THE WORLD AS IT IS

A DIVING DOCTOR'S EXPERIENCE WITH DCS AND PFO

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Key Words

Cardiovascular, case report, decompression illness, medical conditions and problems, recreational diving, treatment.

Introduction

A couple of items in the June 2001 Journal have prompted to me to write with some observations and comments which I trust will at least be of interest if not worthy of further debate.[1,2]

First my credentials. I am a GP in the UK and have a long interest in diving medicine which I inherited from my father (a former GP) who took up diving in 1957. I qualified in diving aged 14 (1965) and have dived each year since. I became a qualified member of the British Cave Diving Group in the early 1980s. I am a HSE approved examiner for professional divers and a medical referee for the UK Sport Diving Medical Advisory committee.

This paper started as a too long *Letter to the Editor* and has been influenced by comments by Dr Bob Wong, co-author of *Modes of presentation of foramen ovale in ten divers*, one of the papers referred to above. Some of Dr Wong's comments appear after this paper, but some, the appropriate DCIEM table stops, have been included in italics after the decompression times in the text.

Case report

I have never been a deep diver (40 m maximum and that only once) and decompression is something imposed on me by circumstance rather that choice.

The situation changed dramatically between 1985 and 1986 and I feel the following narrative from a medically trained observer might be of interest. It has taught me the value of keeping a detailed diving log.

By 1985 20 years incident free diving, using the BSAC/RNPL tables and sometimes close to their limits, had made me somewhat blase. I had always dived with small capacity tanks and I attribute what occurred partly to the longer dive times I could achieve with a 12 litre cylinder! In early May 1985 I dived to 28 m for 23 minutes doing a 4 minute stop at 5 m (*DCIEM tables require 3 minutes at 6 m and 9 minutes at 3 m*). 30 minutess to an hour after the dive I had intense itching over my right biceps preceded by 20 minutes of migrainous fortification symptoms in both eyes. The itching disappeared over 6 hours. There was no rash. I developed a cold the next day and decided this had probably precipitated a mild skin bend.

During the next month I did two more dives, one to 21 m for 40 minutes (*DCIEM tables require 5 minutes at 3 m*) and another to 32 metres for only a few minutes, due to my buddy panicking with narcosis. The headache on that dive I attributed to carbon dioxide retention on descent from fighting the current. It went before surfacing.

Three days after a brief dive to 31 m, with a long period at the end in 6 m, I dived to 23 metres and ascended after 38 minutes, a no-stop dive in the BSAC/RNPL tables (*DCIEM tables require 11 minutes at 3 m*). Unfortunately for the first time in my life I did an uncontrolled ascent from 10 m (dry suit over inflation). Within 10 minutes of surfacing I noticed what can only be described as a flush of chest discomfort followed by a wave of numbness ascending from my feet to the mid chest. At 20 minutes I had paraesthesiae in both legs but could walk and was relieved to find I could empty my bladder. We were within 15 miles of a decompression chamber and in the absence of a phone my wife took me there arriving no more than 1.5 hours after surfacing. The symptoms had regressed completely by then but I opted for a full USN table 6 treatment.

I continued to dive cautiously that season. Nineteen dives later, now in 1986, and, ironically, with another diving physician, after a 30 minute dive to 20 m I again developed an itch over my right biceps, an ache in my shoulders, nausea and, within 40 minutes a florid skin rash. I admit denial, as I concluded the rash was not a skin bend (cutaneous DCS is quite common in the UK) but dry suit pinch. However numbness in the right big toe and over the right knee by the next morning resulted in my taking another trip to the recompression facility. By then it was 18 hours after surfacing. Despite recompression using a helium/oxygen mixture on a Comex table the symptoms were unchanged in the chamber and persisted for another 24 hours before disappearing.

Demoralised at being "bends prone" I nevertheless continued to dive but never below 12 m until I volunteered 3 or so years later to have double contrast echocardiography performed by Dr Peter Wilmshurst. I was shown to have a large PFO.

There is no doubt in my mind that the PFO contributed to my episodes of decompression sickness. My feeling was relief when I found I had a PFO. Before this I was bends prone and really did not know what I could do

about it. Realising that control of bubble development by following conservative tables, using nitrox mixes and controlling my ascent could avert further problems put me back in control of my diving.

After a friendly colleague lent me a copy of the Canadian (DCIEM) tables I continued to dive, albeit defensively, by avoiding dives near no-stop limits, ascending very slowly, doing routine shallow stops and most recently using nitrox. I have no ambitions to dive deep just to continue to admire and photograph the underwater world.

Since then I have completed over 500 dives in a variety of locations with no symptoms after diving. Before my first episode of DCS I had never been deeper than 32 m and then on only a handful of occasions mostly due to lack of opportunity and the unwillingness to consider decompression. Since my episodes of DCS I have done some deeper dives but again on a very limited number of occasions and these have been excursions to about 35 m and once to 40 m. All these dives ended well inside no-stop times with the main body of the dive being in the 10 m zone and, being from the shore, ascent rates were extremely slow. For many years I have spent something like 3 or 4 minutes on average surfacing from my bottom depth of 22 m (most diving in my part of the world is at that depth) with probably something like a minute spent on the last 6 metres or less. This gives an ascent rate of about 5 to 7 m a minute.

I must say that I never consciously measured my ascent rate but usually with a surface marker buoy it is possible to control it quite well. Certainly I feel the precipitating factor in my first treated episode of DCS was the rapid ascent from 10 m to the surface due to an overinflated dry suit. The second treated episode in May 1986 had no obvious precipitating factor with the dive time being 30 minutes at 20 m and a slowish ascent. The dive was curtailed when my buddy (another SPUMS member !) found his watch had malfunctioned and confused my gauge reading with dive time!

Discussion

I think diving practices have a lot to do with the presentation of diving related diseases. Having had the opportunity to dive in the Caribbean, Turkey and the Red Sea I can compare the experience with UK diving. The outstanding difference is the control divers can exert over ascent rate and dive profile outside the UK. Much diving to any significant depth in Britain is done from boats into blue water (not that it is ever that colour here!) wearing dry suits. Dives always end with a direct ascent to the surface. Ascent rates have improved (slowed) over the years but control of speed can be tricky particularly if divers are inexperienced. In many other countries dive profiles allow a slow ascent along a reef and this I think must be a significant factor.

As far as I am aware little research has been done into the incidence of PFOs in a specific diver population. I have unpublished data relating to a questionnaire answered by 75 of the Cave Diving Group (which is about the percentage of current active members in the UK). Seven have had treatment for DCS and another 26 have had symptoms (half of which were skin or CNS) for which they had had no treatment. 10 members have had double contrast echocardiography. Of those 10 there were 5 positives, one treated for DCS, 4 not treated but suspected to have had it in the past and another 5, who had never had symptoms, who were negative.

My next observation relates to the migrainous aura I experienced. I had several episodes unrelated directly to dives in 1986 and they disappeared until last year when I experienced another cluster which seems to have ceased. A MRI scan was entirely normal and anecdotally this would appear to be a common phenomenon which goes largely unreported. Four of the six in our dive boat last week (Sat 23 or Sun 24 June 2001) admitted to such symptoms and none had consulted a physician!

Which leads me to a comment on the study on subclinical decompression sickness on recreational divers. I agree that the numbers are too small to draw any conclusions. My admission that I have had DCS myself, has allowed individuals to "confess" to more than subclinical symptoms for which they have never been treated because they were transient. The most extreme have involved temporary paralysis. I find this quite worrying and perhaps we need to get the message over that transient symptoms probably do need treatment. I refer back to my findings from the Cave Divers Group study.

It is interesting that the 4% figure quoted for many "inside the tables" decompression incidents is so similar to the figure quoted for the presence of significant PFOs in the general population!

Finally I find myself in something of an ethical dilemma when advising other individuals with PFOs. One of the problems we have as medical advisers is that we do not know how much to trust our patients insight into the potential severity of the problem and so we tend to issue blanket bans.

References

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Comment on Dr Glanvill's letter by Dr Bob Wong, co-author of *Modes of presentation of foramen ovale in ten divers*, one of the papers referred to in Dr Glanvill's personal history.

I would like to congratulate Dr Glanvill for putting his own case history into print. He had dived without any incidents for 20 years before finding out that he had a large patent foramen ovale (PFO) . This is consistent with some patients we have seen in Fremantle. It is interesting that since he changed his diving technique, he has been incident free.

On ascent after dives (decompression), bubble formation occurs. This is particularly so after deep dives and with repetitive dives and /or fast ascents. Brubakk has shown in experimental studies that the lung is an excellent filter for gas bubbles, and gas will only pass through if the venous gas load is considerable, increasing the pulmonary arterial pressure by 100-150%. If however, there is a PFO, a pressure increase of only 30% will allow bubbles to pass through.[1]

In my opinion 35 to 40 m is *not* a shallow dive. Pilmanis showed in the 1980s that a 2 minute stop at 3 m (10 ft) after a 25 minute dive (no-stop in USN tables) to 30 m (100 ft) produced a dramatic drop, by more than five fold in the first 15 minutes, in bubble numbers when compared with a direct ascent to the surface.[2] He suggested that short safety stops could be beneficial in reducing the occurrence of "silent bubbles" in divers. It has been shown in goat experiments that decompression can be shortened if the 9m and 6m stops were prolonged and the 3 m omitted.[3] Furthermore, Neuman et al have shown that in humans bubble scores can be dramatically decreased by a deeper decompression stop.[4] I feel that a slow rate of ascent together with a deeper decompression stop would reduce the number of silent bubbles.

Cutaneous decompression sickness (DCS) is an uncommon presentation in Western Australia. However, Wilmshurst has "published data which shows that right to left shunts, usually patent foramen ovale, are associated with certain types of decompression sickness, particularly some types of neurological, cutaneous and cardiorespiratory decompression sickness". Of 60 divers with cutaneous DCS studied by his group, 47 (78%) had a shunt.[5]

The DCIEM Air Decompression Tables only allow shorter no-stop times than the BSAC/RNPL tables.

It is interesting that since he has dived to DCIEM Tables, and by avoiding going near no-stop limits and ascending "very slowly" and doing routine stops, he had clocked over 500 dives without an incident. His slow ascent rate will have minimised the number of bubbles generated.[6]

I agree with Dr Glanvill that "transient" symptoms do need treatment, especially the temporary paralysis. A trial of pressure (USN Table 6) does no harm and could "cure" the symptoms of the diver.

The problems of detecting PFO in divers include that sometimes they have no symptoms, or that the symptoms are so minor they tend to dismiss them. Also symptoms may be transient and ignored. Cerebral symptoms could even have been attributed to the "flu" by divers and doctors alike. It is not easy to obtain statistics on divers with PFO, due perhaps to the fact that most medical practitioners would advise such divers not to dive.

References

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