As an analogy, splints may be very valuable for treating people involved in motor vehicle accidents due to speeding. The answer is not to make splints more available, it is to stop speeding.

In a similar way, I would not argue against people using the underwater oxygen technique, when they develop decompression sickness. I would just prefer them not to require the first aid treatment.

C Edmonds

**Key Words**
Letter, decompression illness, treatment, oxygen

### DIVING COMPUTER PROBLEMS

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2/1/96

Dear Editor

It is established practice to collect information about the diving history in cases of decompression illness. Depths, times, surface intervals, numbers of dives per day and numbers of days diving are all recorded. The presumed reason for the collection of this information is to make deductions about nitrogen uptake and elimination, together with adherence or not to the safer diving practices of making subsequent dives shallower and not diving too frequently.

These deductions have been based on the assumption of square profile dives and the relationship between actual dives and the precepts laid down in the various diving tables. Now, with the massive increase in the use of dive computers, the value of the information received has become questionable. Computers are now available for hire on most dive trips, while it is perhaps true that most experienced divers now own computers. The information recorded in the log book is still depth and time, but the depth is a maximum depth and the duration of the dive is well over that possible in a square profile dive. Every dive is a multilevel dive, so that, without interme diate depth and time data, nothing useful can be deduced about nitrogen and the probability of decompression illness. Without knowledge of residual nitrogen, surface intervals become meaningless.

Is it still safer to make dives progressively shallower? For example, the first multilevel dive could consist of a short excursion to 30 m, with the rest of the dive spent mainly at 10 m.

The second could have a maximum of 25 m, then "push the envelope" allowed by that particular computer all the way to the surface. Is this safe diving practice?

It would appear that the logged dive history of a computer diver is of little use when that diver develops decompression illness. We will have to stay in the dark until every bent diver arrives with a computer which can be interrogated by the desktop computer of the doctor. As those dive computers which can be downloaded have different interfaces and incompatible programmes, the waiting may be prolonged.

Tom Fallowfield

**Key Words**
Letter, computers, decompression illness, treatment.

# SPUMS ANNUAL SCIENTIFIC MEETING 1995

### DECOMPRESSION

Fred Bove

**Introduction**

This is a discussion of decompression theory, gas kinetics and tissue uptake to give an idea of the issues related to decompression, diving tables and the basis for the different tables, based on some general concepts one of which is that with increasing ambient pressure there is increased dissolved nitrogen in the tissues.

**Physics**

There are several physical principles which govern the movement of inert gas into and out of tissues, these govern the amount of nitrogen that exists in tissues in the body. Boyle's law, volume is equal to one over pressure multiplied by the constant, deals with the pressure and volume relationship.[1] This is most important at the lower pressure end of the diving spectrum because the rates of changes in volume are the greatest then. Henry's law tells us that the concentration, that is the number of molecules per volume, in a tissue is proportional to the partial

pressure of the gas that is in equilibrium with the tissues. And Dalton's explains the partial pressure relationships.

There are two basic problems. One is that gases go into tissue any time there is an increase in ambient pressure. That never causes problems unless the ambient pressure is quite extreme when one gets cell membrane effects of the inert gases such as narcosis. That is not a solubility issue, but changes in the structure of cell membranes caused by nitrogen. The other problem is getting the gas out of the tissue without damaging the diver as the ambient pressure is reduced.

**Gas uptake and excretion**

One needs to know how much extra nitrogen is present because by figuring out how much is present we can plan how to get rid of the nitrogen safely. The issues are the flow of the gases via the blood into the tissue capillaries and diffusion of gas from the microvasculature into the tissue. These two factors tell us how fast gases will move in and out of different tissues. The other factor is the solubility, that is the amount of gas that the tissue will hold. One can think about the process as filling a bucket. The solubility determines the size of the bucket and the inflow is determined by the size of the hose filling the bucket. The governing mathematics say that the rate of flow into the tissue is proportional to the gradient between the arterial gas concentration and the tissue gas concentration. If there is no differences in concentration, there is no gas flow. And the greater the difference in concentration, the more rapid the gas flow. An increase in pressure gives an increase in concentration of the gas. Henry's law says that if one doubles the pressure there is twice as much dissolved gas in the tissue. Henry's law does indicate that the gas flow takes time. As the pressure difference between the surroundings and the tissues decreases so does the rate at which gas enters the tissues.

Gas uptake follows an exponential curve that is varied by the solubility of the gas in the various tissues. Water and fat have quite different solubilities for nitrogen. Blood is similar to water, but fat has a five or six times greater capacity for nitrogen. Helium has quite different solubility co-efficients so one cannot use an air table to decompress from a dive on helium, one must use a helium table. They are different tables because solubility and diffusibility are different and these factors control the uptake and the elution of gas from tissues.

When one looks at the body as a collection of different tissues, one finds that different tissues allow gas in at different rates.[2,3] The reason being that the flows into tissues are different and the solubility co-efficients, which govern the uptake, are different and some tissue have a larger capacity. For example, the vitreous humour of the eye is a tissue that has essentially no blood supply. So it has almost no uptake of nitrogen when diving, whereas the blood itself takes up nitrogen very quickly because it is in direct contact with the lung alveolar surface and nitrogen is quickly transmitted to the blood. In some experimental air saturation diving by the US Navy, to 18-21 m (60-70 ft), the only problems were bubbles in the vitreous, because decompression tables had not accounted for these very, very slow uptake tissues in the saturation decompression. Very slow tissues need be accounted for but certainly not in sport diving.

The problem for divers is not the gas uptake. The problem is in returning to the one atmosphere environment. The rates of egress of gas from tissues varies, based on the amount of gas that is in the tissues and the different factors which control gas uptake. Some tissues get rid of gas faster than others. It is often assumed that gas washout is a mirror image of the uptake. It has been demonstrated in many studies over the last twenty years that, in anaesthesia and diving, equivalent volumes of inert gases may take longer to come out than to go in. Washout of nitrogen, or any other inert gas, from tissues is different because pressure differentials driving gas out may be lower than those driving gas in. Cold may reduce blood flow through the skin and muscles by vasoconstriction. Exercise and work warm up and vasodilate the muscles, so the warm tissues takes up more nitrogen. The diver may start the ascent with more of a nitrogen load than the tables assume and may develop decompression sickness (DCS). So uptake of nitrogen and the wash out of nitrogen can occur at different rates in the same tissue. Most diving tables are based on the assumption that uptake and washout are mirror images and work well in practice.

**Decompression tables**

JBS Haldane first mentioned the idea that tissue supersaturation could occur and that the degree of supersaturation determined the incidence of decompression sickness.[4] A diver can rapidly change the environmental pressure by reducing the depth. A diver can ascend from 18 m (60 ft) to the surface in a minute or two. The diver cannot get rid of the all the gas that has entered the tissues, as a result of being under pressure, in those one or two minutes. This circumstance produces supersaturation of nitrogen dissolved in the tissues. Supersaturated solutions are unstable. They will begin to precipitate whatever is supersaturated in the solution. With a supersaturated solution of sugar in water, eventually sugar will crystallise out of solution and precipitate leaving a saturated solution. When supersaturated gas precipitates bubbles form in blood and tissues. Bubble production has pathophysiological consequences which are prevented by decompression tables and computers.

Haldane did several experiments to show the effects of exposure to increasing pressure. He used goats as his

experimental animals as they raised the affected limb when they developed DCS. He calculated gas uptakes for different non-anatomical "tissues" assuming that different tissues were gaining nitrogen at different rates. When the dive ended and the goats began to surface, all the tissues had raised nitrogen levels. He found that if he reduced the chamber pressure by halves the goats did not bend. After a period at that pressure the chamber pressure could be halved again without injuring the goats. Haldane proposed that goats and men could tolerate some supersaturation. He suggested that a supersaturation of twice the ambient pressure could be tolerated by the tissues without decompression sickness. He assumed that there would not be bubbles but we know now that is probably not the case.

Haldane proposed that a diver should ascend to a depth that was half the pressure of the tissue nitrogen on the bottom, then wait until the tissues nitrogen pressures came to a point where the ambient pressure could be halved and then move up again. One could come step wise toward the surface never allowing any tissue to exceed that level of supersaturation. This is the idea behind all the diving tables we use. No matter whether one has them on a card or whether one has them in a computer, whether they belong to PADI, the US Navy (USN), DCIEM or the Royal Navy, they are all based on the Haldane concept, that tissues supersaturate, that if we come toward the surface and do not allow supersaturation to exceed a certain ratio, then we will not get bubbles, or we will not get DCS. Those two are not necessarily the same condition.

In the application of this idea, there were a variety of different hypotheses. The group at the Experimental Diving Unit (EDU) in the USN in the sixties came up with M values.[5] M values are numbers that represent the partial pressure of nitrogen in a tissue which could exist upon surfacing and still minimise the risk for bubbles. The Goodman and Workman group at EDU tested human subjects and discovered that there were certain partial pressures of nitrogen that could be tolerated in tissues with an excursion back to 0.8 bar of nitrogen; that is the surface. In fact the ratio was not two to one. Goodman and Workman mathematically modelled six tissues, which do not represent any anatomical parts of the body but only rates at which gas enters and leaves the tissue, and later found, by experiment, that the allowable supersaturations decreased with tissue half time.

Each of the model tissues had a specific partial pressure of nitrogen that would allow one to reach the surface without problems. One could then calculate how much nitrogen was present in each tissue and as long as it did not exceed the M value for each of the tissues one could come to the surface. And if gas partial pressure did exceed any of these values one would have to wait while the nitrogen left the tissues. Once the M values were reached one could come to the surface. This provides a method to create a decompression table.

The computers that we use in diving use the principle of preventing a certain level of supersaturation from being exceeded. Each tissue has a surfacing M value and one can use a computer program to find the tissue that will stop the diver from ascending to the surface. A computer can calculate the time needed to degas sufficiently to ascend. Repetition of these calculations produces a diving table. The PADI tables differ from the USN tables, they were designed independently, and were tested with a small number of dives that were not adequate for true validation, but they are being used. Let us compare the two for a 30 m (100 ft) dive for twenty five minutes, that is the USN no-stop limit, followed by a three hour surface interval and then an 18 m (60 ft) dive for thirty six minutes. This is what the US navy table would allow you. The calculated partial pressures of nitrogen in the ten minute tissue would be just at the M value, but everything else would be below the M value. The 30 m dive for twenty five minutes would allow one to come to the surface because all the calculated nitrogen partial pressures are below the allowable maximum partial pressure before ascending. The PADI tables have a no-stop time of twenty minutes at 30 m (100 ft). They required a five minute safety stop at 15 ft (4.5 m). After a three hour surface interval PADI would allow fifty minutes for an 18 m (60 ft) dive, whereas the USN would only allow thirty six minutes.

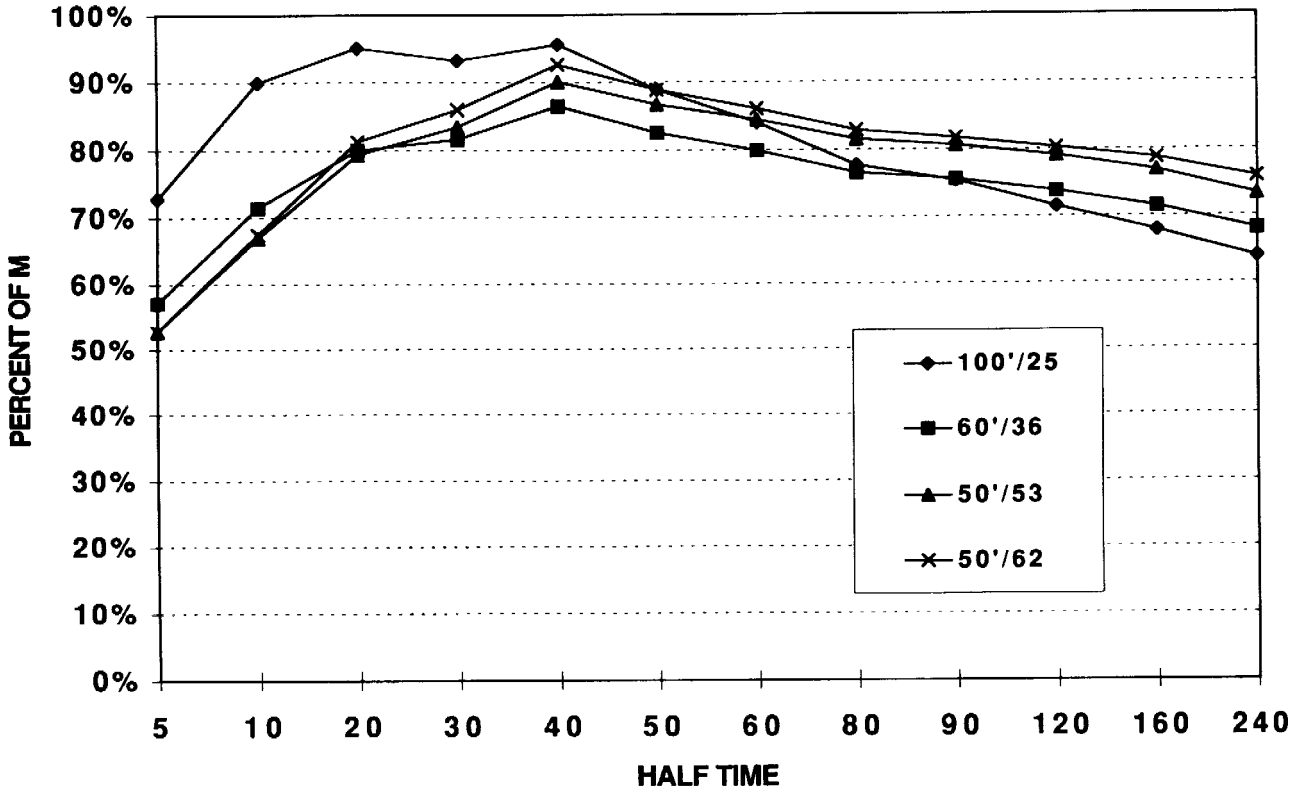
All the numbers are well below the allowable surfacing values on both dives. This is one way to look at a dive table and find out if it is providing the appropriate amount of safety in ascending after a certain kind of dive.

With the first dive and the second dive with the USN table the ten minute tissue, gas partial pressure almost reaches one hundred percent of the M value. The 30 minute tissue and all others are well below the acceptable level in both dives. Using the PADI table the percentages are below a hundred percent on the first dive but the second dive approaches 100% in the ten, twenty, thirty and sixty minute tissues. One need not run every table through a computer program to find this out. In fact that is what the computer is doing during a dive. It checks the pressure and time that one is below an acceptable surfacing value and shows that it is safe to surface. If one has exceeded the M value in one of the tissues the computer will give the decompression stops required.

Figure 1 is a graph of the percentage of the M value for multiple dives with the USN tables, to 30 m (100 ft) for 25 minutes, 18 m (60 ft) for 36 minutes, 15 m (50 ft) for 53 minutes and 15 m (50 ft) for 62 minutes with appropriate surface intervals would not require decompression stops. This would be an acceptable series of four dives based on the navy table. With each dive the long half time tissues move towards one hundred percent of M value. If one does a fifth dive and sixth dive some of these slower tissues become critical, whereas with most sport diving the critical

**FIGURE 1**

**USN TABLES USED TO CALCULATE FOUR NO-STOP REPETITIVE DIVES**




The sequence starts with a 30 m (100 ft) dive to the no-stop limit and continues with the no-stop limit for each dive. A 4.5 m (15 ft) safety stop follows each dive. The curves show percentage of the tissue M value for 12 tissues half times. Long half time tissues increase in saturation in the later dives.

tissues are in the 20, 30 and 40 minute range. With diving all day and accumulating gas, the longer half time tissues begin to become important. This is one of the reasons why the multi-day, multi-dive type of exposures might cause DCS. Some of the early computers did not account for these longer tissues. Now most computers do.

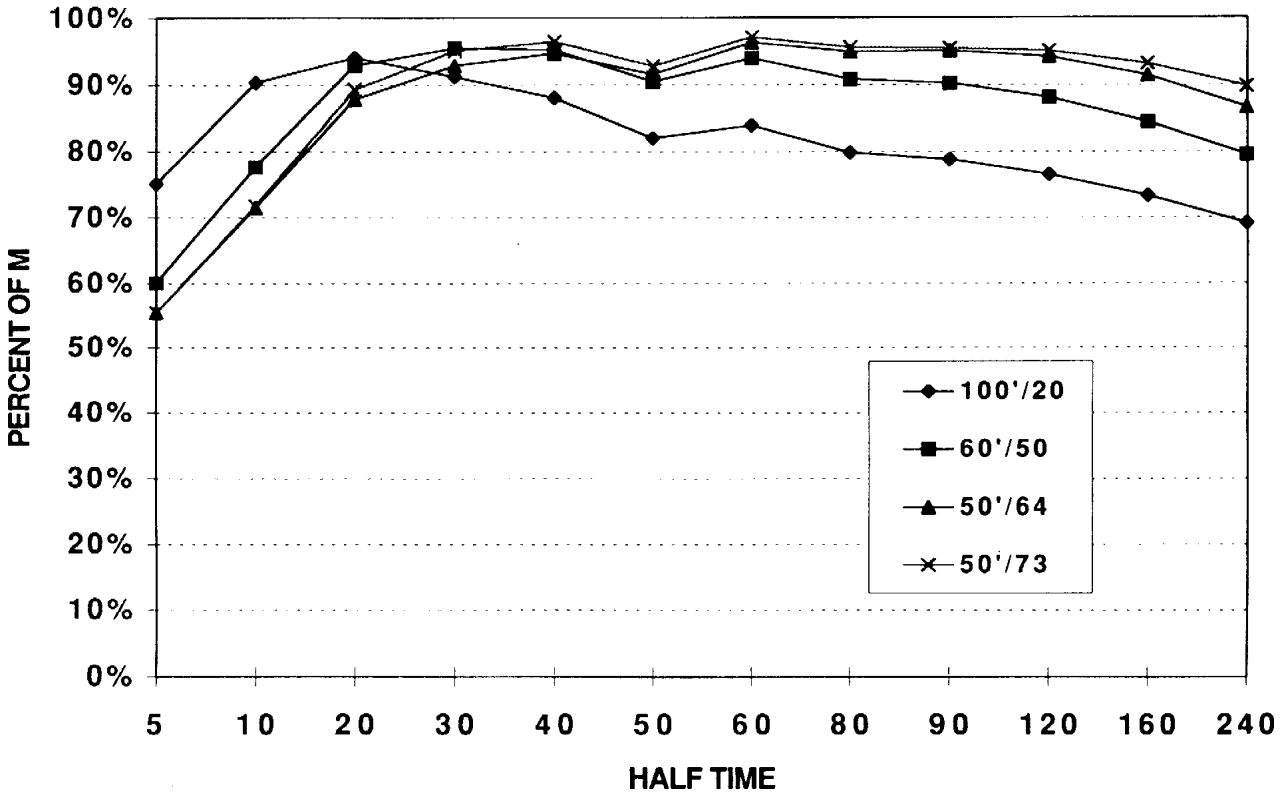
Figure 2 shows the percentage of the M values using the PADI table times for the four dives. The dives become 30 m (100 ft) for 20 minutes, 18 m (60 ft) for 50 minutes, 15 m (50 ft) for 64 minutes and 15 m (50 ft) for 73 minutes. It is clear that 100% of M values is approached earlier in all the slower tissues. It is this multi-dive profile that increases risk for DCS. Doing six dives a day for five or six consecutive days is risky because the longer half life tissues do not completely clear in twenty four hours and adding more nitrogen begins from a higher baseline.

**Problems with tables**

Haldane's group in 1908 worked out the ideas of gas wash in and gas wash out. And they asked whether there were effects that they could not account for with their theory. They asked if there was an independent pressure effect. They exposed goats to a series of different depths, with the time of exposure adjusted so that tissue inert gas loads were about the same, then decompressed the goats on what they thought were appropriate decompression profiles and looked at the number of symptoms in the goats. These were approximately equivalent gas loads, but as the pressure increased there was a higher incidence of DCS. This is one of the concerns with all tables, because even though we do the theoretical calculation, when the pressure is higher, there are factors we cannot account for and there is more risk of DCS. The tables we use and the computers that we use still do not properly account for some of the unknown factors that contribute when one is at the extremes of depth. They also studied the time effects, at a series of depths. All these times and depths were designed to set approximately the same tissue gas concentrations. Yet they found an increasing incidence of DCS in the goats as they expanded the time trying to keep the gas tensions the same. Although excess pressure and excess time are nicely accounted for in the mathematics, whenever we push to extremes, the tables are not going to

**FIGURE 2**

**PADI TABLES USED TO CALCULATE FOUR NO-STOP REPETITIVE DIVES**




The sequence starts with a 30 m (100 ft) dive to the no-stop limit and continues with the no-stop limit for each dive. A 4.5 m (15 ft) safety stop follows each dive. The curves show percentage of the tissue M value for 12 tissues half times. Long half time tissues increase in saturation in the later dives.

be perfect and we must account for those differences empirically.

**Nitrox**

How can we get more bottom time and not accumulate gas in the tissues? Physical laws dictate how much nitrogen one takes up. To reduce nitrogen uptake one can reduce the amount of nitrogen in the breathing gas. To do that one has to the proportion of nitrogen in the compressed air by adding oxygen. Nitrox is now being offered to recreational divers. Nitrox reduces the amount of nitrogen taken up by the tissue because a diver breathes a lower partial pressure of nitrogen. Knowing the oxygen percentage the diver can calculate the equivalent air depth, which is the depth equivalent if a diver was breathing air at the same nitrogen partial pressure. For a thirty percent nitrox mixture (30% oxygen and 70% nitrogen) 30 m (100 ft) is equivalent to about 24 m (80 ft) using air, so a diver can do a 30 m (100 ft) dive and use the 24 m (80 ft) air table to get a bottom time. The USN no-stop limit for 30 m (100 ft) is 25 minutes whereas at 24 m (80 ft) it is 40 minutes. So using 30% nitrox at 30 m (100 ft) allows a 40 minute bottom time. Nitrox was used extensively for mine clearance in shallow waters at the end of the Second World War, and was used in civilian life for commercial divers doing shallow work. Working at 15 to 18 m (50 to 60 ft)if a diver breathes 30% or 40% oxygen there is significant extension of bottom time with minimal decompression risk.

The problem with nitrox is that there is a limit to the partial pressure of oxygen because of acute neurological oxygen toxicity. If a diver breathes oxygen below 1.6 bar the risks of seizures is quite low. If one breathes oxygen above 1.6 bar the risk of seizures is high. Having a fit underwater has a very high risk of causing death by drowning. Using air the partial pressure of oxygen reaches 1.6 bar around 60 m (200 ft). So one could go to 60 m (200 ft) without getting to a dangerous level of oxygen. At that depth one has narcosis which is, in a sense, an intrinsic safety limit. A scuba diver will have very little time at 60 m (200 ft) because gas utilisation will be high, and will never reach the threshold for oxygen toxicity. Using 30% nitrox, when a diver reaches 39 m (130 ft) actual depth the

oxygen partial pressure exceeds 1.6 bar. Breathing 30% nitrox a diver cannot go deeper than 39 m (130 ft) because of the risk of oxygen toxicity. This fact seems to be lost on some nitrox divers who think that with nitrox they can extend their depth without getting extra nitrogen gas load. For example, the equivalent air depth for 30% nitrox at 45 m (150 ft) is 36 m (120 ft) so one would expect more bottom time. The problem is if a diver goes to 45 m (150 ft) with 30% nitrox he is at high risk for an oxygen seizure. With 40% nitrox a diver reaches the toxic level of oxygen at about 21 m (70 ft). With 40% nitrox the equivalent air depth for 30 m (100 ft) is 18 m (60 ft) and one could theoretically do a 30 m (100 ft) dive for 60 minutes with no decompression. Unfortunately a diver is likely to have a seizure long before the 60 minutes are up because he is well over 1.6 bar toxic range. The problem with nitrox is that although it can extend bottom time it adds risk. It can only extend bottom time safely in depths of 18 m (60 ft) or less, where it is quite safe and the $0_2$ partial pressure will not reach seizure levels.

**Saturation**

The other way a diver could extend bottom time is to stay at some depth and saturate the tissues with inert gas. One would then have an excursion range up and down that would not exceed the levels of super saturation needed for bubble formation. The risk of DCS would come when the diver wanted to return to the surface. In commercial deep diving, where there are long jobs to be done, divers reside in a deck chamber at the working depth and travel to the work site in a pressurised bell. At the end of the work they decompress for days in the chamber. Usually the gas is helium and not nitrogen.

Isobaric counter diffusion occurs when one switches breathing gases between mixtures with different inert gases. The phenomenon was first described changing from nitrogen to helium.[6] If a diver is breathing a nitrogen oxygen mixture, with the chamber pressurised with the same mixture and switches to helium by mask without changing the chamber pressure nitrogen comes out of the tissues and helium enters. As helium is more diffusible it enters faster. In a chamber divers may develop skin injury because the high nitrogen level in the chamber prevents nitrogen diffusing from the skin. With helium entering the skin net concentration of inert gas increases and bubbles form in the skin. This can be a problem in commercial diving when divers switch breathing gases. Some switches can produce excess supersaturation without a change in pressure.

**Whales**

Somebody asked me why whales do not get bent. If one takes a standard whale that weighs 100 tons or 90,000 kg, it is about 30 m (100 ft) in length. Lung volume is 5,000 litres. Blood volume is 6,300 litres. The total number of molecules of nitrogen in these different places is calculable based on percentage of body fat and so on. If one does some mathematics, one finds that when a whale dives, and they dive deep for surprisingly long times (up to two hours), the tissues take gas up from the lungs. The whale's lungs can collapse to zero volume because the ribs are disarticulated from the spine. Their trachea and main airways are rigid and do not collapse. As the whale descends, not only are the lungs compressed, but the alveoli are emptied of air so that there is reduced uptake. Residual air is pushed into the respiratory dead space so it does not exchange. There is only a certain amount of nitrogen in the lung, not enough to supersaturate the tissues. So, in a breath hold dive a diving mammal stays out of trouble because there is not enough nitrogen available to saturate the tissues and diffusion from the lungs is reduced.

**Decompression sickness**

When bubbles are present, decompression sickness is present. With small numbers of bubbles the disease is subclinical and is usually unrecognised and not recorded. I will present the classification decompression sickness as Type 1 and Type 2. In the original papers[7] Type 1 was designated as a non-systemic decompression sickness, in the skin or the joints. Type 2 covered all systemic related symptoms. If one breaks down the pathophysiological concepts of DCS into those that affect only the joints or skin and everything else is systemic, clear concept of the pathophysiology can be developed.

There is also an interesting combination of disease which we published a number of years ago. The diver, typically a sport diver, does a dive, has an inert gas load, starts for the surface and sustains an air embolism.[8] The diver has a gas load, with supersaturated tissue, add free bubbles to the body from gas embolism which become foci for growth of excess gas. There results a more severe syndrome with combined pulmonary barotrauma and arterial gas embolism with an inert gas load in the tissues.

Ian Unsworth treated a hundred cases in Sydney.[9] Here are some of his statistics. Forty nine of them had greater than three repetitive dives. Forty four dived to greater than 30 m. This is the depth and time relationship. Thirty eight had exercised immediately after diving. Twenty three missed decompression time and thirty three had other problems like fatigue and equipment failure. Fifteen were in very cold water, fourteen did not record any times, fourteen had out of air emergency ascents, there was a number of inexperienced divers, ethanol was involved in a few, some were flying and one was relying on a computer alone.

**Radiculopathy after diving**

An unusual diving problem is that some lumbar discs have an air pocket in their centres. Radiologists call these vacuum discs. Some cases seem to indicate that a barotrauma like syndrome related to this vacuum space in the disc may aggravate an already present radiculopathy. These spaces obviously take up gas when diving. On ascent when the gas expands, the volume change can cause symptoms of a radiculopathy when a bit of the disc herniates out into the spinal canal. There have been a number of people who have had cervical symptoms or low back symptoms, clearly related to radiculopathy and not spinal cord injury, which is a distinction one can make by careful examination, after diving, usually related to degenerative disc disease. Symptoms of radiculopathy after diving may not be spinal cord DCS, but the equivalent of a Boyle's law effect on some of these vacuum spaces in the discs.

**How to provoke DCS**

Some basic ideas on provoking DCS can be derived from these concepts. Miss decompression stops, do a rapid ascent or go to altitude, dive deeper than 50 m (165 ft), do more than three dives a day, or repetitive deep diving. If your buddy gets bent, and you have been diving together all day, you might get decompression sickness.

**Audience participation**

Knight

Anaesthetists, when I started my anaesthetic training in 1957, had accepted that anaesthetic gases went in quicker than they came out. It is still not fully accepted in the diving community it seems. And that will explain quite a lot of the failures of calculation. Tables are extreme simplifications of an extremely complicated diffusion-perfusion relationship. If one relies on a decompression computer's beautiful algorithm, remember that a lot of the computers have shown that they do not actually dive safely, if one puts them in a chamber they will let one do all sorts of dives that we know bend people.

Bove

No computer that has had clinical testing to prove the algorithm. The PADI tables have had some clinical testing but people argue that not enough dives were done to prove the algorithm. The US Navy did a thousand dives during the last revision of the air tables, which is soon to be published. The work cost ten million dollars and ten years of time. They are obviously going to be used by navy divers but the majority of their value will be for the sport diver. We do not have a lot of tested tables. I am hoping that the navy algorithms will be built into computers so that one set of algorithms for sports divers will be well tested.

Unidentified speaker

It is very interesting that the new US navy tables are going to be very similar to the Canadian DCIEM ones, which I would say is the other major database that was reasonably well tested.

Bove

I agree that the DCIEM tables were well tested.

Molvær

A question about repetitive breathhold diving. We had a colleague visiting our submarine escape tower, or tank, which is 18 m deep. He played around doing repetitive dives to that depth for a day and got neurological decompression sickness.

Bove

It was the Norwegians doing multiple free dives in a submarine escape tank who first discovered that the instructors could get bent. Obviously on a single dive there is not enough inert gas to load the tissues, but it is quite possible with rapid repetitive breath hold diving carried out for a long time. Taravana was described in Tuamotu Islanders, pearl divers who did free dives all day to 24-27 m (80-90 ft), and was in fact decompression sickness.

Molvær

One comment on oxygen. You mentioned acute oxygen toxicity. You did not mention the effect on the lung. It is an aggressive element and we have seen that the diffusion capacity will go down if you bring oxygen partial pressures up.

Bove

Pulmonary oxygen toxicity is a well described and very important issue. It usually is of minor importance in a one to two hour time frame. One sees some decreases in vital capacity at about 60-90 minutes, but that is not clinically important unless one keeps breathing the high partial pressures of oxygen. So in the diving community the most important problem is the acute neurotoxicity of oxygen, not the more chronic pulmonary toxicity. Rebreathers are coming for sport diving and the companies building the rebreathers want to set the partial pressure of oxygen at 1.4 bar. 1.4 bar gives fairly good decompression profiles because of the lower nitrogen, but I think if people swim around for two or three hours with 1.4 bar of oxygen we are going to see pulmonary oxygen toxicity. Every time we try to defy the laws of physics we get into another problem.

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**Key Words**
Decompression illness, tables, nitrox, physiology.

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## NEUROLOGICAL INJURY AND A RETURN TO RECREATIONAL DIVING.

Chris Acott

### Introduction

The development of guidelines for a return to recreational diving following any neurological injury is hindered by the lack of objective data. Furthermore, the available data are limited to commercial or military divers.

A return to diving should depend upon a negative response to all of the following questions:

a Will the continued diving make the condition of the diver worse?
b Will the condition of the diver compromise the diver's or buddy's safety in the water?
c Will the condition of the diver predispose to or aggravate a diving illness?

In addition, if the neurological injury was caused by diving, negative answers must be obtained to the following questions before any consideration of a return to diving can be given:

a Did the diver suffer pulmonary barotrauma?
b Was the diver's illness commensurate with the diving exposure?
c Did the diver respond well to treatment?
d Has the diver any residual problems from the diving injury?

### Decompression induced neurological injury

Permanent neurological damage due to decompression illness has been known for more than a 100 years and while physicians agree that a diver with any objective neurological deficit after a decompression accident is unfit to dive,[1] the suitability of such a diver to return to diving if the deficit "resolves" is debateable. For example animal model data have demonstrated that diving can induce "silent" central nervous system damage.[2] In addition, there is post mortem evidence that lesions may persist in the spinal cord after decompression injury and without clinically evident neurological residua in humans.[3,4] Palmer[5] and Mork[6] in separate studies have also shown a positive correlation between cerebral vasculopathy and diving in post mortem studies of divers with or without a history of decompression injury. These divers studied, as far as it is known, were not incapacitated in any form. Overall, there is a paucity of objective data showing that nervous system damage, that occurs silently (in the absence of both clinical symptoms and signs) after diving and persists, causes any loss of function or impairs activities of daily living. That is, the presence of an abnormality at post mortem does not indicate an inevitable impairment of function. Nevertheless, on the basis of these animal studies, some physicians maintain that any episode of neurological decompression illness permanently disqualifies a diver. This stance may actually delay or suppress the reporting of symptoms, and hence delay treatment, for fear of subsequent disqualification. Although this argument is especially relevant to professional divers, it is still applicable to recreational divers.

The original observation that decompression injury may cause an encephalopathy must be assessed cautiously as the study lacked either suitable controls or established neuropsychological tests and the subjects continued to work in compressed air,[7] it is noteworthy that similar study outcomes are reported. For example, a very recent Norwegian study showed a positive correlation between central nervous system symptoms (problems with concentration, memory, irritability and depression) and un-