and brought to Fremantle Hospital quite quickly. I got the message over the ambulance radio in my car and so arrived at the same time. He was confused and very shaky. His speech was hesitant and respiration was irregular. He had a non-specific loss of sensation from his elbows down and odd feelings of coldness and numbness in his thighs. His haematocrit was a bit raised. There was nothing on his chest X-ray. I considered that he had had a significant arterial gas embolism. The medical registrar on duty felt that it was a whole load of rubbish.

Within fifteen minutes of recompression he was a changed man, completely normal and we had no further problems with him.

The sixth patient was a 25 year old sports diver using a hookah for some crayfish diving. He had spent about an hour and a half at 25 feet and come up as usual on a slow oblique swim up. He was breathing quite normally all the time. At the surface he had chest pain so he drove himself to hospital. It is an excellent hospital about 400 to 500 miles up the coast. When he arrived there he had a pericardial rub. A pneumomediastinum and a pneumo-pericardium were shown on X-ray. There was subcutaneous emphysema in the lower neck. He had no neurological problems of any sort. The doctor there rang me for advice. I suggested that they gave him 100% oxygen for alternate hours, observe him very carefully and if he did not get better to send him to Fremantle. In fact he got completely better and could not understand all the fuss and wanted to dive the next day.

I have no doubt that man has had a tear of his lung substance and has been very lucky not to have had a tear into a blood vessel. I am sure he is at risk if he dives again.

Another air embolism case was a 23 year RAN diver who surfaced rapidly from 15 feet. I believe that he was using an oxygen set. He had two fits as soon as he surfaced. When he was examined he had a left homonymous hemianopia, left sided weakness and proprioceptive deficit. He was clearly confused. He was recompressed to 165 feet on RN table 63. He made a full objective and subjective recovery after about 20 minutes and had no further problems.

These very brief case histories illustrate firstly that all decompression sickness is not necessarily due to diving. Those of us who are involved in recompression treatment should remember this.

Secondly, arterial gas embolism is much more common than we think. In Western Australia we are certainly seeing it more often than we see decompression sickness.

I believe we should be emphasising the dangers of an uncontrolled ascent, even from shallow depths, more during diver training.

AIR EMBOLISM AND CARBON MONOXIDE POISONING

Charles Hackman

At Prince Henry's Hospital we do not have six hour delays between accident and the chamber. Ours are longer, in my first case the delay was 72 hours.

AIR EMBOLISM

Case A

He was an amateur diver who appears to have suffered a severe air embolus while diving with a highly reputable and experienced diving group. However he was not referred for treatment until he finally contacted his general practitioner two days later.

He was a fit 27 year old man who had been diving regularly at weekends for two years. He had dived to 130 feet for twelve minutes. He ascended at '60 feet a minute' and made brief stops at 30 feet and 10 feet before surfacing. So on the face of it he had done things about right.

He then decided to dive again to use up a bit more of his air. He tried standing upside down on the bottom of the dive boat for several minutes to see if he could become disorientated. He claims that he did not. But he became bored, and swam to the surface quite uneventfully.

On reaching the surface he abruptly felt an odd sensation on his left leg. He looked down to see if he had been hit by a shark, but he had not. Everything looked normal but his leg still felt useless although he was able to move it. He swam over to the boat and managed to half pull himself up the ladder before collapsing back into the water, quite helpless. He did not lose consciousness. He was unable to see or communicate in any way. He was dragged into the boat where he was seen again to be hyperventilating vigorously. Shortly afterwards he began to suffer a series of clonic extensor muscle spasms, which were extremely painful, affecting his back, arms and legs. He virtually went into opisthotonos for periods of five minutes at intervals of about 15 minutes. His companions diagnosed hysterical hyperventilation and made him breathe out of a paper bag. Within 30 to 45 his vision began to return and the spasms subsided. By the time the dive boat reached the shore he was able to stand and walk with some assistance, but he had great difficulty controlling his left leg. Soon afterwards, he developed a severe frontal headache, with nausea and vomiting, but these settled in a few hours when he reached his home.

Over the next two days at home he noticed that he was still feeling tired and unwell. He was having difficulty concentrating. He had generalised aches and pains, mainly affecting the muscles that had been involved in the spasms. He also often collided with the furniture, He was unable to position his left arm accurately for tasks requiring co-ordination, he noticed it particularly when he was trying to pour tea. He was quite unable to read consecutive lines of

print. He would get to the end of a line and would not be able to find the beginning of the next line. Finally, presumably after the carpet had become saturated with tea, he consulted his general practitioner. He was referred to Prince Henry's Hospital on the third day after the incident.

On arrival at Prince Henry's he was observed to be very unsteady on his feet and would fall over if he shut his eyes. Movements of his left arm and hand were very clumsy. The examination was suggestive of a cerebellar injury. He had no other signs of neurological damage but he did have an abnormally loud closing sound, pulmonary closure and so we wondered if he had also had some embolism to his lungs. I would be interested to know if this has been seen in other cases of systemic arterial gas embolism.

The differential diagnosis was between a cerebral air embolism from the lungs or possibly some other source, decompression sickness, (although the symptoms had regressed at a time when they should have been worsening), a cerebro-vascular accident, thrombosis or haemorrhage, possibly into a tumour (pretty unlikely), epilepsy precipitated by hyperventilation (possible, but very unusual as he had no previous history of epilepsy of any sort), and finally, hysteria (but this certainly would not explain the pulmonary hypertension, although it could, conceivably, explain all the other findings). There were no other objective neurological findings. The chest X-ray was normal. Because we had already waited 3 days, we decided that we would do a CT scan to exclude some sort of cerebro-vascular accident or tumour. It also was normal.

We decided to recompress him in 100% oxygen at 3 atmospheres absolute (ATA) for 1 hour, then at 2 ATA for 2 hours. If he did get very dramatically better, that would be fine. If he did not, we would send him on to another facility with capabilities for saturation.

In fact, he had very rapid resolution of all his signs and symptoms. However, the following morning, his ataxia had returned very slightly. Although much less than before, it was there. So he had two further similar sessions. These resulted in complete permanent resolution by the fourth day of his stay in hospital, now one week after the accident.

He was reviewed four weeks later when everything was normal, neurologically and cardiologically. He complained of difficulty in writing for the first three weeks after his discharged, but this had now cleared up. He had poor concentration for the first week, several episodes of flushing, and faintness also occurred in the first week. The weekend before being reassessed, he noticed poor co-ordination when he went skiing. He did not notice it any other time, but when skiing he was all over the place. So, clearly, he had suffered some mild, permanent brain damage.

Discussion

The important points about this case are firstly that if he had been treated immediately he would almost certainly not have suffered any significant, permanent, injury. Secondly, that delay can obviously maim or kill a diver who has suffered barotrauma. Thirdly, a point that is not appreciated enough, immediate treatment will not hurt a hysteric. The circumstantial evidence of a recent dive is quite enough to suggest that a diver's illness is dive related. Lastly, despite the dangers of delay, hyperbaric oxygen therapy often does give marked improvement in neurological bends or embolism, even after any bubbles should have dissolved. Of course, it cannot be proved that hyperbaric therapy does more than accelerate an improvement that would have taken place, but it certainly seems to be beneficial.

CARBON MONOXIDE POISONING

We have had two of these cases in the past 18 months. Both were experienced abalone divers over the age of 40. They were both using hookah/demand valve systems with oil free nylon piston compressors. Neither had effective activated charcoal filters. Both were diving in dead calm conditions with known defects in the engine or known faults in the operation of the system.

Case B

The first man was a fifty year old man with no really significant previous history, except for sinusitis and hay fever. He had had an episode of decompression sickness (DCS) some ten years previously. He did a six hour dive, mainly at 60 feet, surfacing briefly every 15 minutes. He may have spent the last one to two hours at 30 to 45 feet, because this was his usual practice, but he had absolutely no recollection of this period. He was using an oil free compressor with dust and water filters on the intake only. He knew that he had a leaking compressor motor exhaust manifold which was close to the compressor inlet manifold. It was dead calm conditions, and he was working close to an overhanging cliff. He had very vague symptoms after surfacing about 5 pm. He came ashore, weighed in his catch, and went home. He showered, according to witnesses, and then, in the shower, two hours after surfacing, he suddenly realised he had no idea how he got to the shower. He did not recall coming ashore. He was totally amnesic for that two hour period. He also realised that he was dizzy and nauseated. He was ataxic. He had blurred vision, myalgia, dysarthria and nominal dysphasia. Eventually he was transferred, via Port Fairy hospital, to Prince Henry's Hospital, by road ambulance with oxygen. There were some subjective improvements in transit. He arrived at Prince Henry's at three o'clock in the morning, 10 hours after leaving the water. By then he was amnesic for the seven hours from 5 pm to midnight. He was slightly nauseated and still had some myalgia. He was noticed to be centrally cyanosed, but when blood gases were done, he did not have low arterial oxygen tension. He also had some decreased power in his quadriceps. This was related to pain. Neurological examination was generally normal.

He was recompressed at 3 ATA for one hour and at 2 ATA for two hours on 100% oxygen. He had some relief of his nausea and his muscle pain. The next morning he felt tired but well and he was only amnesic for the original two hour

period. But later that day he had no recollection of the morning interview. On this occasion some computerised tomography was performed and as usual was normal. However, an EEG showed minor diffused abnormalities.

The diagnosis was that, although he was perfectly entitled to have decompression sickness, he probably had carbon monoxide (CO) poisoning. He was treated with high dose steroids for 72 hours as prophylaxis against possible basal ganglia degeneration. He made a full recovery and returned to work. Over the last 18 months he has not suffered any problems.

Case C

The second case was more recent and was very similar. The diver was a 44 year old man, who also had DCS 10 years previously. By his story, whether one should believe it or not, he had been remarkably good for an abalone diver. He did a four hour dive, with only 30 minutes at 60 feet, one and a half to two hours at 50 feet, 30 minutes at 40 feet and one hour at 15 to 20 feet, with five transient surface breaks. Once again he was using an oil free compressor. He did have an activated charcoal filter, but there was no silica gel filter before it. It was a small, disposable filter cartridge which he changed once a month, if he remembered. He had a new sheller/diving attendant who he had never taken out before. While he was down he noticed that the man was running the main engines for a considerable time while the boat was stationery, and he could not work out why that would be. The man subsequently confessed that he had also kept the compressor running while he was refilling the fuel tank, and on two separate occasions, he had spilt petrol over the compressor. Once again it was dead calm conditions with an oily swell. The diver began to feel unwell in the last hour of his dive. When he got up onto the boat, he breathed some oxygen from a brand new oxygen cylinder that he had there. He did the right thing. However, it was an industrial oxygen cylinder, and there was a very odd smell from it. Later he thought that perhaps the oxygen cylinder was contaminated with another welding gas. At this time he rapidly developed dizziness, nausea, ataxia, blurred vision, tingling in the hands, pain in the right shoulder and in the chest and abdomen. He developed a wide spread itching and a mottled blue skin rash on the trunk which slowly spread out to his limbs. Three hours after leaving the water he arrived at Portland Hospital. By this time, his symptoms and signs had completely cleared except for the rash and the shoulder pain. He was treated with aspirin, steroids, and an antiemetic. A saline drip was put up and he was transferred by road ambulance on 100% oxygen.

We really should not have trusted the story from Portland Hospital because when he arrived he was definitely ataxic. If we had got the story right we probably would not have accepted him in our unit because he would have qualified as a patient who might require more intense recompression than we have available. However, we decided to treat him rather than allow further delay. As usual we did a chest X-ray first. This confirmed the clinical impression that he had chronic obstructive airways disease and quite possibly, some lung cysts. He was treated with hyperbaric oxygen, one hour at 3 ATA and two hours at 2 ATA, with very little change in his symptoms. He was put on steroids. At 7 in the morning, 15 hours after surfacing, he was still slightly ataxic and he still had slight abdominal pain. Forty eight hours after surfacing, he was no longer ataxic but was feeling tired and washed out and was sent home.

He had probably had hydrocarbon poisoning plus or minus CO poisoning, and he may have had decompression sickness as well. Gas samples are, I hope, now being sent to CIG for analysis.

Discussion

People get lulled into a false sense of security by using oil free systems. The protection that they normally provide themselves against oil vapour coming from the compressor is not good protection against other noxious fumes coming into the inlet manifold. All systems should be equipped with effective charcoal absorbers. The compressed gas should be dried before passing through the activated charcoal as water vapour can reduce its capacity to absorb other impurities. If a non-regenerable, disposable system is too expensive to use properly, then an alternative system has to be found. Certainly, there does not seem to be available a cheap, effective monitor to detect impurities in the gas supply. If anyone knows of an available system with sufficient sensitivity, I would be very pleased to hear about it. It is a difficult problem as one is looking for a robust, but quite sensitive, piece of equipment.

These diagnoses are really largely based on circumstantial evidence and they can be challenged. The central cyanosis in the first patient who had a high arterial oxygen tension is quite well described in CO poisoning. The brick red colour applies to corpses only.

Lastly, both of these men, at their age, and particularly the second man with his lung lesions, should be planning retirement from abalone diving. They are not planning any such thing. The Victorian government regulations prevent them from transferring their businesses as abalone licenses are not transferable. This may or may not be a good thing. But it means they cannot sell their businesses. Often abalone divers know no other trade and are trying to save enough money to retire on. They tend to overwork and they cut corners and take considerable risks. I think the attitude of the Victorian government is possibly that expressed by the senior official who I heard quoted recently "We are the fishermen, the divers are just the hooks we use".

A question by Dr Harry Oxer was imperfectly recorded. The answer was properly recorded.

Dr Charles Hackman

We are happy to take people late who we would not have been happy to treat early on when they might need more pressure than we can provide.

Question: Dr Janene Mannerheim

What patients do you intend to treat at Prince Henry's Hospital?

Dr Charles Hackman

Basically, we intend to take people with no neurological deficit, who have not got a serious bend. We take people with niggles and pains. If someone who has got something more serious than that actually arrives on our doorstep, it is because of a communication problem. It may be that we will then decide to treat them anyway. If we do, we put them in our chamber. Partly because of the usually very long time between their coming out of the water and reaching our hospital we are fairly satisfied that they are not going to deteriorate so will not get worse while they are in our chamber. Therefore if we compress them and their symptoms are not largely relieved, we still have the option of contacting another facility and sending them on.

A comment from Dr Peter Laverick about oxygen toxicity, carbon dioxide retention and Royal Navy divers was also imperfectly recorded.

Dr Charles Hackman

I do not worry about pulmonary oxygen toxicity. For completely non-diving reasons, we have to put relatively sick people into hyperbaric oxygen at 2.5 ATA for, in one case, a total of 15 hours in two weeks. Knowing that we were going to do this, we did all sorts of wonderful tests of lung function before and after and found absolutely no change, so we are not really concerned about slight changes.

Central nervous system oxygen toxicity is certainly a problem. I understand that some years ago, one patient actually did fit in the chamber. We normally give them anti-epileptic medication before they go into our chamber. I have had two patients who did develop cerebral oxygen toxicity in the chamber. Certainly in these cases, and I believe in other cases, there were early signs. They do not abruptly go from being perfectly happy and reasonable to suddenly fitting. There is a time during which they either become restless or complain of nausea or of curious sensations and there is plenty of time, in that situation, to drop them down to below 2 ATA in which case you might see the symptoms resolve. We may have been lucky, but we have not had any problems from oxygen toxicity although we have had oxygen toxicity. We usually have one or two people watching them if they are at above 2 ATA. We have a completely transparent chamber so we can observe any restlessness or anything of that nature in which case we drop them back below 2 ATA. There is a very definite difference between someone who is lying completely at rest and someone who has been performing hard, physical exertion, which probably explains the lack of problems.

FIRST STEPS IN FIRST AID FOR DIVING ACCIDENTS

John Knight

First steps in first aid for diving accidents is a message that we all ought to be taking to the divers we talk to so that the people who have diving accidents have a better chance of surviving, because most of the stories we have heard today were mishandled at the first point. A common error is that a doctor who does not know anything about diving accidents says they are hysterical or hyperventilating and that they do not need recompression. I was told quite firmly by the bloke who ran the boat that "everything was all right because we had a doctor on board, and he told us what to do", when he told me about Charles Hackman's air embolism patient. I replied that they story sounded like air embolism and that he should be recompressed if he had any symptoms. Later that evening the patient rang me and I added to the delay. Instead of getting straight onto Charles Hackman late at night I told the patient to see his GP in the morning and ask to be referred to Prince Henry's. I thought that as he had already waited over 48 hours, another 12 would not make much difference as he said that he was getting better. That diver has given up diving and sold his gear.

The chart (figure 1) is a modification of the flow chart Mike Davis presented in Singapore in 1980.(1) I have redrawn it for Australia on the assumption that we had to make it simpler for Australians than for New Zealanders. It is designed for divers to use in an emergency. SPUMS will be producing it on plastic so that it can be taken on a dive. All the diver has to do is find START and follow the arrows asking himself various questions on the way.

DO NOT PANIC is there because panic figures largely in diving accidents. It is no good having a panicking first aider as well as a panicking diver. And then CONTROL MASSIVE EXTERNAL BLEEDING because if a motor boat has run over someone or if a shark has taken a chunk out of him, he will be bleeding well, and he will not survive either affliction unless someone stops his bleeding. SPEED IS VITAL is there to remind the first aider to get on with the job of making sure that the victim has the best possible chance.

The first thing is to ask is "Is he conscious or unconscious?" So I put a question mark after UNCONSCIOUS. If he is unconscious, one must clear the airway and see if he is breathing (BREATHING?). If the victim is not breathing the next step is to give five breaths of expired air resuscitation (EAR) and then feel for the pulse (PULSE?). If he has not got a pulse he needs cardio-pulmonary resuscitation (CPR). Harpur has commented on the failures of CPR in Ontario.(2) I expect Australians have the same problems of failure to establish circulation and fractured ribs. Most doctors are not well trained in CPR nor are most divers. It is something that we ought to train ourselves in, and I brought Resusci Anne with me to remind us that the only way to learn, unless on is a particularly lethal sort of person, is by using a model to practice on.