TABLE 8
TIME TO SYMPTOM ONSET AFTER SURFACING

Time	Cases	Accumulated percentage
10 min	2	8%
20 min	1	11%
First 30 min	9	35%
First 4 hours	16	61%
First 8 hours	23	89%
Between 16 and 24 hours	3	11%
Total	26	100%

TABLE 9

TIME FROM ONSET OF SYMPTOMS TO RECOMPRESSION

Minutes/cases		Hours/cases		Days/cases	
30	1	1	2	2	3
		3	1	3	6
		4	1	4	2
		6	2	5	1
		7	1	6	2
		10	1	9	1
		12	4		
		15	1		
		18	1		

TABLE 10

FIRST AID BEFORE REACHING HST

Oral fluids	3
Oxygen via free flow	3
Oxygen via demand valve	2
Aspirin	1
None	20
Total	29

improvement in the early management of DCI cases in the region.

All 30 patients had a US Navy (USN) Table 6 as initial treatment. For some cases the table was extended. For those who had not completely recovered USN Table 5 was used for follow up treatments. The average number of treatments was 3.83.

Seven patients had residual symptoms after recompression therapy was completed. Because these

patients moved away at the end of their holiday, follow up has not been possible.

Discussion

South Thailand has a booming recreational diving industry. In 1997 we treated 22 cases of DCI, in 1998 there were 42 and the total for 1999 will be higher. More specific data collection and analysis of the factors that resulted in DCI will be undertaken. The findings of the physical examination will also be described and follow up will be included in further reports.

In 1998 divers with less than 2 years experience accounted for 21 (50%) of our injured divers. The high percentage (40%) of locally based instructors or dive guides who suffered DCI deserves a detailed analysis. In the 1998 DAN report this group accounted for only 17% of the reported cases. It is likely that the reason lies in their pattern of diving and complete reliance on their dive computers.

The long delays in seeking treatment, with less than 20% of patients presenting in the first 6 hours after developing symptoms, can be attributed more to ignorance, or denial, rather than to distance to the recompression facility. Education of the local dive providers and medical personnel is needed in prevention, symptom recognition, on site management and evacuation procedures of DCI patients.

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TEAR FILM BUBBLES AND DECOMPRESSION ILLNESS. FINALLY A DIAGNOSTIC TEST TO CRY FOR?

Mike Bennett

Key Words

Bubbles, decompression illness, hyperbaric research.

Introduction

This presentation is intended to outline what may, in time, prove to be an important diagnostic tool for

decompression illness, examination of the tear film for small gas bubbles. It is not clear if this is going anywhere, but there are suggestions that such an examination at the time of presentation may prove clinically useful.

The diagnosis of decompression illness is not a straightforward proposition. In essence it is an historical diagnosis. The character, evolution and relationship of symptoms to the time and nature of the dive are of prime importance. Examination may contribute to diagnostic accuracy in some cases. We measure various neurological parameters, but quite often, there is very little to find on examination. There may be a role in some situations for measuring bubbles by a Doppler technique, but this is uncommon in the clinical setting.

There are other diagnostic tests that have been looked at over the years, and each has found its place in modern practice. For example, it may be useful to look at a chest X-ray if part of the differential diagnosis is pulmonary barotrauma. Clotting studies have been looked at from time to time around the Australasian hyperbaric centres. There are various scanning techniques, but these are more useful in the assessment of persistent symptoms and signs than as primary diagnostic tools. CT scanning, to take an example, is probably no more accurate as a diagnostic investigation than tarot cards, runes, and the favourite at our unit, swirling the tea leaves around while waiting for the patient to arrive.

Tear film bubbles

Why look for tear film bubbles? In 1670 Boyle observed bubbles in the aqueous humour of a viper's eye. This was the first description of a bubble event in association with the eye. Little further interest was shown until 1978 when Simon and Bradley described the presence of bubbles under a hard contact lens after diving. Socks,



Figure 1. Slit-lamp examination

Molinari and Rowey first described bubbles under a gaspermeable contact lens in 1989 and later under a soft contact lens.³ In 1991 Strath and Mekjavic described them under rigid centre, soft surrounding lenses.⁴ It was pretty clear then, that if one wore contact lenses, there was some risk of developing bubbles underneath the lenses. This seemed to have no importance for people who were not wearing contact lenses.

In 1992 however, it was noticed that after repetitive dry diving, attending patients in a chamber, an attendant had bubbles in the eye, despite never having worn contact lenses.⁵ That led to the work that we have done in an attempt to define any relationship between the presence of tear film bubbles and decompression stress.

Technique

The tear film is inspected through a standard slitlamp (Figure 1) which is a standard tool for ophthalmologists and relatively easy to learn to use. However, unlike most slit-lamp examinations, we are not looking into the aqueous humour or at the cornea, iris and lens, but are primarily interested in the tear film.

The procedure with the slit-lamp is to sweep slowly from the medial to the lateral border of the inferior gutter (lower eyelid), counting bubbles, if any, as one goes. Such bubbles are often small and moving rapidly from lateral to medial within the gutter. It is important to limit one's inspection to the gutter itself in order to standardise the examination and because small bubbles on the lid itself are not uncommon, being the result of physical "foaming" after blinking. The subject is asked to close the eyes for 5 seconds, open them again and the examination repeated. Three sweeps are made and the bubble count averaged. This is a reasonably standard protocol as reported in the literature. Some centres have photographed the bubbles and made an estimate of bubble volume for each individual. It is not yet known if this significantly improves the accuracy of the procedure.

Tear film structure

The tear film is a surprisingly complex structure. The tear film's primary roles are to wet the globe, enable the lids to open and close with minimal friction and to reduce shearing stress on the globe itself. This contributes to the cornea remaining pristine and transparent. While there is clearly a film of tears over the whole globe, in the gutter along the bottom it is much thicker. The tear film flows surprisingly quickly from the outer to the inner canthus and down into the tear ducts.

When Strath et al. noted bubbles in the tear film of their chamber attendant, they did a reasonably simple study.⁵

They took 11 volunteers, compressed them in a chamber to 4 bar for 15 minutes and examined their tear films pre- and post-dive. Before compression, there was one individual with one bubble. The rest had none. After compression, there was an average of six bubbles in each eye examined (range 3-12) and this difference was statistically significant, P<0.001. They clearly demonstrated that before exposure there are very few bubbles indeed and that breathing compressed air increased the number of bubbles.

The origin of these bubbles became the focus of some further experiments, as did the possibility that bubble numbers may be related to decompression stress.

It remains unclear where the bubbles arise. There are a number of possibilities, based on the mechanisms by which the tear film is formed. The tear film is essentially a three layered structure (Figure 2). The innermost layer, close to the globe, is a mucin layer, which is composed of glycocalix and mucus produced by goblet cells on the cornea itself. Close to the globe this layer is quite thick and adherent to the cornea and becomes a more broken up and looser structure as one moves further out. This layer reduces much of the shearing stress. The next and most substantial layer is the aqueous, which is primarily produced in the lacrimal glands. The outermost layer is a thin layer of lipid which is produced by the destruction of Meibomian glands. These glands are very small and numerous, found on the lid margin, close to where we look at the film.

The Meibomian gland cells have a life span of three to four days, during which they swell with lipid. On maturity, they rupture into the ducts and the lipid is extruded to form a lipid layer, which stabilises the tear film. The film is therefore mainly aqueous, but with mucin at the bottom and the lipid layer stabilising it on the top.

Tear film bubble origin

Where the bubbles are formed is still not clear, but the likely possibilities have been discussed. They may evolve directly from the globe or the aqueous humour and this was the original concept used to account for these bubbles in 1992.⁵ It is almost certainly the route by which bubbles under contact lenses are formed. Bubbles could also be derived directly by evolution from the conjunctival lid vasculature. It is argued that this possibility is supported by the observation that closing the eyes for some seconds seems to produce more bubbles than when they are kept open. Thirdly, bubbles could be formed in and secreted by the lacrimal gland. There does not seem to be any particular evidence for or against this possibility.

Alternatively, they could be introduced into the tear film with the rupture of Meibomian gland cells, and this we consider a strong possibility. The Meibomian glands are full of lipid and likely, when saturated, to contain

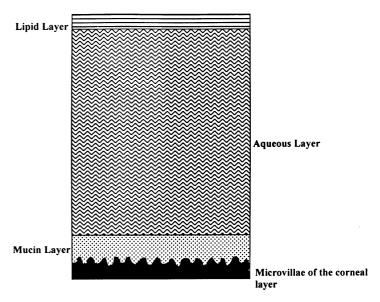


Figure 2. Composition of the tear film.

considerable quantities of dissolved nitrogen. Further, this lipid is introduced to the tear film relatively late compared with the other elements of the tear film. The observed persistence of bubbles in the film for days following an episode of decompression stress may reflect the release of bubble-containing lipid into the film as Meibomian gland cells mature and rupture.

Bubbles and decompression stress

One of the early studies investigating bubble counts to was designed to relate the count to decompression stress. In a study published in 1998, evaluating the decompression stress of a standard diving table, Mekjavic et al. did bubble counts after dives to PADI no-stop limits with increasing bottom times. They took 11 volunteers, subjected them to the compressions indicated in Figure 3 and measured the bubbles immediately on leaving the chamber. They also used a precordial doppler probe to detect any venous bubbles, however none were recorded during the study.

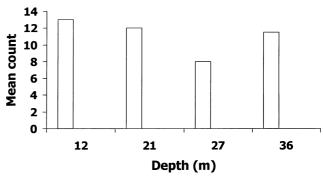


Figure 3. Bubble counts following no-stop limit compressions at increasing depth.⁶

The tear film bubble counts for each profile were essentially the same. The conclusion was that the decompression stress of these dives was roughly similar. As an addendum to that study, the 11 volunteers were compressed for increasing periods at 21 m (Figure 4). The bubble count rose with increasing decompression stress. Checking the statistics, which are not in the paper, there is a significant difference (P < 0.05) between the 15 and the 40 minute dives, but not between the 29 and the 40 minute dives.

About the same time, Morariu and his colleagues were looking at the effect of increasing nitrogen uptake on bubble generation by exercising during a dive. They compressed 9 volunteers to 15 m for 15 minutes and 27 m for 15 minutes, exercising and non-exercising, and compared the bubble counts. The result for the 27 m compression was typical, 2.25 bubbles/subject at rest and 3.75 bubbles/subject after exercise. Mean bubble counts were consistently higher after exercising dives than resting dives, again implying that bubble counts are an index of decompression stress.

In all these studies bubble counts were taken immediately on leaving the chamber after the dive. While such findings are interesting, if bubble counts are to be of any use in the clinical setting, it is necessary to demonstrate that these bubbles persist for some time after diving. This is because frequently there is a significant time delay from diving to presentation. At the Prince of Wales Hospital, for example, the average time from the end of a dive to presentation is well over 24 hours.

To be clinically useful, in fact, the bubble count must persist for several days. In 1997, the first data was published in this area. Six volunteers were compressed on two standard profiles, to 30 m for 15 minutes and 15 m for 180 minutes. The volunteers were examined every 24 hours for three days. Figure 5 shows the counts. It is this data which is used to support the supposition that Meibomian gland secretions are the source of the bubbles. There seemed no really good reason why these bubbles should persist, in essentially constant numbers, for two days after a dive, unless they were being stabilised in some way. Stabilisation within the fat of the Meibomian gland cell seems a likely candidate.

If bubble generation is related to decompression stress, it may follow that if one breathes oxygen rather than air, fewer bubbles will be detected. In 1997 Mekjavic tested this proposition. Eight volunteers, did two simple 8 m, 60 minute chamber compressions, once on air and a week later on oxygen. Their tear films were examined before and after each dive. As one might predict after the air dive there was a significant increase in bubble count immediately after leaving the chamber, but when breathing oxygen there was a non-significant change in the number of bubbles. The pre-compression bubble counts in both groups were 3.0 per

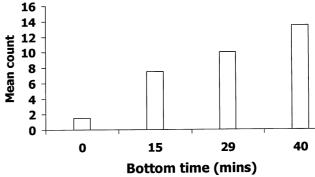


Figure 4. Bubble counts with increasing bottom time at 21m. P<0.05 for 15 minute v 40 minute compression.⁶

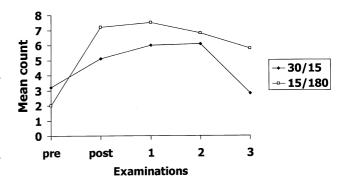


Figure 5. Bubble counts for 3 days following two dry chamber profiles.⁸

subject. Post-compression counts on air were 8.4 per subject and on oxygen 4.0 per subject.

The future

To summarise our current state of knowledge in this area: bubbles can be detected in the tear film following dry chamber diving, they persist for a couple of days and seem related to decompression stress. Counts are increased by a number of things we would expect to increase decompression stress, bottom time, exercise and depth.

Is this observed phenomenon going to be of any use in clinical medicine? To begin to answer this question, a number of interim questions will need to be answered. Does one get the same phenomenon with real (wet) diving? Are counts sufficiently precise a phenomenon to distinguish between people who are really bent and those who are not? Is it going to be a useful measure to check the adequacy of treatment? After treatment and elimination of bubbles, should one re-examine the next day to check whether bubbles have returned, and if so, should one recompress?

At the Prince of Wales Hospital, we are attempting to answer some of these questions. At present, we are interested in confirming that bubbles are detectable after wet dives and are halfway through a planned study in 60 volunteer divers. We are performing bubble counts for three days after a series of standard exposures in the ocean. In the future we intend to repeat that study with multi-day diving and see what sort of numbers we find. In addition, we use this examination for patients presenting with decompression illness to build a prospective data set for future analysis. We can report today that many of these patients have very high bubble counts on presentation, often as many as 20 or 30 on an average sweep.

All of this gives us some hope that finally we may be able to develop an accurate, highly predictive clinical diagnostic test for DCI that can be administered outside the hyperbaric facility and assist in making rational treatment and retrieval decisions. Time will tell, watch this space!

Audience Participation

Rhys Jones, New Zealand

Is it feasible to put slit-lamp microscope into the recompression chamber, and watch the bubbles form in dry dive volunteers?

Mike Bennett

Yes. We have not done it, although there is no reason why we should not. The equipment is on unofficial loan from the ophthalmology department, so we are being gentle with it. But there is no reason that I can see why we cannot do that. However slit-lamp examination is rather uncomfortable. It is a very bright light, but we have persuaded a couple of people to have a fairly lengthy examination. We have detected bubbles emerging from what we are pretty confident are Meibomian gland ducts. We are not experts in using slit-lamps, and these bubbles are notoriously evanescent. The bubble comes out, it appears, it washes down the tear film, and disappears. We attempt to get our ophthalmologists to come and look at them with us. Firstly they are incredulous that one would find bubbles. They say bubbles do not exist in their normal examinations, but of course, they do not usually look for them. When we do get them down and they see the bubbles, they instantly become interested in our discussions about bubbles, and they too feel, given the tear film structure and the nature of the bubble injury, that the Meibomian gland is a likely origin.

Rhys Jones

Have you tried different ascent rates?

Mike Bennett

No.

Richard Moon

When oxygen was given, how did you give it? Was it with a head tent or mouthpiece or mask.

Bennett

That was not our study and the methodology was not reported.

Brubakk

I think they used a mask, but I am not sure.

I would like to make two comments. First, whether this can be used therapeutically, or to know if people have decompression sickness or not. I think that you will find, most likely, the same thing that we have found in vascular gas bubbles, namely that if there are many bubbles, the chance of having decompression illness clinical symptoms clearly increases. It seems that clinical risks are present above Grade 2, or 3 or 4 on the Spencer scale, and with more than 1.5 bubbles per cubic centimetre, if we actually measure bubbles in a vascular system. The more bubbles the more the risk of having clinical symptoms of decompression illness increases dramatically. To put it the other way, with a patient with vague symptoms, in whom you do not find any bubbles, then the likelihood of decompression illness is very low. It is our experience, and most other people's, that if one cannot find any bubbles at all, the diagnosis of decompression sickness is probably wrong. That is not always true, but one can find a lot of people, who have a lot of gas bubbles and no clinical symptoms. I think the major worth of this technique would be in the negative, to say if the symptomatology is dubious and if the exposure is dubious, and we cannot find any gas bubbles, then it is probably not decompression illness.

This has been shown in altitude decompression sickness with vascular bubbles. It is useful for taking out those who do not have decompression sickness.

My second comment relates to the lifetime of these bubbles. That is no surprise at all, because what happens can be shown theoretically and has been shown experimentally. With a significant number of bubbles, they mop up all the gas, and the gas tensions in the surrounding fluids become very, very low, which makes the gradient for removing the excess gas very low. So these bubbles have a very long lifetime. If they have a coating of lipid proteins that also increases diffusion resistance. Where they come from, we do not know, but all these bubbles grow from some sort of nuclei. So there must be a starting point for all these bubbles, they are not created, they grow from something. I think the Meibomian glands are a good candidate.

Mike Bennett

I agree with you on the likely usefulness. Even a highly negatively predictive test would be of use. As you know, we quite often have to transport our divers a long way, on a pretty dubious set of symptoms and a normal examination. A slit-lamp is equipment which is pretty freely available in Australia. One could get a potential candidate for treatment, who is now 36 hours after some pretty

innocent diving with some vague symptoms, competently examined with a slit-lamp and find there are no bubbles. Perhaps we would then be in a position to say "Let us save the country \$30,000, and not fly him down to Sydney."

References

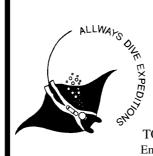
- 1 Boyle R. New pneumatical observations about respiration. *Phil Trans R Soc* 1670; 5: 2035-2056.
- 2 Simon DR and Bradley ME. Cornea edema in divers wearing hard contact lenses. Am J Ophthalmol 1978; 85:462-464.
- 3 Socks JF, Molinari JF and Rowey JL. Rigid gas permeable contact lenses in hyperbaric environments. *Am J Optom Physiol Opt* 1988; 65: 942-945
- 4 Strath RA and Mekjavic IB. Softperm contact lens use for SCUBA diving. *Optometry and Vision Science* 1991; 68 (Suppl): 111
- 5 Strath RA, Morariu GI and Mekjavic IB. Tear film bubble formation after decompression. *Optometry and Vision Science* 1992; 69: 973-975
- 6 Mekjavic IB, Campbell DG, Jaki P and Dovsak PA. Ocular bubble formation as a method of assessing decompression stress. *Undersea Hyper Med* 1998;

- 25 (4): 201-210
- Morariu GI, Strath RA, Lepawsky M and Longley C. Exercise induced post-decompression ocular bubble development. In *Proceedings of the Eleventh International Congress of Hyperbaric Medicine*. Marroni A, Oriani G and Wattel F. Eds. Bologna: Grafica Victoria, 1996; 509-512
- 8 Mekjavic IB, Dovsac P and Kindwall EP. Persistence of tear film bubbles following decompression. In *Proceedings of the XXIII Annual Scientific Meeting of EUBS*. Mekjavic, IB, Tipton MJ and Eiken O. Eds. Ljubljana: Biomed, 1997: 85-87
- 9 Jaki P, Fidler P, Juric P, Dovsak P and Mekjavic IB. The effect of PO₂ on tear film bubble formation. In Proceedings of the XXIII Annual Scientific Meeting of EUBS. Mekjavic, IB, Tipton MJ and Eiken O. Eds. Ljubljana: Biomed, 1997: 88-90

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