treatment delay;
lack of first aid oxygen.

However when adjusted for confounders, the only variables that had significance with poor outcome were age over 35, treatment delay and further diving after onset of symptoms.

Seventy-six (29%) of 263 patients were left with residual symptoms. The shallow oxygen tables appeared to be more effective for non-neurological than for neurological DCS and neurological symptoms appeared to be more likely to persist after completion of all treatment. Certainly, he demonstrated a tendency for the residual symptoms to improve spontaneously. One of the conclusions was that the shallow oxygen tables appear adequate for the old Type 1, or non-neurological, symptoms but perhaps we should be seeking different treatments for neurological disease.

The 1990s

I believe that there are different subsets of patients. We see the patients who come in a week after diving with some mild paraesthesia and they just do not feel right. I treat them with Table 6 or an extended Table 6 and it seems to work quite well for them. Another group is patients who come in with serious, progressive, neurological disease and who, despite early surface oxygen, despite early recompression and despite aggressive fluids, do not seem to get better. That produces difficulty for us at the clinical level. The 22 year old who leaves the chamber paraplegic is a treatment failure and a significant burden for the community. My treatment philosophy is that the very acute patients, with progressive disease, who present within 24 hours of their injury and are not resolving at 18 m, are recompressed to 30 m. I use 50:50 heliox at that depth. Why? Why not? We do not know whether it is the right thing to do, but that is what I have elected to do. In the last 5 years, we have had a number of patients who have not done well at 18 m and who have resolved at 30 m. We have also used lignocaine for some of those.

There are divers who dive on open circuit, using trimix (helium, nitrogen and oxygen), to 78 m off Sydney. There was a group of 5 such divers, two of whom have died as a result of this activity. Two of the survivors presented a week apart after one of these deep dives. The first presented with shoulder pain and weakness in his arm. He deteriorated at 18 m, so I took him to 30 m on heliox. He responded very well and was asymptomatic after that treatment and at follow up. His buddy, who presented a week later after diving a very similar profile, had shoulder pain, arm pain, weakness and paraesthesia and was treated by one of our other physicians on an extended USN Table 6. He was very slow to respond and had to have a number of follow up treatments. He was still symptomatic at discharge. This is not statistical evidence, but anecdotally, it influences our decisions.

The Next Millennium

I am not sure what treatments we will be using, however we can only await the outcome of a double-blind randomised multi-centre trial assessing treatment outcome, to help guide our clinical decisions.

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TEN YEARS OF TREATING DIVERS

Chris Acott

Key Words
Accidents, asthma, decompression illness, flying after diving, incidents, sequelae, treatment.

Introduction

I am going to discuss of some of the clinical gems that I have gathered over the past 10 years. I think it was Carl Edmonds who said “How do you know when a diver is lying to you? You just have to watch his lips move.” That

is perfectly true. However, the only advantage of dive computers is that they do not lie.

Rapid ascents

Rapid ascents are good at providing work for diving doctors. They cause bubble disease. They cause morbidity and mortality. From my diving incident monitoring study (DIMS) one of the main causes of rapid ascents is that the diver does not understand his buoyancy jacket. BCs are badly designed ergonomically because the inflate and deflate buttons are next to each other. Often, in an emergency, divers push the wrong button and ascend instead of descend or vice-versa.

We treated one man who loved quick ascents. On most dive profiles he would get up to 8 or 9 m and go straight to the surface. A lot of this was due to the fact that he did not understand his BC. He was a novice diver, who had been sold a buoyancy jacket which floated him face down when he got to the surface. It was a type of BC designed for technical divers, with the buoyancy mainly in the back of the jacket. These jackets, which float the wearer face down, can be described as Darwinian for they occasionally eliminate those who use them. It was a totally inappropriate jacket for him to be using, but he liked novelties, so he bought the most expensive jacket in the shop. It was also unfortunate that he did not understand the inflate and deflate mechanisms. We advised him to retrain before going back to diving.

Denial

Of the divers with decompression illness (DCI) that I have seen, between 10% and 20% had been diving while symptomatic. The result of treatment is not very good when this occurs.

I have often wondered why people do this. I think denial has quite a lot to do with it. Could it be that during diving training they are not taught very much about decompression sickness? I do not think that is a big factor. I think there is ignorance, that they do not realise they have a problem. I have found that it does help to get an accurate history, if one can go through the dive with them step by step and try to work out where "things went wrong". This is particularly useful at follow up, when discussing whether they should give up diving.

Often a diver will come in complaining of minor symptoms, omitting the major symptoms. He will complain of something he associates with decompression sickness and other symptoms are just forgotten or ignored.

Everyone who treats divers talks about denial in divers, and why they do not present promptly for treatment. It is a major cause of delay to treatment. Do divers deny that they have symptoms? Or is it actually part of the disease, organic denial due to cerebral changes?

Recently our neuropsychologist presented me with some articles on organic denial. I am beginning to believe that the denial seen in the majority of divers is actually organic, due to cerebral changes. It is seen in people with right hemisphere strokes.

The reason that I started to think about this was a patient, who came in complaining of elbow pain and dragging his left leg. He actually said "Doc, you have got to fix my left elbow." He kept pointing to his left elbow as he walked. No mention was made of his noticeable limp. I said "Have you got a pain in your left elbow?" and he said "Yes". I said "Well, how long have you been dragging your leg like that?". His reply was "Since I got the pain in my elbow. You have got to fix my elbow". Then I thought that there had to be more to DCI denial than just emotion.

Treatment sequelae

At the medical review after DCI treatment, I always interview the partner. In this way one can find out a lot of things about how the diver has been behaving at home. Has he or she been grumpy? Does he or she go off the deep end a lot quicker than they used to? One can also estimate how the diver is progressing if one sees them at a week, at a month, six months and 12 months.

This story came from seeing a diver whose spouse insisted she be seen as well. She walked in and said "How do you think Joe's going". I said "I think he is doing well clinically. I have examined him. I cannot find anything wrong with him. He says he is fine and he is not having trouble with his memory." or something like that. She said "Well, that is fine Doctor, but can you tell me why he sleeps on the freezer in the garage every night?". One should always interview the spouse!

All our divers are seen by a neuropsychologist, and ours is very good. She understands decompression illness and is very interested in it. But she admits that there is a need for standardisation and validation of these tests. One does not really know how hospitalisation affects performance during these tests nor how any illness will affect the patient's performance. However they can be useful tools.

Returning to diving

After treatments when do we allow diving again? We consider several factors. These include the response to treatment and whether their dive "deserved" the disease they got.

Patients' questions after treatment

How will this affect my future diving? Will I get decompression sickness again? These are common questions. The answers depend on several factors. If we cannot find any problem with their diving we put it down to a chance occurrence and we tell them that they are at no greater risk of getting decompression illness again. But we add the rider, if you do get it, we cannot guarantee that you will get a satisfactory result following treatment.

Often divers ask "Why me?" I explain this by suggesting that he may be the only one admitting to symptoms, particularly if they were diving in a group.

I also explain to divers that DCI is similar to 'flu, it has something to do with the diver's immune system and that the diver was unlucky on that particular day. I use the analogy if one sits in a room with six people and someone is coughing, or has a respiratory tract infection, two or three people in that group will develop symptoms later, and two or three will not. The same applies to DCI, the more one dives, the greater one's chance of being injured. It is just a statistical chance.

Divers always say "I dived within my tables (or computer)". I always think, "Did they?" and look at their profiles. Usually they are denying what they have actually done. I like to look, if I can, at the profile on the dive computer and to compare it with the DCIEM tables.

Then, if all else fails, I tell them how the tables are derived. The majority of diving computers that we use today are derived from Haldane's concepts developed from his goat experiments. Some of the symptoms that Haldane recorded for serious decompression sickness in his goats were, "..the animal was obviously ill and apathetic." and "was not able to move nor be tempted with corn, of which goats are inordinately fond.". It is only a minor exaggeration to claim that if the diving tables or computers were based on the Haldane perfusion model then they are based on the desire of goats for corn.

Asthma and diving

Recently there has been a lot of discussion about asthmatics and diving.

Whether asthmatics are fit to dive keeps on coming up as does their chances of suffering barotrauma.

A factor that is often overlooked, is that the majority of asthmatic divers that I have seen, have had to be rescued on the surface because they became so short of breath that they could not swim back to the boat. Someone else had to jump into the water to rescue them, putting the rescuer at risk.

Are diabetics fit to dive?

I think that if a diabetic wants to dive, then one should dive as the British Sub-Aqua Club do it, with very strict controls. But if a diabetic diver develops decompression illness, he is going to get it from both ends. Hypoglycaemia will affect the outcome. If the diver is hyperglycaemic, to avoid hypoglycaemia during the dive, that too will adversely affect the outcome. We do know that hyperbaric air exposure causes hypoglycaemia in patients who are hyperglycaemic. Dr Orville Cunningham showed this in the 1920s. He used hyperbaric air was to treat diabetics. Hyperbaric oxygen frequently makes diabetics hypoglycaemic.

Flying after diving

When does one allow flying after treatment? This is very important, particularly for tourist divers. We advise 4-6 weeks, but when insurance companies are involved, that is significantly shortened. There are limited data available to us.

At the Diver Emergency Service, we are often rung by divers asking about when can they fly after diving. Frequently the caller dived within the last 24 hours. We reply "We always recommend waiting 24 hours before getting on an aeroplane." The question is repeated, again we reply "We advise you to wait 24 hours." This conversation goes on for about 5 minutes, and is concluded by "You know our number, and where you are going there is a recompression chamber, so you might like to give them a call if you develop symptoms on the flight." I cannot understand why they bother to ring just as they are about to board an aeroplane. Perhaps they expect some magical blessing from the Diver Emergency Service! We actually do get quite a number of calls from Alice Springs because of this! Somebody from the Alice Springs Hospital will ring up and say "I think I have a diver here with symptoms of decompression sickness, who has just flown in from Cairns."

Mask flooding

Mask flooding is a very dangerous event in novices. It is usually associated, particularly in training, with morbidity and maybe mortality. As John Bevan said last year, when diving happiness is a comfortable mask.

Safety stops

It is now built into our diving culture to have a safety stop. I think this is good and it has been shown to decrease bubble count. It is also good for stopping and pausing before you break the surface, because the last 4 m or 5 m of

the dive, where the volume change is largest, is the most dangerous part of the dive.

Unfortunately the safety stop is sometimes used inappropriately. By that I mean that if one has been diving between 10 m and 15 m for 30 to 45 minutes, there is probably no real need to do a safety stop at 5 m, unless to adjust buoyancy, particularly when a diver is low on air. We have treated a few people where this has happened. They were low on air, they signalled and started up. Unfortunately the diving instructor made everyone sit at 5 m for the 5 minute safety stop. The first diver to get low on air, usually a novice, often runs out of air, panics, does a breathhold ascent to the surface and ends up in our chamber with a cerebral arterial gas embolism.

Five minute neurological examinations

Another thing which is creeping into our culture is the on-site neurological examination. This has been published in some books for divers to perform a 5 minute neurological examination on a diver to see whether they have decompression sickness or not. One can probably give one patient with neurological disease to 20 doctors, and they will all find different signs. We have seen divers who had delayed treatment because they went to their dive leader who did a neurological examination and said "I cannot find anything wrong with you", and sent the diver away. I believe that on-site neurological examinations are a hindrance to the diver getting to the appropriate treatment. Some of these 5 minute neurological examinations have been abbreviated even further to what the authors consider to be a standard Romberg test.

Oxygen as first aid treatment

One hundred percent oxygen is the first aid treatment of choice, after securing the airway, breathing and circulation. We teach the Diving Medical Technicians (DMTs) on the courses that we run in Adelaide, DRABC (Diver Rescue, Airway, Breathing and Circulation), give 100% oxygen, and then to think about what is going on. Often when oxygen is used the patient will get better and occasionally they will not want to be evacuated for treatment. The use of surface oxygen has caused delay to evacuation, because a few hours after stopping the oxygen the diver became symptomatic again.

Robyn Walker alluded to someone having 15 hours of continuous oxygen before reaching treatment. That is rare in my experience. Usually I find that divers want to give an air break as soon as the oxygen is started. They will give 20 minutes on oxygen and then a five minute, or longer, air break. We advise giving oxygen for long periods of time, 4 to 5 hours, without air breaks, depending on how long it is going to take to reach the chamber.

Often the data on the use of oxygen does not show the percentage of oxygen given. Neither does it show the interval from the dive to development of symptoms, nor how long it took before the diver was given oxygen, nor how long the oxygen was given for, nor how many air breaks were given, nor the total length of time on oxygen before recompression. This is data that is seldom recorded.

What percentage oxygen to use?

Is it better to give a lower inspired oxygen over a longer period than a higher oxygen for a shorter period? In the 1970s and 1980s, a lot of divers were reported to have received "100% oxygen". But with the equipment they were using, the divers were probably only receiving 50% or 60% oxygen. However, they seemed to lose their symptoms anyway.

Audience participation

Vanessa Haller, Victoria

In the early 80s I used to work in the hyperbaric chamber in Hobart. We mainly dealt with abalone divers. Whether denial is organic or not, we noticed that when we questioned them on the surface after treatment their answers were very different to what to what they said when they were at 18 m (60 ft) on oxygen. For one thing they were a lot more truthful about their dive profiles.

Chris Acott

I agree that one can get two conflicting stories. I have noticed many divers under pressure in the chamber say "I feel like a cloud has been lifted. I can think clearly now". I have often found when trying to talk to divers about when the symptoms developed, that they did nt really know where they had been a few hours before.

Unidentified speaker

If a person develops DCI, is treated and recovers, is he or she likely to be worse affected or more difficult to treat, if DCI recurs ?

Chris Acott

We have only had a couple of patients like that in our chamber. Talking to other diving physicians, they have found in the second case of decompression sickness the outcome has not been good. But there are a lot of compounding factors. Just because a diver develops DCI it does not mean that it will happen again. If the diving profile did not deserve the disease one would have to consider a PFO or some anatomical defect. In that situation, we would advise the diver not to dive again.

Unidentified speaker

But there is no evidence that a second DCI incident would be worse?

Chris Acott

No. We do not really know what is actually happening when we treat patients. What we are arguing about is that perhaps recovery might be neuronal recruitment. If so, divers are knocking off their neuronal reserves with each attack of DCI.

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UNCERTAINTIES IN PREDICTING DECOMPRESSION ILLNESS

David Doolette

Key Words

Bubbles, decompression illness, physiology, research.

Introduction

Decompression tables present a list of ostensibly safe schedules. Divers may expect that dives conducted according to such schedules will be free from decompression illness (DCI) and dives outside the limits will result in DCI. This belief is embodied in Haldane's statement, subsequent to the publication of his decompression tables, "that compressed-air illness has now practically disappeared except in isolated cases where from one cause or another the regulations have not been carried out".[1] The basis for this misconception might be the classification of diving outcome into DCI or no DCI. Using this classification, a particular dive either will or will not result in DCI for an individual. However, the outcome of an identical dive profile may differ for another individual, or the same individual on another occasion. The categorical assertion that decompression schedules distinguish safe (zero risk of DCI) from unsafe dives for the entire population is not only untrue but also impossible. Many commonly used decompression tables have a reasonably low risk of DCI, but any assumption of safety obscures the fact that there will be exceptional incidents of DCI.

Despite his later unequivocal statement, Haldane's original work with goats showed typical biological variability in individual animal susceptibility to DCI.[2] Figure 1 shows some of Haldane's goat data plotted in the form of a dose–response curve. This curve illustrates a low, but finite risk of DCI, following a trivial diving exposure (in this case low exposure pressure) with the risk increasing with the exposure. The exposure where risk rises most rapidly defines the most common limiting exposure for the population. Haldane's (and all subsequent) assertion of safety is based on defining the limiting exposure from a point towards the left of this curve. However, theoretically, there is no point on such dose–response curves where risk is zero.

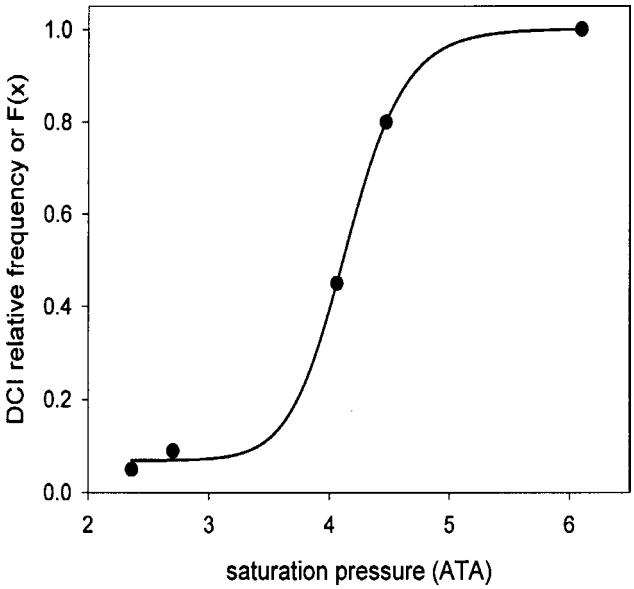

Figure 1. Dose–response curve (cumulative distribution function) for DCI constructed from data tabulated in Haldane's experimental studies with goats.[2] Groups of 4 to 23 goats were exposed to the pressure indicated on the x-axis for 4 hours (3 hours at 4.47 ATA) and decompressed to 1 ATA over 2 to 10 minutes (31 minutes from 6.1 ATA). The circles show the proportion (relative frequency) of goats experiencing any symptoms of DCI. The line is a sigmoid curve, F(x), fitted to the original data.

This paper examines two aspects of uncertainty involved in the prediction of DCI. Firstly, DCI is the result of complex processes that are only superficially evaluated in the decompression theory that underlies decompression tables. Secondly, the main aim is to illustrate that sensitivity to DCI will be normally distributed in a population of divers.

The normal distribution

The sigmoid dose-response curve in figure 1 is derived from an underlying bell-shaped distribution of sensitivity to DCI (see Figure 2). Many biological phenomena conform to a particular bell-shaped distribution called the "normal distribution".

Figure 3 that shows computer simulations of 3 different dice experiments. The distribution of values found