequences, particularly on central nervous system function. Measurement of $S_{tc}O_2$ is of special interest in breath-hold diving. Under normal conditions, more than 98% of the O_2 in arterial blood is bound to haemoglobin (Hb), the remaining 2% is dissolved in plasma. At a normal arterial pO_2 of 13 kPa, Hb-O_2 saturation is about 97.5%, while in mixed-venous blood, the pO_2 drops to 5 kPa and Hb-O_2 saturation is approximately 75%. Thus, during apnea, $S_{tc}O_2$ will reflect the amount of O_2 that is delivered to tissues and, in turn, $S_{tc}O_2$ depends on the partial pressure of O_2 in the alveoli.[14]

In the dive shown in Figure 7, the rapid blood desaturation during the ascent is because of the rapid decrease in pO_2 in the alveolar space due to the reduction of environmental

Figure 5
Simultaneous recordings of $S_{tc}O_2$ during a dry static apnea using the apnea dive computer and the reference plethysmograph each on a finger; vertical lines delineate the apnea (02:45 min)

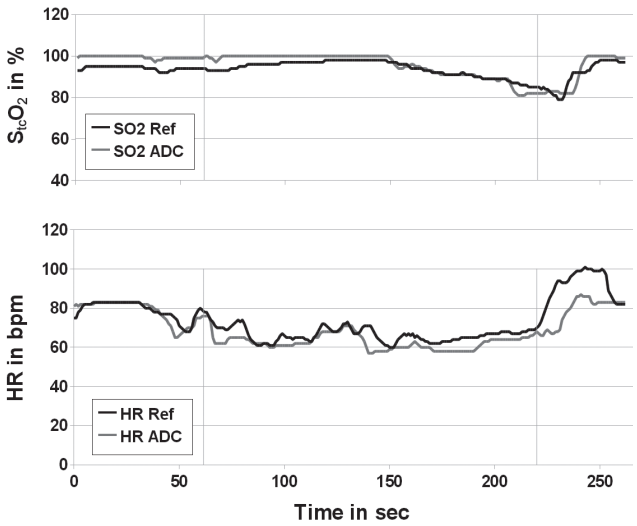


Figure 6
Simultaneous recordings of $S_{tc}O_2$ in the same diver as in Figure 5 during an immersion static apnea with face immersed using the apnea dive computer on the forehead and the reference plethysmograph on a finger; vertical lines delineate the apnea (04:15 min)

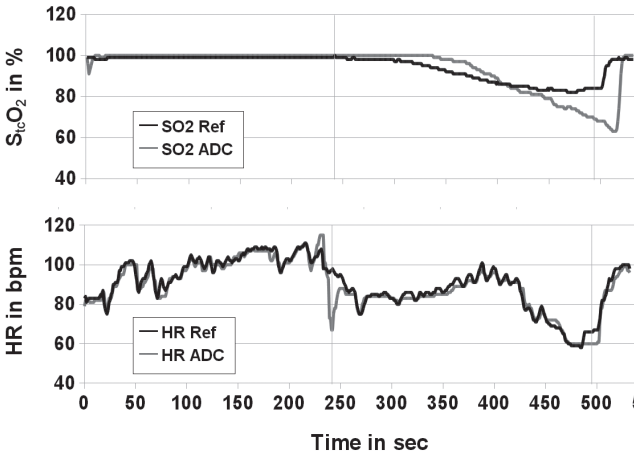


Table 3
$S_{tc}O_2$ measured during repeated dives to 1.5 mfw and 10 mfw depth performed by two divers, one elite and the other untrained in breath-hold diving

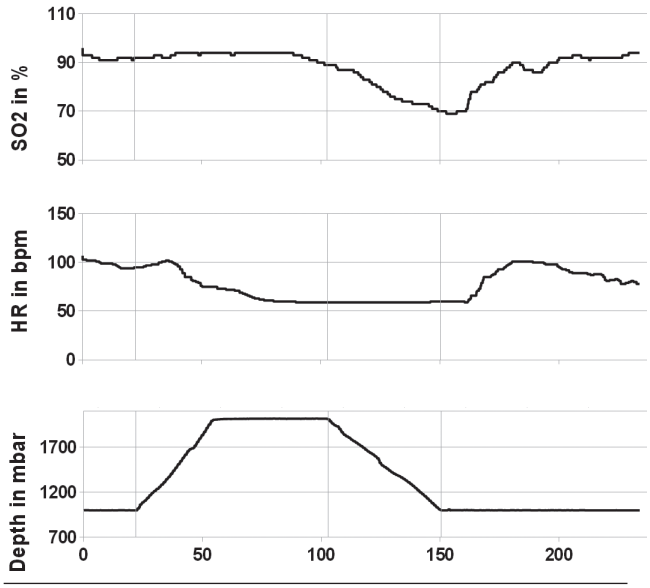
Apnea (min)	Depth (mfw)	Time	min $S_{tc}O_2$	max $S_{tc}O_2$	min Heart rate	max Heart rate
Elite						
02:30	1.5	10:20	96	100	44	91
03:01	1.5	10:40	95	100	45	93
03:12	1.5	13:30	87	100	41	83
02:47	1.5	14:00	85	100	42	85
02:20	10	09:00	95	100	44	96
02:40	10	09:20	96	100	42	96
02:43	10	09:40	97	100	44	97
02:55	10	11:20	95	100	41	95
Untrained						
02:44	1.5	09:42	78	98	54	98
02:45	1.5	10:16	79	99	55	87
03:04	1.5	10:40	69	96	52	96
02:33	1.5	11:18	70	98	55	83
02:07	10	14:36	84	98	70	114
02:29	10	14:58	73	98	58	106
02:13	10	15:30	69	97	59	105
02:34	10	16:37	77	96	58	119

pressure from 2 to 1 bar. This condition is opposite to the descent, when pO_2 progressively increases according to the increase in environmental pressure thus maintaining a constant $S_{tc}O_2$ in spite of continuous oxygen consumption. The fall in $S_{tc}O_2$ during the ascent and its continued drop after surfacing explains why ascent syncope occurs at or near the surface.[15] Continuous monitoring of the $S_{tc}O_2$ screen could help the diver to be more aware of his own limits and provide him with an objective warning of dangerous conditions. In addition, monitoring of $S_{tc}O_2$ during apnea would contribute to a better understanding of underwater physiology. To reach these goals, however, the accuracy of the measurements has to be validated in open-water studies.

In the present work, we utilized a new approach, the use of a reflectance oximetry probe, which can be positioned on a skin region less influenced by vasoconstriction than a digit, and is easily protected from cold water. Preliminary tests of the ADC probe against a reference, finger pulse oximeter showed equivalence of signal during dry apneas but an underestimation of $S_{tc}O_2$ drop in the immersed subject at the end of apnea probably related to vasoconstriction of the finger compared to the temple. A second advance is the utilization of the same pulse oximeter signal for monitoring heart rate. This approach bypasses the difficulties encountered in ECG recording in sea water, i.e., electrical insulation, and makes the device as small and user-friendly as possible.

Although pulse oximetry is used widely to monitor blood oxygenation, it cannot normally determine oxygen

Figure 7
Record of $S_{tc}O_2$, heart rate and depth during a 10 mfw dive; vertical lines delineate the apnea and the start of the ascent



consumption. However, in the context of apnea, the sudden and complete interruption of the external O_2 supply leads to a fall in $S_{tc}O_2$ once O_2 consumption has exceeded the initial body stores of oxygen.

The simultaneous display of $S_{tc}O_2$, heart rate and depth profiles allows an accurate analysis of time relationships between physical and physiological parameters. As an example, immersion bradycardia may vary in different subjects depending on pre-dive preparations used by the diver, such as hyperventilation and/or lung packing to modify body O_2 storage.

The main limitation of the present study is the small number of subjects studied. However, the principal objective was to document the technical feasibility and reliability of the new device and its applicability to field studies. From these preliminary results, the device appears capable of providing new information on diving physiology and potentially enhancing diver safety. Further studies on larger cohorts of divers and exploration of a wider range of depths and conditions, especially the impact of thermal (cold) stress, are needed before the performance of the ADC is fully validated. Such studies are currently ongoing. Future studies could also address detailed analysis of the plethysmographic waveform, for instance, to investigate heart-rate variability during diving. However, advanced signal analysis will be required, as the rounded peaks in the plethysmographic waveform are not as clear cut as the R-wave of the ECG.

Conclusion

We present a novel, wrist-mounted apnea dive computer (ADC) capable of measuring and displaying $S_{tc}O_2$, heart rate,

the plethysmographic waveform, depth, water temperature and time during breath-hold dives. The measured data are stored in a memory chip, which is read by data-processing software and the processed data are written into a CSV file, for analysis by applications such as Microsoft Excel™ or Open Office Calc™.

Preliminary results give us confidence that the ADC has the potential to provide continuous monitoring of $S_{tc}O_2$ and heart rate for long periods.. Together with depth and temperature monitoring, these measurements may contribute to a better understanding of the complex relationships between physiological cardio-respiratory parameters over time during dives, and to the possible definition of objective criteria for fitness for breath-hold diving, based on simple underwater testing.

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Conflicts of interest: none

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The world as it is

Future synergism in diving accident management: the Singapore model

Chong Si Jack, Liang Weihao, Kim Soo Joang and Kang Wee Lee

Key words
Hyperbaric facilities, diving accidents, hyperbaric oxygen therapy, treatment, research, general interest

Abstract

(Chong SJ, Liang W, Kim SJ, Kang WL. Future synergism in diving accident management: the Singapore model. *Diving and Hyperbaric Medicine*. 2010;40(1):41-3.)
With the rise in popularity of recreational diving in recent years comes increasing numbers and risk of diving-related injuries and demand for professional medical treatment of such injuries. Concurrently, hyperbaric oxygen therapy (HBOT) has become more readily available for the treatment of various medical conditions. In Singapore, diving and hyperbaric medicine was largely a military medicine specialty confined to the Singapore Armed Forces for many years. The new Hyperbaric and Diving Medicine Centre set up in Singapore General Hospital (SGH) offers an excellent opportunity for collaboration between the Singapore Navy Medical Service (NMS) and SGH. This combines the expertise in the field of diving and hyperbaric medicine that NMS provides, with the resources and specialized services available at SGH. This collaboration was formalized by the recent signing of a Memorandum of Understanding between the two organisations. The partnership will allow both organisations to build on each other's strengths and enhance the development of research and training capabilities. This collaboration will also be an important step towards formal recognition and accreditation of diving and hyperbaric medicine as a medical subspecialty in the foreseeable future, thus helping its development in Singapore. This synergistic approach in diving accident management will also promote Singapore as a leader in the field of diving and hyperbaric medicine in the region.

Introduction

The popularity of recreational diving has increased in recent years in the Asian region. With the rise of this sport inevitably comes increasing numbers and risk of diving-related injuries and demand for professional medical treatment of such injuries. Concurrently, HBOT is increasingly being accepted for certain medical conditions and there has been an expansion of hyperbaric services in other countries in this region.[1] The new Hyperbaric and Diving Medicine Centre (HDMC) set up in Singapore General Hospital (SGH) offers a unique opportunity for collaboration between the Singapore Navy Medical Service (NMS) and SGH. This combines the expertise in the field of underwater medicine that NMS provides, with the resources and specialized services readily available at SGH.

Aim

The collaboration between the NMS and SGH seeks to achieve the following synergism in diving accident management:

- to streamline and optimise available resources through cooperation rather than competition between the different services, and allow ample resources to be diverted to research;
- to house a wide range of facilities and clinical services together with the hyperbaric chambers at a single location, thereby increasing convenience and reducing need for unnecessary transfers, especially of ill, unstable patients;
- to increase our patient pool and widen our reach through the promotion of our services to local and regional patients;
- to work towards formal recognition and accreditation of diving and hyperbaric medicine as a medical subspecialty in the future;
- to establish Singapore as a subject-matter expert in the fields of diving and hyperbaric medicine in the region, with world-class facilities, services, training and research.

Navy Medical Service (NMS)

In the 1970s, the practice and knowledge of hyperbaric treatment in Singapore was in its infancy. HBOT was predominantly used in the treatment of decompression illness (DCI) in fishermen divers,[2] and infrequently as adjunct treatment for cases of gas gangrene, burns and diabetic ulcers. The highlight in the 1980s for hyperbaric medicine and its specialists in the Republic of Singapore Navy (RSN) was when the nation was building its first subway/train network, the Mass Rapid Transit (MRT).[3] The

project involved extensive underground tunnelling with men working in compressed air environments. The expertise of the NMS in compressed-air works was called upon, and for the four years of the MRT project, diving medicine specialists supervised the construction sites and successfully treated all cases of DCI.

NMS physicians continued to gain exposure in the years after the MRT project and saw an increasing number of DCI cases amongst recreational divers.[4] Clinical hyperbaric medicine cases were also an increasing trend, with non-healing diabetic wounds constituting the majority of the cases. Since then, NMS's expertise in diving and hyperbaric medicine has grown and is now a vital part of the support it provides to the RSN. NMS has also established itself as a subject-matter expert in Singapore and the region through the provision of specialist consultation and treatment services.

To ensure currency and competency, medical officers and senior specialists are regularly enrolled in overseas courses and seminars for training and knowledge-sharing, and the expertise and knowledge they bring upon their return is incorporated into the service.

Singapore General Hospital

In Singapore, military diving contributed to the vast majority of all diving activities in the past. As such, the fields of diving and hyperbaric medicine have largely remained a military medicine specialty and its practice confined to the military until the establishment of the first civilian hyperbaric chambers in 2002. With the advent of diving as a popular leisure sport and an increasing number of applications of HBOT, SGH commissioned the Hyperbaric and Diving Medicine Centre (HDMC) in partnership with Hyperbaric Health (Figures 1 and 2) in 2008, offering HBOT for a variety of conditions including decompression illness, gas embolism, gas gangrene, non-healing wounds and compromised skin flaps and grafts, amongst other indications. This is the first publicly funded multi-place, hyperbaric medical facility to be set up in a tertiary government hospital in Singapore.

The benefit of having the HDMC within the premises of SGH itself is the ready availability of a wide spectrum of clinical services and facilities that SGH can provide to the diving patients. These include ward facilities to provide monitoring and nursing care, an intensive care unit for the severely ill, and operating theatres, as well as specialists in various fields providing round-the-clock, expert medical advice. The presence of a state-of-the-art chamber within the premises of the hospital is invaluable to the treatment of ill patients who may otherwise be too sick to be transferred to a hyperbaric facility at a different location.

Collaboration between NMS and SGH in diving accident management

The relationship between the NMS and SGH was formalized by the signing in October 2008 of a Memorandum of Understanding in areas of clinical practice, research and training. Since then, the two organisations have collaborated, whereby accredited military personnel will manage both military and civilian casualties at the HDMC in SGH. NMS personnel currently provide 24/7 recompression therapy care for all diving casualties. This ensures clinical competency and facilitates management of complex decompression illness cases that may be associated with other injuries, due to the close proximity and support of the full range of clinical services in SGH.

Collaboration in research

One of the potential benefits of the collaboration between NMS and SGH will be to allow more to be channelled towards research and development. An example of research collaboration between the two organisations is a joint, prospective study to evaluate the effects of hyperbaric oxygen therapy on acute thermal burns. Recruitment of study participants began in early 2009.

Figure 1
The Hyperbaric and Diving Medical Centre, Singapore General Hospital

Figure 2
Medical staff at SGH HDMC monitoring a patient during hyperbaric treatment.

Conclusion

In a small country like Singapore, collaboration between different services in providing professional and holistic hyperbaric treatment will serve to benefit all parties involved with better resource utilisation and achieving synergy in diving casualty management, which will benefit our patients. This will likely be our approach to the future development and evolution of the field of diving and hyperbaric medicine in Singapore.

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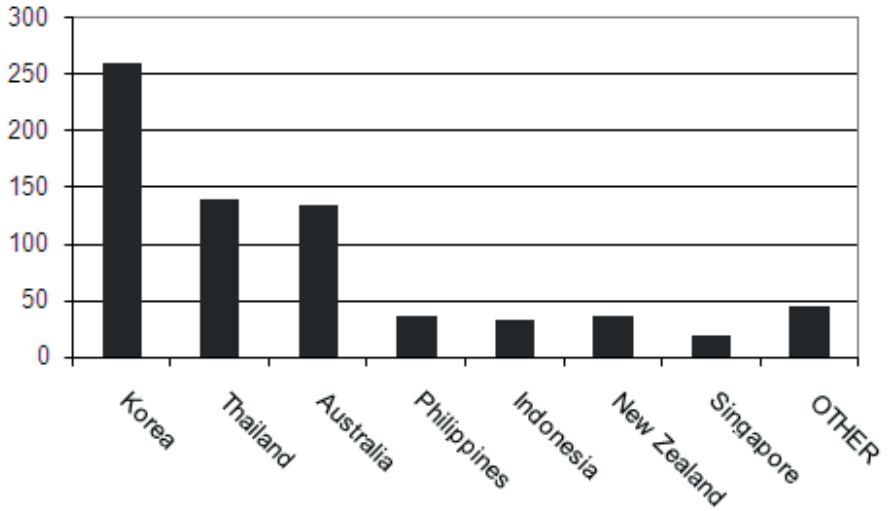
Decompression illness in the Asia-Pacific region in 2008

Below are the data as reported to DAN Asia Pacific for the number of recreational divers treated for decompression illness in the Asia-Pacific region during 2008. In most countries, these numbers appear to be similar to those of the previous year. The number for Thailand is an estimate as data were not provided by all of the chambers. The data from Indonesia do not include cases treated at some of the Indonesian naval facilities, so is likely to be an underestimate. Once again, the data from Korea appear to be disproportionately high and may indicate a safety issue for Korean divers, a low threshold for treatment and/or possibly an issue with reporting.

Reproduced with kind permission from Anon. Decompression illness in the Asia-Pacific in 2008. *Alert Diver Asia Pacific*. 2009;Sept–Dec:9.



Continuing professional development

2010/1 Dysbaric osteonecrosis (DON), decompression illness and the diver

Michael Bennett

Accreditation statement
To complete a course successfully, 80% of questions in each quiz must be answered correctly. Activities published in association with *Diving and Hyperbaric Medicine* are accredited by the Australia and New Zealand College of Anaesthetists Continuing Professional Development Programme for members of the ANZCA Diving and Hyperbaric Medicine Special Interest Group under Learning Projects: Category 2 / Level 2: 2 credits per hour.
Intended audience
The intended audience consists of anaesthetists and other specialists who are members of the ANZCA SIG in Diving and Hyperbaric Medicine. However, all subscribers to DHM may apply to their respective CPD programme coordinator or specialty college for approval of participation.
Objectives
The questions are designed to affirm the takers' knowledge of the topics covered, and participants should be able to evaluate the appropriateness of the clinical information as it applies to the provision of patient care.
Faculty disclosure
Authors of these activities are required to disclose activities and relationships that, if known to others, might be viewed as a conflict of interest. Any such author disclosures will be published with each relevant CPD activity.
Do I have to pay?
All activities are free to subscribers.

Answers should be posted by e-mail to the nominated CPD co-ordinator (for members of both SPUMS and the ANZCA Diving and Hyperbaric Medicine Special Interest Group, this will be Associate Professor Mike Bennett, <M.Bennett@unsw.edu.au>). On submission of your answers, you will receive a set of correct answers with a brief explanation of why each response is correct or incorrect. Successful undertaking of the activity will require a correct response rate of 80% or more. Each task will expire within 24 months of its publication to ensure that additional, more recent data have not superceded the activity.

Key words
MOPS (maintenance of professional standards), diving, scuba, dysbaric osteonecrosis, decompression sickness, decompression illness,

Practitioners are referred to the article in the last issue (Gempp E, Blatteau J-E, Simon O, Stephant E. Musculoskeletal decompression sickness and risk of dysbaric osteonecrosis in recreational divers. *Diving and Hyperbaric Medicine*. 2009; 39(4):200-4.) and the relevant chapter (11.2) in *Bennett and Elliott's physiology and medicine of diving*, 5th edition, for a discussion relevant to the exercise below.

Question 1: Which of the following is not a recognised long-term effect of compressed-gas diving?
A. Juxta-articular sclerotic areas and linear opacities on X-ray
B. A modest increase in vital capacity of the lungs, but not of forced expiratory volume in one second
C. Development of dysbaric osteonecrosis (DON) in divers never treated for decompression illness
D. Neurocognitive deficits
E. Fractures through necrotic subchondral bone

Question 2: Risk factors for DON include
A. A history of frequent long and deep dives, even with the use of good decompression practices
B. A patent foramen ovale
C. Obesity
D. High alcohol intake
E. Decompression illness

Question 3: Differential diagnosis of DON includes
A. Osteoarthritis
B. Avascular necrosis secondary to hypofibrinolysis
C. Bone islands on routine bone survey
D. Paget's disease
E. Decompression illness

Question 4: Concerning the diagnosis of DON, which of the following is the least accurate statement?
A. Stage 3 lesions are often indicated by the onset of pain and dysfunction
B. Stage 1 lesions are only rarely discovered because they are painless, radiologically undetectable and relatively short-lived
C. MRI is the most sensitive diagnostic procedure available after Stage 1
D. Scintigraphy is likely to provide the best evidence of a lesion in Stage 1
E. Stage 4 lesions can be distinguished from other causes of osteoarthritis by observation of marginal osteophytic proliferation

Question 5: Concerning the treatment of DON, which of the following is the least accurate statement?
A. Cessation of diving on first detection may modify the subsequent development of DON lesions
B. Joint replacements are not indicated for DON as continued bone infarction renders them quickly loose and requiring revision
C. Early recompression for musculocutaneous DCS may reduce the risk of subsequent DON
D. Patients with bone infarctions may have an increase in pain on compression
E. No specific therapy that has been shown to improve the prognosis of DON once established

Book reviews

Proceedings of the Fifteenth International Congress on Hyperbaric Medicine

Jordi Desola, editor

Hardcover, 300 pages
ISBN 978-84-612-6672-2
Barcelona: SUB-HELP; 2008
Available from the publisher: <http://www.sub-help.com>
Price: Euro 65 (excluding p&p)

Every three years, the International Congress on Hyperbaric Medicine (ICHM) brings together clinicians and scientists from more diverse geographical and institutional origins than is usual for the various annual society meetings. This results in an eclectic mix of presentation types and subjects that is always a stimulating, if sometimes variable, academic experience. Invariably, some papers are presented on the use of hyperbaric oxygen in conditions not considered standard elsewhere, and some unusual clinical or scientific ideas are explored, which is always a useful intellectual challenge for those whose experience is limited to working within a list of 'accepted indications'.

The 2005 congress was a well-attended and very rewarding meeting held in one of the world's finest conference destinations, Barcelona, Spain. It was organised as a joint ICHM/EUBS meeting and this volume of the proceedings records the majority (76) of the presentations and posters from the joint main meeting.

There is a tradition of the proceedings of each ICHM congress being published in book form and Jordi Desola, President of the 2005 congress, has brought these proceedings to print with the financial support of the international hyperbaric medicine community. The book is in the same size format as all other ICHM proceedings from the eighth meeting on, but it will stand out on the bookshelf as a result of having its covers printed with the eyecatching blue artwork that was the signature of the Barcelona congress.

The format in which the different presentations are printed varies throughout the book, presumably depending upon the material available. In some cases there is detailed text, in others abstracts only, and in many the poster is reproduced, mostly in colour. The colour and print quality is good but the readability of the posters varies, with some authors having apparently submitted low resolution files. Most are readable, however, but a large magnifying glass is an essential item for reading those posters with smaller print.

Proceedings may seem to have limited appeal in the years following, as the more scientifically important papers should be either published in peer-reviewed journals or superseded by new work. The value of ICHM proceedings often lies in material that would not so readily find its way into other types of publication, however. Useful clinical perspectives, ideas for research, rare but important observations, historical origins and truths long-forgotten are included. There is, therefore, much to be gained from periodically re-reading the material published therein and this volume is no exception.

Hyperbaric physicians and hyperbaric facilities will benefit from having the ICHM proceedings sit in their library. Researchers and literature reviewers should also ensure they have access, for ICHM proceedings often contain relevant presentations that do not get re-published and which can be usefully referred to. For those primarily interested in diving medicine and physiology, this particular volume should not be overlooked based upon the "Hyperbaric Medicine" title – about half of the papers are on diving-related subjects, and there is much of interest here.

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Key words
Hyperbaric oxygen, underwater medicine, medical society, meetings, book reviews

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The database of randomised controlled trials in hyperbaric medicine maintained by Dr Michael Bennett and colleagues at the Prince of Wales Hospital Diving and Hyperbaric Medicine Unit is at:

<www.hboevidence.com>

Another Whitstable trade

An illustrated history of helmet diving
John Bevan

Hard cover, 436 pages
ISBN 0-95082242-5-9
Gosport, UK: Submex Ltd; 2009
Available from Submex Ltd: <www.submex.co.uk>
Price: Aus$150.60, £66.50 in UK, £72.00 in Europe
(includes postage and handling)

To those SPUMS and EUBS members with an interest in diving history, John Bevan needs no introduction. Many will have read *The Infernal Diver* and will be familiar with his founding of the Historical Diving Society. Equally, those of us with a military and commercial diving background are familiar with *The Professional Diver's Handbook* and his world record dive in 1970 to 1,500 feet. So, when the press release for *Another Whitstable Trade* arrived in my inbox and the Editor asked if I'd like to review it, I jumped at the chance.

Whereas *The Infernal Diver* was a history of John and Charles Deane's invention of the diving helmet, *Another Whitstable Trade* follows the development of the diving industry that sprang up as a direct result of this invention. The Deane brothers lived in Whitstable, a small town on the southern coast of the Thames estuary, famous for its oyster beds. Given its proximity to London and the many shoals of the Thames estuary, it was only natural that the fledgling diving industry should make its start in Whitstable, dealing with the many wrecks in the area because of the treacherous local waterways. The first section of the book is a chronology of a series of diving 'eras' in Whitstable. At the end of each era, Bevan has added a section on what was happening elsewhere around Britain and overseas.

The second section is a history and description of the major organisations utilising divers. Not surprisingly the military features heavily, with first the Royal Engineers and then the Royal Navy in the UK training and employing divers. However, salvage was one of the main tasks for the early working diver and the Liverpool Salvage Association features in this section along with Trinity House and the Admiralty Salvage Section. Trinity House features heavily in the book, but it is not until halfway through that we get to understand why this is. This may be because John is expecting a mainly English readership; for the non-English amongst us, Trinity House is responsible for the upkeep of all navigation aids in UK waters – buoys, beacons and lighthouses – and removing hazards to navigation. Consequently they were heavy users of the early working divers.

The third section deals with civil engineering applications, rivers, canals, harbours, docks, bridges and tunnels. An excellent way to read this chapter, which deals with some incredible projects, is to have *Google Earth* open on your computer beside you to have an aerial look at the sometimes immense breakwaters constructed largely with manual labour over 120 years ago.

Next, we have a history of the major diving equipment manufacturers. Starting with Augustus Siebe, this equally fascinating section looks at the different companies' individual advances in equipment design, with histories of each company's major personnel. It is interesting to note that despite the number of patents taken out, as soon as one company made an improvement, it was eagerly copied by the rest.

We are then treated to a wonderful look at a selection of notable divers. I am not going to name them here; you need to buy the book. Suffice to say, the exploits of this hardy bunch are hard to believe, especially for someone who has been a working diver. While reading of the globetrotting exploits of these early divers, I was struck by the realisation that, for the commercial diver, little has changed. For today's diver, global travel is a matter of course to chase employment and a project, just as it was in the 1800s. Last but not least is a section on diving physiology and medicine. This is not the sort of chapter you would be used to; rather, it is a look at how little was known in the mid to late 1800s. Divers complained of rheumatism; they died of apoplexy; some were paralysed (literally!) by the cold.

Undertaking the research for this book would have been a Herculean task and, as John himself states, it is bound to be incomplete. However, he is to be commended for the incredible amount of work put into this publication. Beautifully bound, with an embossed dust cover, what really stands out is the large number of quality illustrations dating from the time covered in the book. Tracking down all of those must have been a huge task.

If I have one complaint, it is that the book would have benefitted from a professional proof reader. There are a number of minor mistakes, missing letters, etc, which detracted from my enjoyment of what is otherwise an excellent read. Others less picky than me may not even notice. Having said that, I would heartily recommend this excellent publication to anyone with an interest in diving history. The review copy is number 147 of a first edition of 750, so I would get online at <www.submex.co.uk> and order your copy as soon as possible.

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Key words
History, diving industry, diving at work, military diving, equipment, salvage, book reviews

Commentary

The performance of dive computers at altitude

Following the re-printing last year of the abstract from a paper that appeared in the *International Journal of the Society for Underwater Technology*,[1] the Editor invited me to make some comments on this issue, given the work that our unit has been conducting on decompression computers.

Dive computers measure ambient pressure, which is then converted to depth estimation. The conversion is further influenced by how the pressure sensors are calibrated, the physico-chemical properties of the water in which they are immersed and the barometric pressure. Some of these conversions are standard for all computers, many are not. Ambient water pressure is related to water density which, in turn, is directly related to water salinity. Dive computer pressure sensors are calibrated to one of three main salinities depending on the manufacturer: fresh-, brackish or full seawater. Although a column of 10 metres of standard freshwater may exert a pressure equal to one bar pressure, the column heights would be progressively less for brackish and full seawater in order to exert the same pressure. In addition, many sensors have some form of temperature measurement that is used to modify pressure readings.

The influence of low barometric pressure is increased markedly at high altitudes. Low barometric pressures do not affect the pressure-related effects of diving unduly as pressure measurement is absolute. However, depth estimation will be affected and, at high altitude, a diver will need to dive deeper in the water column to achieve the same depth reading as at sealevel. This effect will be amplified in freshwater if the dive computer pressure sensor is calibrated to brackish or full seawater. Altitude has an obvious effect when the diver surfaces and continues to off-gas at much different pressure gradients to those expected at sea level.

There are many long-established, table-based methods to correct decompression schedules for altitude. Dive computers also have altitude settings, with some models claiming to measure barometric pressure/altitude. In their report, Buzzacott and Ruehle highlight two related facts: that recreational divers predominantly use dive computers to manage their decompression and that, in some parts of the world, there is a significant recreational diving industry based at high altitude. Dive computer manufacturers rarely (if ever) publish the decompression algorithms that underpin the performance of their computers. Allied to this, increasingly the newer breed of computers are employing differing decompression theories with 'in-house' modifications and permitting more user interactions. So, in many cases, physical testing is the only option available to those researching the behaviour of dive computers. Buzzacott and Ruehle test 11 models at altitude (4 fail!) and compare them with sea-level performances. Their conclusion is one of caution, in that the diver at altitude should, if using a computer for decompression management, be aware that corrections for most computers, at present, can only be assumed to be experimental.

Reference

1 Buzzacott PL, Ruehle A. The effects of high altitude on relative performance of dive decompression computers. *International Journal of the Society for Underwater Technology*. 2009;28(2):51-5.

Abstract reprinted in *Diving and Hyperbaric Medicine*. 2009;39(2):87.

The full article is available to download at:
<http://www.ingentaconnect.com/content/sut/unwt/2009/00000028/00000002/art00003>

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Key words
Decompression, computers - diving, altitude, safety, risk assessment

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Link to Doppler study of spirometry in the presence of a patent foramen ovale

Providing the link to the Doppler study[1] reported in the December 2009 issue has proved difficult because of the size of the file. We will attempt to incorporate a link on the SPUMS and EUBS websites in due course, but this may also not be possible.

Reference

1 Maddox IEC, Smart DR, Bishop WLJ. The impact of performing spirometry on shunting across a patent foramen ovale. *Diving and Hyperbaric Medicine*. 2009;39(4):213-5.

Article reprinted from other sources

Utility of regular medical examinations of occupational divers [Abstract]

Sames C, Gorman D, Mitchell SJ, Gamble G

The utility of regular medical fitness-for-diving examinations of occupational divers is unknown. The aim of this audit was to investigate the impact on the employment of occupational divers of a five-yearly medical examination and an annual health surveillance questionnaire administered in intervening years. The medical records of all New Zealand occupational divers registered with the Department of Labour for at least five years were audited (n = 336). Each record included at least two full medical examinations (mean spacing of 5.6 years). An impact on career was defined as the diver being issued with either a conditional certificate of fitness or being graded as temporarily or permanently unfit for diving. The means by which the relevant medical issue was identified was recorded. Ten (3%) of 336 divers had an assessment outcome, that had a career impact. One was considered permanently unfit, four were temporarily unfit, and five were issued with conditional certification. Two were identified by respiratory function testing and eight by way of their responses to the questionnaire; none was found by the medical interview and examination process. The questionnaire system did not 'miss' any divers who developed a critically important health problem, and detected most of those with less important problems. Five-yearly medical examinations have a low detection rate for important health problems, but remain useful for discussion of risk understanding, acceptance and mitigation.

Naval Health Services, Royal New Zealand Navy, Diving Medical Directorate to the New Zealand Department of Labour, and School of Medicine of the University of Auckland, Auckland, New Zealand

Reprinted with kind permission from Sames C, Gorman D, Mitchell SJ, Gamble G. Utility of regular medical examinations of occupational divers. *Internal Medicine Journal*. 2009;39:763-70.

Commentary

At each pre-dive risk assessment working divers have the duty to report any recent illness or injury that might affect their fitness to dive. Examination by a medical practitioner ('approved' and audited by the relevant diving jurisdiction) is needed periodically to assess fitness to return to diving after illness or injury but is more commonly met as an 'annual diving-medical examination'. It is refreshing that this paper from New Zealand provides evidence to challenge the comfort of that universal tradition. Potential application of the five-yearly medical examinations in New Zealand to other jurisdictions (as determined by their maritime boundaries or by the 'flag' of diving support vessels) should next be assessed where there are some thousands of working divers, many of whom may be exposed to a great range of occupational hazards.

Such a review needs to include the extent to which an annual questionnaire can or cannot recognise and monitor the consequences of exposure to diving hazards, e.g., hearing loss, juxta-articular necrosis, welding-fume exposure or exposure to in-water contaminants, including carcinogens, acid muds and complex, dissolved hydrocarbons. Perhaps a central expert is best placed to evaluate the level of risk determined by occupational hygienists and others, and to recommend the need for medical surveillance of the individual. Additionally, what checks need to be recorded on the physical strength and endurance that each diver needs to maintain for an emergency out-of-bell rescue? In jurisdictions in which there is no statutory upper age limit for working divers, should the full assessment of cognitive and cardio-vascular factors by an approved doctor become more frequent with the diver's increasing age?

The minimal training required by a medical examiner of divers is determined by the relevant national authority. Many national authorities no longer approve 'overseas' doctors because these cannot be held legally accountable for errors they might make from within another jurisdiction. Training requirements differ around the world and some nations also require medical examiners to have refresher training plus minimal annual experience to maintain validation. International training objectives, with a greater definition of detail, exist to cover potential gaps in sparse national regulations but, though required by some international diving contractors, these are not applied universally.

The foundation has now been laid for introducing an effective change for divers but, for international acceptance, more may be needed on the medical surveillance of exposure to hazards at work.

Professor David H Elliott is a life member of SPUMS.
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Key words
Occupational diving, diving at work, health surveillance, occupational health, fitness to dive, abstracts

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates (updated October 2008)

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

1 The candidate must be medically qualified, and be a current financial member of the Society.
2 The candidate must supply evidence of satisfactory completion of an examined two-week full-time course in Diving and Hyperbaric Medicine at an approved facility. The list of approved facilities providing two-week courses may be found on the SPUMS website.
3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
4 The candidate must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval *before* commencing their research project.
5 The candidate must produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this written report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.
6 In the absence of documentation otherwise, it will be assumed that the paper is submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the instructions to authors – full version, published in *Diving and Hyperbaric Medicine* 2008; 38(2): 117-9.
7 The paper may be submitted to journals other than *Diving and Hyperbaric Medicine*; however, even if published in another journal, the completed paper must be submitted to the Education Officer for assessment as a diploma paper. If the paper has been accepted for publication or published in another journal, then evidence of this should be provided.
8 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 accepted or published in other journals will be forwarded to the Editor of *Diving and Hyperbaric Medicine* for consideration. At this point the Diploma will be awarded, provided all other requirements are satisfied. Diploma projects submitted to *Diving and Hyperbaric Medicine* for consideration of publication will be subject to the Journal's own peer review process.

Additional information – prospective approval of projects is required

The candidate must contact the Education Officer in writing (e-mail is acceptable) to advise of their intended candidacy, and to discuss the proposed subject matter of their research. A written research proposal must be submitted before commencing the research project.

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice (available at <http://www.health.gov.au/nhmrc/research/general/nhmrcavc.htm>) or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documented evidence of approval by an appropriate research ethics committee. 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.

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

The Academic Board reserves the right to modify any of these requirements from time to time. As of October 2008, the SPUMS Academic Board consists of:
Associate Professor David Smart, Education Officer;
Associate Professor Mike Davis;
Associate Professor Simon Mitchell.

All enquiries and applications to the Education Officer:
Associate Professor David Smart
GPO Box 463, Hobart, Tasmania 7001
E-mail: *<david.smart@dhhs.tas.gov.au>*

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

Notice of SPUMS Annual General Meeting 2010

The AGM for SPUMS 2010 will be held at Berjaya Redang Resort, Redang Island, Malaysia at 1700h, Thursday 27 May 2010.

Agenda

Apologies:
Minutes of the previous meeting:
Minutes of the previous meeting will be posted on the notice board at Berjaya Redang Resort and were published in *Diving and Hyperbaric Medicine.* 2009;39(4):243–7.
Matters arising from the minutes:
Annual reports:
President's report
Secretary's report
Education Officer's report
Annual financial statement and Treasurer's report
Journal Editor's report
Presidents' Committee report
Subscription fees for 2011:
Treasurer
Election of office bearers:
No positions are open for election in 2010.
Appointment of the Auditor 2010:
Treasurer
Business of which notice has been given:
No notices have been received at this stage for other business. Any notice for other business must be received in writing to the Secretary by 31 April 2010.

Members' news

Commodore Robyn Walker, former President of SPUMS, has been made a Member of the Order of Australia (AM) - Military Division for exceptional service as a medical officer in the Australian Defence Force.

Dr Simon Mitchell has been promoted to Associate Professor in the Department of Anaesthesia, Faculty of Medicine and Health Sciences, The University of Auckland, New Zealand.

ANZHMG Chairman's report

Members of the Australian and New Zealand Hyperbaric Medicine Group (ANZHMG), representing the comprehensive diving and hyperbaric medicine facilities (as defined in the Commonwealth Medicare Benefits Schedule, CMBS) in Australia and New Zealand have just completed a major submission to the Australian Federal Government Medical Services Advisory Committee (MSAC). This submission seeks to achieve long-term funding from Medicare for hyperbaric oxygen treatment (HBOT) of soft-tissue radiation injury and necrosis, and non-diabetic problem wounds where hypoxia can be demonstrated. The process of submission was an enormous undertaking, akin to performing a complete Cochrane evidence review, combined with disease load, epidemiology, quality of life and economic analyses, submitted in Government format for each specific condition to be funded.

The issue has caused headaches (literally) for ANZHMG members for over a decade, and we have yet to achieve full closure. The current submissions (two documents totaling over 500 pages) are supported by the Australian Healthcare and Hospitals Association and the Australian Society of Anaesthetists as co-applicants, and are also backed by the Australian Medical Association. A brief history is outlined below, as this may be of value to hyperbaric physicians fighting the same battles in their own jurisdictions.

HBOT has been funded since the CMBS started, and, until 2001, the use of the treatment was at the discretion of the specialists who work in the field. These specialists were already exercising considerable self-regulation by adhering to treatment for cases where clinical opinion considered the evidence to be high enough for efficacy. For example, treatment was not offered for multiple sclerosis, sports injuries, cerebral palsy, autism, and many other conditions. In 1999, a manufacturer of monoplace hyperbaric chambers applied to have a separate Medicare item number added to the CMBS, via MSAC. There was, of course, no difference in the treatment (monoplace versus multiplace), in a properly constituted hyperbaric treatment facility, but MSAC in its first report 1018-20 sought to severely restrict the medical conditions that could be treated by HBOT, citing "evidence" as their guiding principle. In essence, a process that had been set up to review new technologies had been used to review an existing technology, and then to withdraw the funding from that technology (HBOT). As a result, ANZHMG was inadvertently dragged into a saga that has lasted a decade.

An example of how far evidence-based health-funding ideology can be taken is demonstrated in Germany, where recompression treatment is not accepted for decompression illness (DCI), because there is no level 1 or 2 evidence supporting the therapy. Fortunately there was some common sense prevailing in Australia, and DCI is one of seven conditions fully funded by Medicare. In 2003, ANZHMG

made further submissions to MSAC to restore full funding of soft-tissue radiation injury and necrosis, and non-diabetic problem wounds where hypoxia can be demonstrated. Four ANZHMG members joined the supporting committee to review the evidence. Despite positive conclusions from the supporting committee, the conclusions that were published in the final report (2004) were not the same as those that left the supporting committee, and had been modified by MSAC, without further consultation, to a negative tone with only short-term funding provided. Since 2004, three-yearly funding only has been achieved.

This year, the submissions have been split. The HBOT for soft-tissue radiation injury or necrosis submission has reviewed all of the evidence for all treatments, from the perspective of a patient presenting with the condition. If a patient has soft-tissue radiation injury, what are the best treatment options and what is the evidence supporting the treatments? The review found that there were only 27 papers in the world literature of level 3C evidence or above, for all treatments including surgery and other medical treatments! HBOT had evidence of clinical efficacy that was equal to or superior to all other available treatment options.

For non-diabetic problem wounds, a detailed literature review has also taken place, but with considerable emphasis on the results of an ongoing prospective study of refractory hypoxic problem wounds being treated at Australasian facilities (see a previous progress report in *Diving and Hyperbaric Medicine*).[1] We believe that the work completed by members represents an extremely strong case, and we have been assured by Federal Department of Health Australia representatives that MSAC has a fresh, new approach. The process will unfold during the year.

Reference

1 Hawkins GC, Bennett MH, van der Hurst AE. The outcome of chronic wounds following hyperbaric oxygen therapy: a prospective cohort study the first year interim report. *Diving and Hyperbaric Medicine*. 2006;36(2):94-8.

Associate Professor David Smart
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Key words
Medical society, wounds, hyperbaric oxygen therapy, general interest

Standards Australia report 15 January 2010

For the past two years, there has been very little activity with Australian Standards. Soon after Australian Standard 2299.1(2007) was released, a major corporate shake-up occurred at Standards Australia, which has prevented further progress on diving standards. From 2008, a change in strategic direction meant that all future development of Australian standards required full funding and sponsorship by industry groups. That, coupled with an apparent embracement of 'international standards', regardless of their quality, has led to serious disillusionment amongst members who have previously given much effort and time to serve on Standards Australia committees.

I have seen e-mailed documents, touted as international diving training standards from the recreational diving industry, that are nowhere near the quality of documents that already exist among our training organisations within Australia and New Zealand. The move to adopt 'ISO or perish' seems to be politically driven, and, in my opinion, the latter is likely to be the consequence. I had become somewhat cynical of the whole process until recently, when a breath of fresh air blew through with the latest Acting CEO's newsletter (December 2009):

"In November, Standards Australia announced the immediate reopening of its funded and supported development pathways for new projects. This revised approach was developed in conjunction with and supported by the Commonwealth and major member groups. In brief, if proposed Standards development projects can demonstrate the delivery of Net Benefit to communities of interest, and these projects cannot be financially resourced, then Standards Australia will allocate resources on a prioritized basis to see the work through to completion."

This may offer an opportunity for SPUMS to develop its newly revised recreational diving medical assessment form into an updated Australian recreational diving standard.

David Smart
South Pacific Underwater Medicine Society Representative to Standards Committee for Occupational Diving Australia, Standard Australia

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Key words
Fitness to dive, medicals – diving, recreational diving, standards

Australian and New Zealand College of Anaesthetists

Certificate in Diving and Hyperbaric Medicine

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

Eligibility criteria are:

1 Fellowship of a Specialist College in Australia or New Zealand. This includes all specialties, and the Royal Australian College of General Practitioners.

2 Completion of training courses in Diving Medicine and in Hyperbaric Medicine of at least four weeks' total duration. For example, one of:
   - a ANZHMG course at Prince of Wales Hospital Sydney, **and** Royal Adelaide Hospital or HMAS Penguin diving medical officers course **OR**
   - b Auckland University Diploma in Diving and Hyperbaric Medicine.

3 **EITHER:**
   - a Completion of the Diploma of the South Pacific Underwater Medicine Society, including six months' full-time equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS
   - b **and** Completion of a further 12 months' full-time equivalent clinical experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA.

   OR:
   - c Completion of 18 months' full-time equivalent experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA
   - d **and** Completion of a formal project in accordance with ANZCA Professional Document TE11 "Formal Project Guidelines". The formal project must be constructed around a topic which is relevant to the practice of Diving and Hyperbaric Medicine, and must be approved by the ANZCA Assessor prior to commencement.

4 Completion of a workbook documenting the details of clinical exposure attained during the training period.

5 Candidates who do not hold an Australian or New Zealand specialist qualification in Anaesthesia, Intensive Care or Emergency Medicine are required to demonstrate airway skills competency as specified by ANZCA in the document "Airway skills requirement for training in Diving and Hyperbaric Medicine".

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

Dr Margaret Walker, FANZCA, Cert DHM (ANZCA)
Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine

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ANZCA Certificate in Diving and Hyperbaric Medicine
Examination results

Successful candidates, November 2009
On behalf of the SIG executive and examiners court, I wish to congratulate the following candidates who were successful at the recent examination:

Neil Banham (Western Australia)
David Cooper (Tasmania)
Ian Dey (Western Australia)

Michael Bennett
Chair, Examination Committee

The Executive Committee of SPUMS would also like to congratulate the candidates, all three of whom are SPUMS members.

1st Joint Meeting
South Pacific Underwater Medicine Society
Asian Hyperbaric and Diving Medical Association

The 39th SPUMS Annual Scientific Meeting combined with the 6th ASM of the Asian Hyperbaric and Diving Medical Association

23–28 May 2010

Venue: Redang Island Beach Resort

Theme: Decompression and Hyperbaric Medicine into the 21st Century

SPUMS Guest Speaker
Michael L Gernhardt, PhD
NASA Astronaut, Manager of the Environmental Physiology Laboratory and
Principal Investigator of the Prebreath Reduction Program
Johnson Space Center, Houston, Texas

AHDMA Guest Speaker
Folke Linde, MD, PhD
Professor, Department of Anaesthesiology, Surgical Services and Intensive Care
Karolinska University Hospital, Stockholm, Sweden

MEETING CONVENOR
Dr Glen Hawkins
PO Box 1674
Maroubra, NSW 2035
AUSTRALIA
E-mail: <glen@hawkeyemedical.com.au>

Full details for submission of Abstracts and Posters are on line, and
Registration, Accomodation and Travel should be booked on line.
Full details are available on the SPUMS website.
Members are urged to book as soon as possible.

The
website is at
www.spums.org.au

EUBS European Underwater and Baromedical Society

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The EUBS website is at
www.eubs.org
Members are encouraged to log in

EUBS website news

Explore the EUBS website! It offers the following sections:

For all (non-members and members):

Events, courses and Research pages: lists the currently available multicentric research initiatives seeking collaboration and recruiting investigators; also listing courses and events to provide you with possibilities for CME and scientific exchange and interaction.

Links page: lists international, national and regional organisations and scientific societies dealing with hyperbaric and diving medicine.

Our Constitution and Bylaws: so that you know what EUBS stands for.

The Executive Committee or ExCom: you are invited to contact any of them if you have suggestions, critique, propositions etc.

Our Corporate Members: institutions and organisations who support the works of the EUBS with their contribution.

For members only:

A direct link to the comprehensive bibliography database: offered graciously by the GTUeM (German Society for Diving and Hyperbaric Medicine). This database provides full text files of all published material in the field of diving and hyperbaric medicine – also non-peer-reviewed material such as Annual Meeting Proceedings. The full text of previous issues of DHM will also be available, from three years previous.

EUBS membership directory: with full search capability: your source to locating colleagues within Europe.

Membership maintenance: check your membership dues history; renew your membership online.

Private Discussion Forum: while it takes some discipline to regularly visit the Forum, this may be a good way to obtain information and/or exchange ideas. It is divided into topics so there is little chance that off-topic discussion will clutter your screen. Who will start a discussion topic?

Peter Germonpré
EUBS webmaster

Election of Member-at-Large: candidates sought

A new Member-at-Large (2010–2013) needs to be elected to serve a three-year term on the Executive Committee. Candidates should be a full member of EUBS and willing to donate some of their time to help run the Society. This is your chance to actively help the EUBS grow and develop!

Please send a one-page CV with digital photograph to our Honorary Secretary <joerg.schmutz@eubs.org> before 30 April. If you do not think you are up to this task, please nominate someone you think might be willing and able (send an e-mail with your suggested nominee to Joerg).

Towards a European diploma in diving and hyperbaric medicine

The current European standards for diving and hyperbaric medicine post-graduate education, as defined by the European Committee for Hyperbaric Medicine (ECHM), together with the European Diving Technology Committee (EDTC), and in agreement with the Diving Medical Advisory Committee (DMAC – commercial diving), were defined during the ECHM 2004 European Consensus Conference on Hyperbaric Medicine (Lille 2004) and the coincidental closing meeting of the European Union COST B14 Action. It was decided that the above-mentioned medical education programmes be accredited by the European College of Baromedicine (ECB), in strict accordance with the standards as set out by the ECHM/EDTC.

This international endeavor has so far involved a number of academic and clinical institutions in many European as well as extra-European countries, such as, to name a few, Belgium, Cyprus, Denmark, France, Germany, Holland, Italy, Malta, Portugal, Serbia, Sweden, UK, South Africa and the USA, through the Undersea Hyperbaric Medical Society. The educational process is currently organized into three levels:

- Level I, basic information: Medical examiner of divers and/or assistant in a hyperbaric medicine practice;
- Level II: Diving medical officer and/or hyperbaric medical officer;
- Level III: Expert in diving and/or hyperbaric medicine.

The ECHM/EDTC standard and ECB accreditation system foresees the development of a European post-graduate education modality which will be both modular and offer full reciprocity amongst the participating academic and medical education institutions. Therefore, it accepts courses which are modular in design so that they cover selected, specific sections of the entire curriculum, whilst allowing for their accreditation, methods for the recognition of prior learning and reciprocity with other participating institutions where the education programme may then be continued or completed. The goal is to achieve a recognized European diploma in baromedicine!

Medical institutions (academic, clinical and educational) interested in receiving more information and participating in this European programme are invited to visit the websites of the ECHM, <http://www.echm.org>, and the ECB, <http://www.ecbm.eu>, or to write to <ecb@daneurope.org>.

Alessandro Marroni,
Vice President, ECHM, Secretary General, ECB

Key words
Diving medicine, hyperbaric medicine, qualifications, training, general interest

EUBS European Underwater and Baromedical Society

EUBS Annual Scientific Meeting 2010

14–18 September 2010
Istanbul (European Cultural Capital 2010)

Venue: The Marmara Hotel, Istanbul, Turkey

Prof. Maide Cimsit, Istanbul University
Secretary General, EUBS ASM 2010
E-mail: <mcimsit@istanbul.edu.tr>

Istanbul is a centuries-old city, located on the Bosphorus Strait connecting Asia and Europe. It was the capital of three empires: Roman, East Roman (Byzantine), and the Ottoman Empire. Many historic areas are on the UNESCO World Heritage List. Istanbul is unique with its location, cultural and historical heritage, palaces, and monuments, museums and bazaars, blending with modern architecture, shopping centres, and all sorts of restaurants, clubs and friendly wine houses.

The scientific programme will cover a broad spectrum of topics in diving and hyperbaric medicine.
Full details of the scientific programme and workshops are available on the meeting website:
<http://www.eubs2010.org>

Submission of Abstracts

Deadline for Abstract submission is 15 June 2010. Work can be presented as either a poster or an oral presentation.

Zetterström Award:

Authors of accepted poster abstracts are encouraged to submit their poster for the Zetterström Award.

Student Travel Grants

Applications for student travel grants should be in the hands of the Scientific Secretariat by 15 May 2010.

Details about how to submit an Abstract, apply for and the conditions of the Zetterström Award, or apply for a student travel grant can be found on the conference website.
Official language of the conference will be English.

Social programme

Details of the many optional activities, including a diving programme, tours and excursions, in the Social Programme may be found on the website or are available from the Congress Secretariat.
We hope that you will enjoy the meeting, and the unique ambience and hospitality of Istanbul.

For important dates, registration, accommodation and other details please visit the meeting website.

Contacts:

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Central European Conference of Hyperbaric and Diving Medicine and Ostrava's Days of Hyperbaric Medicine

Dates: 17-18 June 2010
Venue: Lanterna Hotel, Leskove 659, Velke Karlovice 756 06, Czech Republic
Conference Chairman: Michal Hajek, MD
Scientific topics:

- Diving medicine and physiology; decompression illness; news in diagnostics and treatment of PFO
- Hyperbaric medicine – recommended, optional and experimental indications
- Hyperbaric physiology; experimental HBO; oxygen toxicity
- Organizational aspects of HBO treatment; education of HBO staff
- Aviation medicine

Organizing Committee address:
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Website: <www.hbova.cz> and <www.cshlm.cz>

Diving Diseases Research Centre (DDRC), Plymouth, UK

Diving medicine courses for 2010

- Level I (Medical Examiner of Divers) Course: 17 September
- Medical Examiner of Divers, Refresher Course: 10–11 June and 25–26 November
- Level IIa (Diving Medical Physician): 20–24 September
- Introduction to Hyperbaric Medicine Course for Physicians (UHMS): 13–17 September
- Combined Introduction to Hyperbaric Medicine Course for Physicians (UHMS) and Level I (Medical Examiner of Divers) Course: 13–19 September

For further information: <www.ddrc.org>

7th National Congress of the Società Italiana di Anestesia Rianimazione Emergenza e Dolore

Dates: 29–31 May
Venue: Villasimius, Cagliari, Sardinia.

A major part of the programme is dedicated to diving and hyperbaric medicine, with an international faculty.

Website: <http://www.siared.it/congresso/siared_2010_congresso_locandina.pdf>

The Royal Swedish Navy in cooperation with Sahlgrenska University Hospital, Gothenburg University

Basic course in diving medicine and HBO

Dates: 20 September – 1 October 2010
Venue: Gothenburg, Sweden

For further information:
E-mail: Lena Fridman <lena.fridman@mil.se>

Oxygen and Infection; European Committee for Hyperbaric Medicine (ECHM) Conference Proceedings

Free video lectures <www.hyperbaricoxygen.se>
Video lectures and panel discussions from three exciting conference days in Stockholm, Sweden 7–9 May, 2009 are now available for your iPhone or computer for free!

5th Karolinska Postgraduate Course in Clinical Hyperbaric Oxygen Therapy
14 lectures on fundamental concepts and front-line knowledge in the clinical use of HBO.

ECHM Conference 'Oxygen and Infection'
22 lectures and three panel discussions are available on topics such as necrotizing fasciitis and the diabetic foot.

Our site offers free, high-quality presentations from leading authorities and principal investigators in the field of Hyperbaric Medicine. Our goal is to spread knowledge, and to support clinical practice and research for the benefit of patients around the world. Welcome and enjoy!

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

Inter-university Diploma in Diving and Hyperbaric Medicine, France

University Course (1-year duration) in diving and hyperbaric medicine, organised concurrently by 13 French universities (Angers, Antilles-Guyane, Besançon, Bordeaux II, Lille 2, Lyon II, La Réunion, Marseille, Nancy, Nice, Paris XIII, Strasbourg, Toulouse).

For further information go to:
<http://www.medsubhyp.org> or
<http://medecine.univ-lille2.fr/format/diu/hyperbar.htm>

German Society for Diving and Hyperbaric Medicine (GTUeM)

An overview of basic and refresher courses in diving and hyperbaric medicine, organised by the GTUEM in Germany, can be found at:
<http://www.gtuem.org/212/Kurse_/_Termine/Kurse.html>

Scott Haldane Foundation, The Netherlands

The Scott Haldane Foundation is dedicated to education in diving medicine, and has organised more than 100 courses over the past few years, both in the Netherlands and abroad. Below is an overview of courses planned for 2010.

More information can be found at:
Website: <www.scotthaldane.nl>
E-mail: <info@scotthaldane.nl>

29 May–6 June: Basic course "Diving medicine for ENT specialists" (Negros, Philippines)
11 June: 16th Advanced course "Diving and ENT" (Driebergen, NL)
2–9 October: Basic course "Diving medicine for pneumologists"
14–15 October: Advanced course "Evidence-based diving medicine" (Doorn, NL)
6–13 November: Basic course in diving medicine (Zanzibar, Tanzania)
13–20 November: 17th Advanced course in diving medicine (Zanzibar, Tanzania)
13–20 and 20–27 November: 17th Advanced course in diving medicine (Zanzibar, then Mafia Island, Tanzania)
11 December: Refresher course "Neurology and diving"

Undersea and Hyperbaric Medical Society Annual Scientific Meeting 2010

Dates: 3–5 June 2010
Venue: Tradewinds Grand Island Resort
St Pete Beach, Florida, USA

Pre-courses: 2 June
Wound care
How to prepare for accreditation

UHMS is accredited to provide continuing medical education for physicians

Full details of the programme, registration and accomodation are available on the UHMS website <www.uhms.org> or
for further information contact:
Lisa Tidd, UHMS
Phone: +1-(0)877-533-UHMS/919-490-5140
E-mail: <lisa@uhms.org>

Conference proceedings available
The future of diving: 100 years of Haldane and beyond

Michael A Lang and Alf O Brubakk, editors

Smithsonian Institution Scholarly Press

The proceedings of "*The Future of Diving: 100 Years of Haldane and Beyond*" symposium, convened 18–19 December 2008 in Trondheim, Norway, by the Baromedical and Environmental Physiology Group of the Norwegian University of Science and Technology, are reported in 28 papers and 3 discussion sessions.

Download a PDF of this publication through:
<www.scholarlypress.si.edu>

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<schol_press@si.edu>
Print copies of this publication are free upon request, while supplies last; limit five (5) copies.

The Hyperbaric Research Prize

The Hyperbaric Research Prize encourages the scientific advancement of hyperbaric medicine and is awarded annually whenever a suitable nominee is identified. It will recognise a scholarly published work or body of work(s) either as original research or as a significant advancement in the understanding of earlier published science. The scope of this work includes doctoral and post-doctoral dissertations. The Hyperbaric Research Prize is international in scope. However, the research must be available in English. The Hyperbaric Research Prize takes the form of commissioned art piece and US$10,000 honorarium.

For detailed information please contact:
Baromedical Research Foundation
5 Medical Park, Columbia, SC 29203, USA
Phone: +1-803-434-7101
Fax: +1-803-434-4354
E-mail: <samir.desai@palmettohealth.org>

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village
Victoria, 3172, Australia
E-mail:
<deswill@dingley.net>
Website:
<www.classicdiver.org>

British Hyperbaric Association 2010 Annual Conference

Dates: 18–21 November
Host: East of England Hyperbaric Unit
James Paget University Hospitals NHS
Lowestoft Road
Gorleston Great Yarmouth
Norfolk NR31 6LA

For further information contact:
Karen Turner <karen.turner@jpaget.nhs.uk>
or
Maxine Palmer <maxine.palmer@jpaget.nhs.uk>
Phone: +44-(0)1493-453526
Fax: +44-(0)1493-453261

Introductory Course in Diving Medicine New Zealand

Dates: 24–27 September 2010
Venue: Navy Hospital, Devonport, Auckland

This course is designed to provide GPs who have an interest in diving medicine with a basic understanding of the principles involved.

RNZCGP approved for 20 hours CME.

For details and application form please see the website: <www.navyhyperbaric.mil.nz>

CO_2: The rebreather incident

Freely available from: <www.hse.gov.uk/diving/video/co2video.htm>

The following is a quote from the HSE website:
"*This video made by SKY news shows real footage of an incident where a rebreather diver suffers from acute carbon dioxide poisoning. The video takes the viewer through the incident, explaining how it came about and the lessons that can be learnt. Whilst the video is primarily aimed at rebreather divers, there are issues such as bail-out gas consumption, team size and rescue that may be useful to all divers.* [Also, it gives insight for diving physicians into one of the potentially fatal causes of rebreather accidents. – Editor] *HSE is providing approved recreational training organisations with DVD copies of the video for distribution to their rebreather instructors. Any divers who want a copy of the DVD should contact their training agency. The film carries no copyright and can therefore be distributed to all interested parties.*"

Royal Adelaide Hospital Diving Medicine Medical Officers and Diver Medical Technician Courses 2010

Medical Officers Course:
Week 1, 21–25 June
Week 2, 28 June – 2 July

Full DMT Courses:
2nd DMT course in November still t.b.d.

DMT Refresher Course:
26–30 October

For more information contact:
Lorna Mirabelli
Senior Administrative Assistant
Hyperbaric Medicine Unit, Royal Adelaide Hospital
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lmirabel@mail.rah.sa.gov.au>

2010 Royal Australian Navy Medical Officers Underwater Medicine Course

Dates: 25 October–5 November 2010
Venue: HMAS PENGUIN, Sydney
Cost: to be advised

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

For information and application forms contact:
Mr Rajeev Karekar for Officer in Charge,
Submarine and Underwater Medicine Unit
HMAS PENGUIN
Middle Head Rd, Mosman, 2088 NSW, Australia
Phone: +61-(0)2-99600572
Fax: +61-(0)2-99604435
Email: <Rajeev.Karekar@defence.gov.au>

Instructions to authors

(revised March 2009)

Diving and Hyperbaric Medicine welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts, including SPUMS Diploma theses, will be subject to peer review. Accepted contributions will be subject to editing.

Contributions should be sent to:
The Editor, Diving and Hyperbaric Medicine,
C/o Hyperbaric Medicine Unit, Christchurch Hospital,
Private Bag 4710, Christchurch, New Zealand.
E-mail: *<editor@dhmjournal.com>*

Requirements for manuscripts
Documents should be submitted electronically on disk or as attachments to e-mail. The preferred format is Microsoft® Office Word 2003. Paper submissions will also be accepted. All articles should include a title page, giving the title of the paper and the full names and qualifications of the authors, and the positions they held when doing the work being reported. Identify one author as correspondent, with their full postal address, telephone and fax numbers, and e-mail address supplied. The text should generally be subdivided into the following sections: an Abstract of no more than 250 words, Introduction, Methods, Results, Discussion, Conclusion(s), Acknowledgements and References. Acknowledgements should be brief. Legends for tables and figures should appear at the end of the text file after the references.

The text should be double-spaced, using both upper and lower case. Headings should conform to the current format in *Diving and Hyperbaric Medicine*. All pages should be numbered. Underlining should not be used. Measurements are to be in SI units (mmHg are acceptable for blood pressure measurements) and normal ranges should be included. Abbreviations may be used once they have been shown in brackets after the complete expression, e.g., decompression illness (DCI) can thereafter be referred to as DCI.

The preferred length for original articles is up to 3,000 words. Including more than five authors requires justification, as does more than 30 references. Case reports should not exceed 1,500 words, with a maximum of 15 references. Abstracts are required for all articles. Letters to the Editor should not exceed 500 words with a maximum of five references. Legends for figures and tables should generally be less than 40 words in length.

Illustrations, figures and tables must NOT be embedded in the wordprocessor document, only their position indicated. No captions or symbol definitions should appear in the body of the table or image.
Table data may be presented either as normal text with tab-separated columns (preferred) or in table format. No gridlines, borders or shading should be used.
Illustrations and figures should be submitted as separate electronic files in TIFF, high resolution JPG or BMP format. If figures are created in Excel, submit the complete Excel file. Large files (> 10 Mb) should be submitted on disk.
Photographs should be glossy, black-and-white or colour. Colour is available only when it is essential and may be at the authors' expense. Indicate magnification for photomicrographs.

References
The Journal reference style is the 'Vancouver' style (Uniform requirements for manuscripts submitted to biomedical journals, updated May 2007. Website for details: <http://www.nlm.nih.gov/bsd/uniform_requirements.html>). References must appear in the text as superscript numbers at the end of the sentence after the full stop.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used (<http://www.nlm.nih.gov/tsd/serials/lji.html>). Examples of the exact format for a standard paper and a book are given below:

1 Freeman P, Edmonds C. Inner ear barotrauma. *Arch Otolaryngol*. 1972;95:556-63.
2 Hunter SE, Farmer JC. Ear and sinus problems in diving. In: Bove AA, editor. *Bove and Davis' diving medicine*, 4th ed. Philadelphia: Saunders; 2003. p. 431-59.

Place a full stop after the journal name and at the end of the reference. Titles of books and journals should be in italics. Accuracy of the references is the responsibility of authors.

Any manuscript not complying fully with the above requirements will be returned to the author before being considered for publication.

Consent
Studies on human subjects must comply with the Helsinki Declaration of 1975 and those using animals must comply with National Health and Medical Research Council Guidelines or their equivalent. A statement affirming Ethics Committee (Institutional Review Board) approval should be included in the text. A copy of that approval should be available if requested.

Copyright
Authors must agree to accept the standard conditions of publication. These grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in printed form in *Diving and Hyperbaric Medicine* and in other media, including electronic form. Also granting the right to sub-licence third parties to exercise all or any of these rights. *Diving and Hyperbaric Medicine* agrees that in publishing the article(s) and exercising this non-exclusive publishing sub-licence, the author(s) will always be acknowledged as the copyright owner(s) of the article.

Full instructions to authors (revised June 2009) can be found on the EUBS and SPUMS websites.

DIVER EMERGENCY SERVICES PHONE NUMBERS

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

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

SOUTH-EAST ASIA
+852-3611-7326 (China)
010-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

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

EUROPE
+39-06-4211-8685 (24-hour hotline)

UNITED KINGDOM
+44-07740-251-635

USA
+1-919-684-8111
+52-5-629-9800 (America-Mexico))

LATIN AMERICA
+1-919-684-9111 (may be called collect; Spanish and Portuguese)

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

DAN Asia-Pacific DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. Information, all of which is treated as being confidential in regard to identifying details, is utilised in reports on fatal and non-fatal cases.

Such reports can be used by interested people or organisations to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence unless otherwise agreed) to:

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

DIVING INCIDENT MONITORING STUDY (DIMS)

DIMS 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.

Diving Incident Report Forms (Recreational or Cave and Technical)
can be downloaded from the DAN-AP website: <www.danasiapacific.org>
They should be returned to:
DIMS, 30 Park Ave, Rosslyn Park, South Australia 5072, Australia.

CONTENTS

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