came to mind was his friend who was paralysed, and he thought he was going to be paralysed. He was so hysterical that he went totally blind. He just could not see any more and he just fell on the deck. This sort of blindness is unusual in decompression sickness. Most commonly it is a patchy fog, they see a black fog all over and rarely do you get a complete blindness of this nature. After talking to him we asked him to stand up and walk. The chap just stood up and he was walking around and he was quite happy. We really could not call him a case of decompression sickness so we discharged him. That night we went out to dinner together and that was all the treatment he got. The next day we telephoned the American Embassy and said that the patient was quite well and that we had discharged him. So you can see that all this takes us back again to basics. Watch out for the symptomatology again the clinical examination and the symptomatology. You have to match them up.

Question

Should a patient with air embolism be put in the Trendelenberg position?

Dr John Miller

I do not think it is a tremendously rationalor useful thing to do. Even if there were a lot of gas trapped around the valves it would be the mitral valve that would have bubbles of gas trapped under it and not the tricuspid valve. Elevating the legs might be useful if you are dealing with a venous air embolism. Also it is often very inconvenient to do when you are transporting a patient. However, it is taught fervently to most paramedical personnel and it certainly does no harm. It certainly allows a little bit of extra venous return for somebody who for various reasons has had some impairment of cardiac output. If the people transporting the patient really fervently desire to do this why not let them do it?

But the Trendelenberg position increases the central venous pressure and therefore increases the intracranial pressure. That is going to tend to increase the back pressure which would tend to develop into cerebral oedema. So it may even be a bad thing to do.

Dr Jimmy How

I see it in another way. The moment you have bubbles reaching the brain you can get into a shock situation. If you compare decompression sickness in aviators with decompression sickness in divers you find cerebral effects are commoner in aviators, while strangely enough in divers it is the spinal cord that is more commonly affected with decompression sickness. You can get a shock syndrome when the blood pressure falls rapidly. I suppose that it is due to the micro-damage in the vessels and the effects of anoxia. By and large when you record the blood pressure it is low. I think it would help if we use that position. It would push the blood back to the vital organs. When you have bubble formation it is mainly venous. Venous return is increased when one is in the Trendelenberg position. I think it should be the left lateral as well. This would tend to allow the blood to rush through and the lungs to filter out the bubbles. Someone has said that there is always a small intra-auricular defect even in the adult. This would allow bubbles to reach the arterial blood. So if you allow the bubbles to go freely through the heart the chance of this is reduced. I still teach the medics to put the patient into that position, because it will help in at least these two situations.

Dr Chris Acott

If bubbles reach the right side of the heart the blood flow to the right ventricle will be decreased. So the amount of blood that is going into the left side of the heart will drop. Therefore the cardiac output will drop. That is why you get a drop in blood pressure. Because of the air bubbles trapped in the lung you get an increase in pulmonary vascular resistance. The pulmonary artery pressure is increased. So you get pressure rise in the right ventricle. As 33% of adults have got a patent intraventricular septum you can get air going into the left side of the heart. And it may reach your brain. I think the decrease in blood pressure is due to the decrease in cardiac output. Secondly it may be due to the decreased blood flow to the vasomotor centre of the brain.

Dr John Knight

As a change from the people who have got good chambers I am going to talk about treating decompression sickness without a chamber.

TABLE I

PRESENTING SYMPTOMS OF DECOMPRESSION SICKNESS. US NAVY

From Rivera (1963). US Navy. 900 cases

Cerebral (including inner ear)	6.4%
Spinal	0.2%
Cardiorespiratory	0.4%
Pain only	82.7%
Other	10.3%

If you follow the tables closely, serious decompression sickness has a pretty low incidence, adding up to somewhere about 7% (Table 1). However, if you treat sports divers, who have deep water to dive in, you get a different picture. Edmonds worked in Sydney and Erde in Hawaii. They treated 100 people and somewhere above 50% had serious decompression sickness (Table 2). Depending on where you are situated you are going to get a very different group of people to treat. We are lucky here, we are not likely to get serious decompression sickness because we have not been in any water deeper than about 60 feet so far. So even if we are going to have trouble, we would be very unlikely to get anyone seriously ill with decompression sickness.

TABLE 2

PRESENTING SYMPTOMS OF DECOMPRESSION SICKNESS. SPORTS DIVERS

From Erde & Edmonds (1975). Sports divers. 100 cases.

Cerebral (including inner ear)	33%
Spinal	13%
Both spinal and cerebral	5%
Cardiorespiratory	1%
Pain only	33%
Other	15%

How to avoid decompression sickness ought to be emphasized to divers. Table 3 is a simple collection of information. Most diving boats in Australia do not have a shot rope that will dangle directly beneath the boat. They have a rope and they may tie a tank on it, and it dangles at an angle depending on the currents going under the boat. Some years ago the Royal Australian Navy rescued a helicopter when they should not, as it was in 200 feet of water. They would have done much better to have employed a commercial firm using helium to get it for them. But they felt they had to prove that they could do it. It was in a very current prone place. Their decompressions were done on shot ropes. But the shot ropes were not vertical owing to the current. It is said that every diver got bent during the recovery.

TABLE 3

HOW TO AVOID DECOMPRESSION SICKNESS

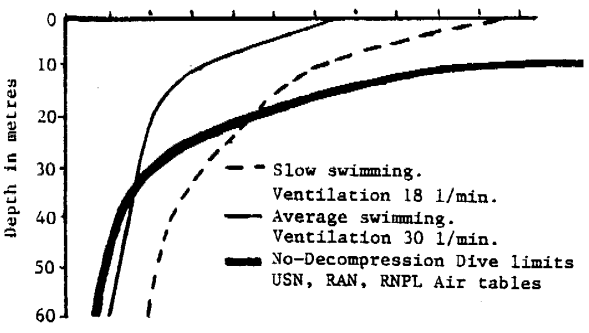
Always do “no decompression” dives
Know the depth of water
Watch the time
Ascent at 60 feet per minute or slower
Do not fly within 12 hours of diving

If you must decompress:
Follow tables
Use a shot rope
Have spare air on the shot rope

FIGURE I

NO-DECOMPRESSION LIMITS AND 72 CU FT CYLINDER ENDURANCE

Time in 10 minute intervals



There is still a myth taught in Australia that you cannot get bent on one tank. In Figure 1 the black line is the no decompression limits of the USN, RAN and RNPL air Tables. They are all within the thickness of that line. If you swim very slowly and do not work too hard you can make your air last to take you outside that envelope from 30 metres downwards (the dotted line). Even if you are working reasonably hard and ventilating at nearly double the rate (the thin line) you can persuade your tank to last you outside the safety envelope at about 35 metres, which explains why I will not go deeper than about 80 feet. Remembering that the safety envelope is not safe for everybody.

This is a story that was published in the British Medical Journal in 1973. A 34 year old diver who dived quite deep on air was a little short on decompression time. He used the usual treatment that divers use for their pain, beer or whisky or some other alcoholic beverage. He turned up at the hospital on an island in the English Channel that did not have a recompression chamber, a little bit outside John Miller’s envelope of 4-6 hours. Actually 15 hours after surfacing. He complained of a pain in his right shoulder, that he was giddy, that he could not walk very well, and that he could not move his right arm. The doctors discovered other things wrong with him. Treatment was rather difficult as he was drunk. They could not get the patient over to England until the next day. They thought that they really ought to try something in the interval. They used Dextran 40 to decrease the sludging and correct the haemoconcentration and stop lipid emboli. They used some heparin to try and slow down the clotting process. They used Mannitol to reduce the cerebral oedema, and they used fructose to sober him up. They gave him aminophylline for the broncho-constriction. Probably the most important thing that they did was to give him oxygen. The point is that he improved with that treatment.

TABLE 4

PROBLEM AND TREATMENT

Problem	Treatment
Bubbles	Reduce size by compression oxygen breathing
Haemoconcentration	Infuse liquids
Circulatory stagnation	Dextran 40 and fluids
Tissue anoxia	Oxygen

When you are away from home and nice chambers you still have got the same problems with decompression sickness (Table 4). You have got bubbles that have got to be squashed. You want to get the gas out of the bubbles and you use oxygen for that. You can rehydrate them by putting liquids in. You can improve the stagnation of the shut down circulation with Dextran 40 and fluids. Ordinary electrolyte fluids are probably just as good. The Dextran 40 does work miraculously in people who have got shutdown areas and it seems to unclump and free the red cells. But you must give them

plenty of fluids with it otherwise you get a jelly in the terminal renal tubules. And you cannot do much about that in the wilds.

TABLE 5

FACTORS AFFECTING SUSCEPTIBILITY

	Previous Dives
Acclimatisation	Age
Obesity	Physical Condition
Alcohol	Exertion
Fatigue	Hydration
Chilling	Intercurrent Illness
Anxiety	Drugs

Table 5 lists the factors affecting susceptibility to decompression sickness. Age and physical condition and hydration all affect us. The morning after the night before is not a good time to go diving but we all do it. Those of us who have not dived for a year, are a little anxious when we fall into the water. Those of us who are diving without adequate protective clothing are prone to get chilled. Those of us who have a headache may have taken aspirin and be improving. Those of us who have got anxieties and are taking Valium do not know what will happen underwater. Alcohol and fatigue and obesity are pretty regular components of a SPUMS meeting. As for acclimatization, those of us who do not dive very often are more at risk than the bloke who is working under pressure every day.

TABLE 6

ADVANTAGES OF OXYGEN THERAPY IN 9 METRES OF WATER

No nitrogen added to tissues during treatment.
Bubbles approximately halved in volume.
Diameter reduced approximately 20%.
Increased tissue oxygenation.
Large nitrogen pressure gradient.
No risk of CNS oxygen toxicity.
No risk of DCS for attendants.
Wet suits still effective insulation.

Now to turn to Carl Edmond's underwater oxygen treatment which is considered to be controversial but is in fact only taking the 60 feet oxygen Tables out of the chamber and popping them in the water at 9 metres (30 feet) (Table 6). It has some advantages, you are not adding nitrogen to the tissues, which you do if you recompress somebody on air. The bubble diameter goes down and there is a large nitrogen pressure gradient while you are breathing oxygen at 9 metres (30 feet). You improve tissue oxygenation. You should have no risk of CNS toxicity. You are certainly not going to bend the attendant. And the wet suit still keeps you reasonably warm. I am not going to say that this treatment is ideal. In fact I do not think it is ideal, but when you are faced with a 12-24 hour delay before getting the patient to a chamber, and he has got to pay for the aircraft, I think it is probably worthwhile considering, if you have got this equipment with you, treating him, because you may cure him and save him that journey.

TABLE 7

REQUIREMENTS FOR EMERGENCY RECOMPRESSION IN WATER USING OXYGEN

Full face mask
Depth limit 9m
Wet suit
Shot rope
Support
Attendant in the water
Communication system

Table 7 shows what you need. The patient needs to have a full face mask so that he can vomit or go unconscious without drowning. The patient must wear protective clothing and insulation, a wet suit. You have got to have a shot rope, so you know where the patient is. You need to have ropes tied to the patient so that he cannot drop deeper than 9 metres. They have got to be supported because trying to stay at one depth in the water is extremely difficult.

The patient must be overweighted. Sitting him in the bight of a rope is a perfectly adequate way of supporting him. But the patient will not thank you. In a trial of the equipment at Truk, Janene got very fed up after about 20 minutes sitting in the bight of the rope. We had weighted her around the waist, so her feet were light and floated up tipping her backwards. Also the bight of the rope cut into her bottom. It will cut in even more uncomfortably if it is tied round the waist. These people are going to be in the water for nearly three hours. You have got to have an attendant down there to make sure that the patient is getting better. It is best to have two attendants. This allows an efficient communication system. The patient can speak into the full face mask and the attendant can hear. A yank on the line to get the second attendant down to take over and the first attendant to take a message back to the surface.

TABLE 8

TREATMENT PLAN

Patient to 9 metres on oxygen (hose length 12m)
Attendant breathing air.
30-90 minutes at 9 metres then ascend at one metre event 12 minutes (or 1 foot every 4 minutes).
If symptoms recur, halt ascent for 30 minutes. If oxygen runs out during the patient to the surface.
Do not give the patient air underwater.
After surfacing give 100% oxygen alternate hours for 12 hours.

All you need on the jetty is a large oxygen cylinder and the yellow box. SPUMS has a back pack that you bolt the oxygen hose to so that the mask will not pull off the patient's face. It is a good idea to have a non-return valve at the bottom of the oxygen line so that if anything goes wrong the face mask does not suddenly fill up with water. The hose is marked off in feet, because the treatment is

extremely simple (Table 8). You take him down to 9 metres. You leave him there for 30 minutes, and then if he is better, you pull him up at the rate of a metre every 12 minutes or a foot every four minutes. Marks which is probably a better method of reducing pressure than coming up in metre steps every 12 to 15 minutes. Marks on the hose are a great help in controlling the ascent.

If he has a neurological bend he gets at least 60 minutes at 9 metres before being brought up. The stay at 9 metres can be safely extended to 90 minutes. The E size cylinder will allow 90 minutes at 9 metres and the 120 minutes of decompression as well as having some left for oxygen at the surface.

FIGURE 2

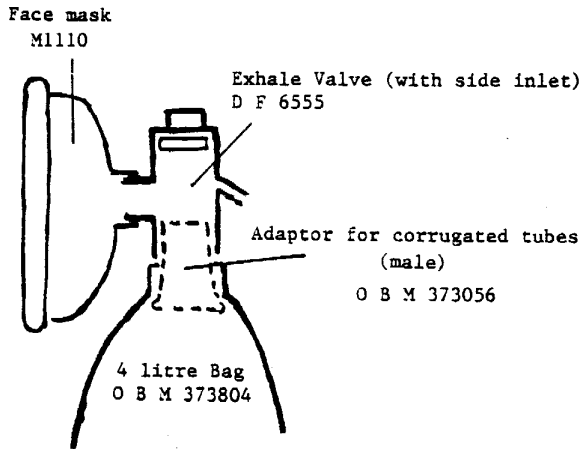
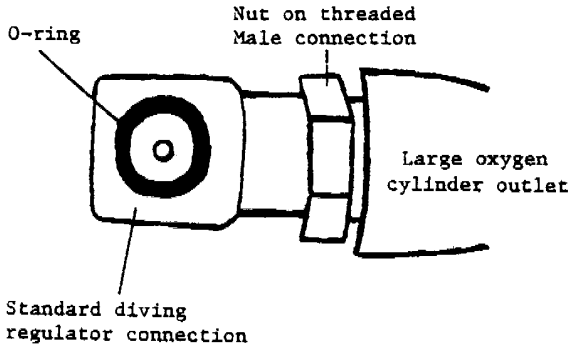


FIGURE 3

ADAPTOR TO FIT A DIVING REGULATOR TO A LARGE OXYGEN CYLINDER



One must continue to give him oxygen once he has been decompressed. For oxygen at the surface one can either use a simple bag and mask set up (Figure 2) made from readily accessible anaesthetic equipment or the diver's regulator as recommended by Mike Davis. To use a regulator with a D or E cylinder one needs an adaptor (Figure 3) to screw into the cylinder outlet in place of the reducing valve used for the underwater treatment. The bag and mask have the advantage that you can watch the bag moving with each breath and see that the chap is breathing properly.

Chairman (Dr Tony Slark)

In New Zealand we recommend that divers besides staying within the no-decompression (no-stops) limits use up the spare air that should remain at the end of a no-stop dive doing a decompression stop. There is often a great deal of interest to be found during the 20 foot level on the cliff face. This stop enables you to use up your gas and will add to your safety margins.

Question

You say that the person can vomit into the mask. How do you get rid of the vomit?

Dr John Knight

It is a full face mask and there is plenty of space to vomit into. After vomiting, you just pull the chin away and the vomit drains out. Because it is a full face mask, if the patient goes unconscious, he will not drown, but I would not use the equipment for an unconscious patient.

Dr John Miller

In spite of the use of a wet suit the water temperature is terribly important. This system should not be used if the water temperature is less than 32-33°C. 2.5 to 3.5 hours in a wet suit in cooler water makes hypothermia during treatment a very real issue. It is probably useful in the equatorial belt. It is not useful in places like the Caribbean. It is not useful in Southern Australia nor in New Zealand waters. It is useful only in this warm water part of the world.

Dr Nick Cooper

Recently a treatment at Heron Island, using the Edmonds Underwater Oxygen Apparatus had to be abandoned after 30 minutes because the patient got too cold to tolerate staying down any longer. So hypothermia is a real risk with this treatment. Water temperature there is about 24°C.

Dr Jimmy How

This problem is very real. You will be hearing a lot about fishermen divers at the conference who are three to four days away out in the South China sea diving to get their fish. When they get bent they take about three to four days to reach Singapore. That is a very real problem and this underwater oxygen treatment could help them tremendously.

Our fishermen divers do almost exactly what is recommended here. With one important difference, they use air. Also they go much deeper. They do wet decompression therapy, and do it very badly. The victim is unconscious, and is lowered in a basket back to 110 feet or to 130 feet. Somebody comes down to accompany him. The saddest part of the whole story is that after he wakes up from his unconsciousness and his paralysis has improved

they winch him right back into paralysis again because they pull him up without any decompression stops. In fact they repeat another dive. We keep on trying to teach about the need for extra decompression on the second dive.

Dr John Miller
To emphasize that some more. The standard way of producing an animal model of serious decompression sickness is to give an animal a dive that is either bends producing or almost so. Then, after a surface interval, recompress to a predetermined depth, stay there for a short while and decompress straight to the surface. That produces a very good spinal cord decompression sickness. An analogue of that sometimes happens in humans. This is one of the things that bothers me about this underwater oxygen therapy. If the treatment has not been completely effective, then it carries with it the potential of turning the pain only bend inadequately treated into a very severe spinal cord injury. That would be another caveat.

Dr John Knight
The instructions that come with the kit say that you take him to 9 metres, 30 feet, and keep him there for half an hour. If he is completely better then you start decompressing him. If be is not completely better, you give him another 30 minutes, and if he has got a spinal problem in the first place, you give him at least an hour, and you can extend that by another 30 minutes if you are worried. One E size cylinder will do all that treatment and still have oxygen left for him to breathe at the surface. I do not know how many times it has been used. I know it has been used in New Guinea, Rabaul and Bougainville. It has been used on patients with neurological bends. Carl Edmonds claims that everyone has been considerably improved and most of them have been rendered symptom free. When you consider that the alternative is a long delay, and then a delayed treatment, I do think that it may well have a part to play.

Dr John Miller
I agree than underwater oxygen therapy can have a place, but within the caveats of the need for a high water temperature and the possibility that if the treatment is not adequate that you could be in a worsening situation.

Chairman (Dr Tony Slark)
This only treats with pressure and oxygen and is not in any way going to help the long term biochemical results of bubble formation. It is possible that relying upon this system at a delayed stage in the patient's management could result in further delays at a time when one should be treating, not bubbles, but what the bubbles have subsequently produced in the blood, the preliminary stage of coagulation coagulopathy.

Dr Jimmy How
I have my reservations about this treatment. I would subscribe to 30 minutes at 9 metres. But I am worried about convulsions if you extend it for another 30 minutes. We train our Navy divers in oxygen sets and I have seen convulsions at 25 feet. The only difference here is that the treatment is free flow, whereas we have a circle system, a closed circuit, for our divers when breathing oxygen. To my mind, if you try to push it for another 30 minutes, the chances are quite high that you will get a convulsion even if you give them free flow.

Dr John Miller

I treat people for 6 hours or more at 2 atmospheres absolute breathing 100% oxygen and they do not convulse. Admittedly they are in a chamber. The important thing is that they are at rest. You are talking about one situation and transposing to another. The working diver is breathing on a closed circuit rig which requires a certain amount of CO_2 to activate the soda lime. It is equipment that has got a fairly high breathing resistance. The effort of working and of breathing against this resistance is quite enough to raise the $PaC0_2$ which lowers the threshold for oxygen convulsions. With the underwater oxygen treatment you have virtually no breathing resistance and the patient is at rest, not working. if the patient were to start shivering, that might be a whole different thing.

Question

Do you regard oxygen therapy as essential in the treatment of decompression sickness?

Dr John Miller

Yes, but not necessarily in the water. Even without recompression you can give fluids and simple things like aspirin. That was the point in my talk about reports from the South of France. I would give the patient a good fluid load and maybe some aspirin. I would do that even when I was going to use this underwater oxygen equipment.

Question

What would you do if you were in the middle of the Coral Sea with a diver with a pain only bend 12 to 14 hours from a chamber?

Dr John Miller

It is quite simple. You give him a good fluid load. I would also give him a couple of aspirin and while I was doing that, I would be having him breathe oxygen as much as possible. By then something like 30 minutes would have gone by. If he was getting better by himself, I would continue fluid and oxygen. If he was not I would put him over the side to soak. There are some advantages in giving him oxygen at the surface. You will need time to examine him, because I want to find out whether or not he has in fact got decompression sickness. And if he has, whether or not he has any neurological complications associated with it. If you have the oxygen treatment equipment on hand, it is awfully easy and awfully tempting to use it when a guy says he has got

a pain in his knee. I think that what you should do is to proceed with due caution, knowing that you have got that as a back up. You can then afford to be thorough.

Chairman (Dr Tony Stark)
I view the pathology of decompression sickness as going through various phases in which different aspects of treatment are most important.

In the first phase it is recompression pressure itself, that is the most important factor. Then hyperbaric oxygen becomes more significant than pressure. Later, the pathology changes further and it may be that hyperbaric oxygen is the only part of the treatment that is necessary at all. Going on further, neither is going to make any difference and the lesions are absolutely permanent.

Other questions were asked and statements made by the audience. Unfortunately, the quality of the recording was not good enough to allow transcription.

SPUMS SCIENTIFIC MEETING 1980

FRIDAY JUNE 27th
DECOMPRESSION SICKNESS SESSION TWO

Dr Chris Acott

Cerebral oedema and the use of barbiturates in the treatment of head injury is one of my interests. I have made the assumption that bubbles in the cerebral circulation causes the same patho-physiological responses as an acute head injury or an acute stroke. If so, the ischaemia, which is the primary neurological damage, is followed by secondary neurological damage. We all know that the most important thing in treating head injuries is not the actual primary insult, but the problem that secondary damage is caused through hypoxia and retention of CO_2 and an unclear airway. The mainstay in the treatment of cerebral decompression sickness or cerebral air embolism is recompressional pressure, but before this can be instituted we have to make the diagnosis. A recompression chamber has to be made ready and available. The diver has to be transported to the recompression chamber. And delay has to be minimized between the diagnosis being made and the diver actually reaching the chamber. In South Australia we have a recompression chamber at the Royal Adelaide Hospital. It is a Vickers monoplace oxygen chamber which I regard as a one man coffin.

In South Australia the majority of diving is done either on the Eyre Peninsula or down the coast from Adelaide or at Mt Gambler. Now Mt Gambier presents a very special problem and this is what stimulated me to think about this. People dive in sink-holes, caves in the limestone where the roof has fallen in to allow access. These are almost all on farms, and so transport and getting facilities to the diver is difficult. If somebody gets bent at Mt Gambler, it is 250 miles to Adelaide and about 250 miles to Melbourne, so there will be delays of up to probably three or four hours.

After Tuesday night's talk, I think we have a very, very good retrieval system in South Australia. An Anaesthetic Registrar, or an ICU Registrar, in his final year, or a Staff Anaesthetist or Staff ICU person goes out. We always go out to the patient and bring the patient back. This is in preference to bringing the patient to us. We have a St Johns Ambulance aircraft on ten minute standby. The pilot has to be within ten minutes of the Adelaide airport. We have a helicopter and a very efficient road system. We take everything with us, including resuscitation equipment and monitoring equipment. We can intubate in the field, we can monitor ECG's, we can put in a CVP, and we can ventilate the patient with a Bird respirator. Here is an example of how efficient our system is. The pupils of a girl, aged 13, who had an extradural haematoma, dilated up while the doctor, 150 miles from Adelaide, was talking to me. We had that girl on the table with her head open within 2 hours.

Because of the physical impracticalities of Mt Gambier, I considered what we should do if some diver came up convulsing and was unconscious. Is there any first aid we can give the diver at the site? And if recompression treatment fails, is there any other treatment that we can give the diver after his treatment? So I began to think about the use of barbiturates in cerebral oedema, which after a delay of about three of four hours is probably the thing that we are treating. You have the primary cerebral insult which is followed by cerebral hypoxia. This can either be global or focal. We know that if we examine divers with bends very closely, you find subtle neurological changes, which means they probably have got focal lesions. The cerebral hypoxia sets up a chain of events which then follows a never-ending circle and ultimately leads to neuronal death and death of the patient.

Catecholamine-mediated hypermetabolism is increased. There is an increase in anaerobic glycolysis which leads to an increase in lactic acidosis. This acidosis can lead to two things. First an inter-cerebral hyperosmolarity and oedema which causes neuronal damage. Also, it leads to reactive hyperaemia and disturbed autoregulation of the cerebral blood flow. Both of these lead to vasogenic oedema. So you get a raised intercranial pressure which itself will decrease cerebral perfusion pressure, and so reduce cerebral perfusion and so you have got secondary damage to the neurones which then completes the vicious circle leading to cerebral hypoxia, and so you get the whole circle starting all over again.

Why do I advocate the use of barbiturates? Experimental evidence, mainly from the United States, in baboons, monkeys and dogs, has shown that experimental animals, subjected to ischaemia, either global or focal, have a 100% survival rate if they are treated with barbiturates, either post or pre-ischaemic episode. The response to post-ischaemia