DECOMPRESSION SICKNESS AND AIR EMBOLISM CASE REPORTS

Ian Unsworth

The first patient is not a diver. She was a 52 year old lady, of 51 kilos, who was admitted for the excision of a large solitary secondary rumour in the posterior quadrant, from an un-differentiated large cell carcinoma in the apex of the left upper lobe. She had presented with cerebellar ataxia. Diagnostic bronchoscopy and CAT scan had been done, which showed what the problem was. She was admitted for a sitting posterior fossa exploration. The pre-medication and induction of the anaesthetic was quite standard. She was monitored after she had been positioned head up on the table, with an arterial line, a right atrial catheter and an end-tidal CO_2 monitor. Induction occurred at 11.30. One hour 30 minutes after induction air was heard to be induced into the venous system, in the posterior fossa. The end expired CO2 fell from 2% to 1%. The blood pressure fell transiently to 60 mm mercury, from a very steady 150. The nitrous oxide was turned off. Halothane in 100% oxygen was turned on. A Valsalva was applied and about 30 mls of air was aspirated from the right atrial catheter. Peripheral vasoconstrictors were used and a large volume of fluids. I was not anaesthetising this patient, so this story is from the anaesthetist concerned.

Full recovery to the status quo was fairly quickly assumed and the operation proceeded. A further 28 minutes later, the air was again heard to be bubbling and hissing into the venous system in the posterior fossa. The end-tidal CO_2 was observed to fall by one half percent to 1.5%. There was not an alteration in the blood pressure, and no further air was aspirated from the right atrial catheter. On neither occasion was there any cardiac arrhythmias. The patient left the operating theatre, with the excision completed, at 14.20.

I happened to be near the recovery ward shortly after that, and I overheard a conversation about two episodes of air embolism in the theatre, so I was rather interested, The recovery room people said, “You might be interested in this case, perhaps you could advise us. What do you think we ought to do?” So I asked “What is the patient like?” They replied “We have given her 0.8 mg of naloxone because we wanted to see what she was like as she has not completely recovered from the anaesthetic and is not very well. She has a dense triplegia and she has not got much movement in her fourth limb either.”

The neurosurgeon was consulted and he agreed that hyperbaric oxygen might help the patient. She was given Decadron 60 mg intravenously.

I was at the hyperbaric unit to receive this lady, and I was absolutely amazed. She was fully conscious, she had no evidence of cerebral involvement whatever, no cranial nerve involvement, her speech was perfect, she had full appreciation of the situation. She was a charming lady. I was able to explain to her in simple layman’s terms what was going to happen to her. She said that it was fine by her, so there was certainly no apparent cerebral involvement. However, she had no movement in either of her legs, and she had no movement of any part of her left arm, and she could only just move the right hand, and wriggle the fingers very slightly. Sensation was apparently normal.

We put her into the chamber some two hours after the termination of the operation. There was some discussion about going deep. I did not. I took her only to 60 feet. I do not take patients off oxygen. I do not give air breaks, except in the very long treatments as in Table 6. It is useful to have the patient off the mask occasionally to have a drink. It also allows proper conversation with the attendant. I seldom have more than two breaks in four hours. I feel that if you interrupt the oxygen with air then you will get less effective nitrogen removal. She had 120 minutes on oxygen at 60 feet. She had intravenous dextran 70, at 125 ml per hour. We decided that Decadron 60 mg 8 hourly might be satisfactory.

Following the first session we found that the only improvement was that there was some slight movement in her right leg. No more than that. Everything else remained as was. So we were not all that optimistic. We put her in the chamber next morning, 14 hours after the first treatment. She had the same 60 feet, 120 minute treatment. After that treatment, we could move her right arm and her right leg on command. I wondered about treating her again. But before I could get around to giving her a third treatment, she improved in an almost miraculous way. She regained all limb movements. She could move all the small muscles of the hands and feet. The day after that not only was she sitting out of bed, but she was able to walk with support. Certainly there was no-one more impressed than the neurosurgeon, who had been talking to the relatives about unfortunate spinal damage, and this sort of thing.

I would like to hypothesise that air entrainment probably occurred during the second episode and that the entrainment was into the vertebral venous plexus, up in the brain stem, and that it crept down and affected the cord. As you how, Fred Bove has done some elegant work with David Elliott and John Hallenbeck many years ago, on the influence of air, admittedly air released during decompression, in the aetiology of spinal decompression sickness. What this lady may well have suffered from was air entrainment into the venous plexus of the cord, or the brain stem, without any cerebral involvement at all. If you do get air into the venous plexus, of course this will result in a brain stem infarct.

At the 1980 Scientific Meeting, John Miller made the point that these cerebral and spinal problems, following neurological procedures or open heart surgery, are often apparently refractive to treatment. You treat them for perhaps 24 hours to 36 hours without success and then improvement occurs. This lady we treated within 2 hours or 2 1/2 hours of entrainment, but still she did not come good on the first treatment.

The next patient was a fit 21 year old male, a sports diver. His diving history was one year's experience of diving. He had full equipment, including a watch, depth gauge, buoyancy compensator, so he was quite well equipped. He had even had a diving medical, from myself as it so happened, the year before. He and three friends decided that they would dive off the heads in a spot known to be 170 feet or more in depth. Just before they got to the dive site, they all ingested very large amounts of Seconal. The idea being that this would be an extra "hit" and the "narcs" would really send them. It did.

On the bottom, after an undetermined time, the patient lost consciousness. He was dragged up by his mates, and dragged backward into the boat. They returned to the shore and this boy was taken to the flat of one of his mates. He remained unconscious for 48 hours. His "mates" thought that he was sleeping off his drugs. When he awoke, he was, to say the least, befuddled. He had paraesthesiae in both legs. He had profound weakness in both legs. He was ataxic. Apparently there was no visual disturbance. His mates decided that they could not look after him any longer and they dumped him in casualty. One of them stayed long enough to give about 30 seconds of history to the nurse in casualty. Then they just beetled off into the night.

So I collected this boy, who spoke very slowly and in a particularly drugged fashion. He was feeling a profound muscle weakness in his legs, paraesthesiae in his legs, severe lower back pain, and paraesthesia in the palm of his left hand. Reflexes were increased in both legs. Interestingly the plantar response was down. He really had to be treated as a spinal decompression sickness. He was given intravenous dextran 40, half a litre in four hours. We had no history of any bladder problems. He did not produce any urine, so perhaps he had made no urine at all in the last 48 to 60 hours. He certainly was rather dehydrated. He had dextran and other fluids, but we did not give him steroids. He was given table 6, the four hour haul, with a very good result. He did take a little time to respond, but once he did, all function returned quite well. By the end of the treatment time he was approximately 99% which was very fortunate.

You might think that was the end of the story. He was thinking that it was. But he developed a reduced air entry and breath sounds at the right base. X-rays showed that he probably had a lower lobe pneumonia. He had a fever and purulent sputum. This cleared up with physiotherapy and antibiotics and it was probably due to lying immobile for 48 hours.

Our drug screen on his urine showed barbiturates, phenobarb, paracetamol, codeine, so he had a good run of the drugs. His past history showed that three years before he went diving, he had been admitted to Prince Henry Hospital suffering from fits secondary to barbiturate withdrawal. He had been on various drugs at that time, including barbiturates, for insomnia. His friends used heroin. A year later, in 1980, he was admitted to Prince Henry again with an overdose of Seconal, and admitted at that time the occasional use of marijuana and heroin sniffing.

We had a happy ending to an unhappy boy. What is the moral here? I believe that this could well be the tip of the iceberg. We have got to admit that there are an unknown number of divers who dive for kicks. We have also to admit that there might well be a number of divers who are on drugs or use drugs, or combine the two. I believe that we have to be firmer. It is difficult how in fact one can screen out addicts, because he did not admit to any drug taking at all when I saw him for his medical. This of course is what drug addicts are going to do.

We have to educate divers that it is foolhardy to use drugs and then dive. I have written in the Medical Journal of Australia that I consider if a diver dies after taking drugs such as narcotics, sedatives or psychotrophics it is suicide, and if his buddy dies then it is murder. Perhaps we have to be more careful in assessing our cases of decompression sickness. I wonder whether we should do urine screens routinely on all cases of all types of decompression sickness and air embolism.

Dr David Brownbill

Fred Bove spoke earlier about the two basic causes of oedema. We ought to extend that to the central nervous system, to both the brain and spinal cord. Steroids for example may well prevent new oedema formation, but will not magically reverse oedema that has already occurred. In the case that Ian Unsworth has described, damage had already occurred. There was central nervous system dysfunction, so that was cell damage. In this case, the patient improved after 24 to 36 hours and one may postulate that oedema had settled down in that time.

This does bring up one point that I would like to make. When people have central nervous system dysfunction either the spinal cord or the brain, where cerebral oedema is a problem, whether it is due to being hit with a 10lb sledge hammer, a stroke, a cerebral metastasis or decompression sickness, the one thing that can do untold harm is the well-meaning bolus of fluid. I appreciate the problem in decompression sickness as a general proposition, that you must keep up the fluid to prevent dehydration. But if there is central nervous system involvement, I would very strongly recommend not to over hydrate. Overhydration may well push the patient into a worse situation.

Dr Fred Bove

This problem of rehydration is always a difficult one to deal with. If the circulation is unstable you have got to rehydrate to get the stable circulation. I think that is the first indication for the need for rehydration. If you had a hypovolaemic shock type of syndrome, you would have to get that blood volume back up to where it belongs. One must restore normal circulation instead of worrying about the central nervous system or the lungs for that matter. The approach that I have always taken is to treat where the problem is most acute and deal with that first. If there is

circulatory instability then you need to treat with fluids. So go ahead and do it. But be ready to deal with possible complications. You have got to take care of one thing at a time.

If the patient is stable and has a normal haematocrit and you are concerned about cerebral oedema, I agree, we ought to hold back on the fluids as much as possible. But one must maintain an appropriate amount of rehydration so that the patient does not suffer from deprivation of fluids.

Dr Ian Unsworth

We all know about the conflict of interest in the intensive care unit between the neurosurgeon who comes in, looks at the patient and says to leave him for half a day and goes away. And then the intensivist comes along and says that those kidneys will never stand that dehydration. Usually there is some sort of compromise, to preserve both the brain and the kidneys.

A CASE OF PULMONARY BAROTRAUMA IN AN ASTHMATIC DIVER

David Clinton-Baker

I am a general practitioner from Wangarai in New Zealand. Last month I was called to the evacuation of a twenty year old, an asthmatic, who suffered from pulmonary barotrauma with air embolism. The story follows on quite nicely from the comments about cerebral oedema.

It was his first deep water dive, the first dive of the day. He spent about eight minutes at 100 feet, seven minutes at 80 feet, and then about fifteen minutes getting up to 50 feet, from where he was seen to make a more rapid than usual ascent to the surface by his two buddies. I have spoken to the patient about this since. He says that at 50 feet he had a bursting sensation in his chest. He also describes burping up air. He remembers getting back to the surface. He remembers, just, getting to the stern of the boat. He remembers nothing after that.

He was pulled semi-conscious into the boat, and he was not breathing. CPR was instituted. The boat took some three hours to reach the coast. On the journey he breathed oxygen enriched air. During this time he had four grand mal fits. I first saw him at the coast when he was semi-conscious. When I asked him his name he responded. Very soon after that, he had another grand mal fit. Examination was unremarkable. His reflexes were normal and there were no neurological deficits at all.

We evacuated him to Auckland, which was an ambulance drive of another three hours. There was a total lapse of six hours from the time of the accident to when he was received at the chamber in Auckland.

As soon as I saw him I started a dextrose saline infusion and gave him 10 mg of Valium IV. During evacuation to Auckland, he had three more grand mal fits, and for the last hour of the evacuation was fitting continually, initially generalised, but towards the end with focal right sided fitting involving the right arm. He was received by Tony Slark at the RNZN Hospital at Devonport. We tried to X-ray his chest but due to the fitting this chest X-ray left something to be desired. However we were almost 100% sure that there was no pneumothorax. He was put into the chamber. He was taken initially to 60 feet on intermittent oxygen. He appeared to be very sensitive to oxygen and did not really respond. So we took him down to 165 feet on air. He spent a total of sixteen hours in the chamber, and was then transferred over to the Department of Critical Care at Auckland Hospital. During the evacuation from the coast to the chamber, I gave him a total of 880 mg of diazapan and Tony gave him a further 60 mg of diazapan with virtually no effect on the fitting.

In the Department of Critical Care at Auckland Hospital, shortly after his arrival, he developed extensor spasms and it was decided to ventilate him to a PCO2 of 30 to 35. His circulation later became unstable requiring infusions of Stabilized Plasma Solution and Lactated Ringers Solution for maintenance of his left atrial pressure, later supported by dopamine and dobutamine. On this regime he stabilized after a septic episode requiring ampicillin, cloxacillin and tobramycin for control. Sedation was continued with phenobarbitone. An early EEG was reported as Grade 3. On the fifth day he was breathing spontaneously and over the ensuing seven days he improved. He was left with a residual right hemi-paresis, which affected his right arm more than his right leg. Over the last three days of his admission he rapidly improved, with increasing power and co-ordination of his right arm. He was discharged home after fourteen days. He had to be re-admitted a fortnight later with further grand mal fits. He was then put onto prophylactic dilantin. When I spoke to him about a fortnight ago, he still had some residual weakness of the right arm, but was improving.

A brief outline of his asthmatic history and how he managed to get into the diving course. He developed a wheeze aged eight. In his early teens he required continuous prednisone, 10 mgs a day, as well as inhaled ventolin and several intramuscular injections of adrenaline, but no hospital admissions for his asthma. The five years or so before this diving accident, his asthma had been quite good, only requiring occasional ventolin and nightly inhaled beclomethasone. He went to his university club about four months ago, asking to be accepted into the diving course. He was referred to his local hospital, which is the largest in New Zealand. He was investigated by a physician there, which involved three hours of interrogation, examination and investigations and was told that he would be fit to dive.

He then went into the diving course, and the dive I have detailed was his first deep water dive.

During the evacuation by ambulance I was giving him fluids, I could not see him improving, so I stopped the