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

treatment is quite important. Reye's syndrome, which occurs mainly in children following a viral illness, and consists of cerebral oedema, hepatic coma, liver failure and hypoglycaemia had a mortality rate of 75% until people began to use barbiturates in the treatment. Now the mortality rate is zero. In a trial in Pittsburg, in the United States, 14 out of 22 patients who had a cardiac arrest survived when treated with thiopentone after the arrest. The arrest time was between 5 minutes and 22 minutes of cardiac standstill. There is also good evidence coming from the United States about the use of barbiturates in head injury patients. Anaesthetists here know that cerebral air embolism occurs in patients on bypass, and they seem to survive and not have any neuronal damage at all even though they have been shown by Doppler monitoring to have bubbles being lodged in the cerebral circulation. I think that probably the reason is that they were all induced with thiopentone beforehand.

How do the barbiturates work? They supposedly depress the lactic acidosis induced by catecholamine release intracellularly. They also, since 1950, have been known to decrease the cerebral metabolic rate. They decrease the neuronal metabolism. They decrease blood pressure and so decrease cerebral blood flow which becomes quite important in treating cerebral oedema. It is said that they have a membrane stabilizing effect.

If you are going to use barbiturates in head injuries or the sort of situation I have postulated, one has to consider when you have to give them and in what dosage. Experimental evidence suggests that you have to give barbiturates within 3 hours of the primary insult for it to have any effect. So when you read about the inefficiency of barbiturates in the journals, consider when the barbiturates were actually given. They can be given either pre- or post- ischaemia, but they have to be given within 3 hours of the insult and this is quite important. It is also no use deciding 24 hours after all treatment has failed to give the fellow barbiturates. Clinically, the dose varies, however one has to give enough barbiturate to make the EEG on the patient isoelectric. After that you can continue giving barbiturate and it will have no effect- on survival at all. Our regime for head injuries at the Flinders Medical Centre, in Adelaide, is to start with an initial dose of 3 to 15 mg per kg of thiopentone. This dose is tolerated haemodynamically. This is followed up by 1.54 mgs per hour for the next 24 hours. Then we switch over to saline and amylobarbitone 1.54 mg per kg three times a day and this is titrated against the patient's intracranial pressure, as we have a Richmond screw intraventricular drain in place and are monitoring his intracranial pressure.

There are obviously several problems associated with this giving of barbiturates:

1. You need to have haemodynamic monitoring blood pressure, pulse rate etc., even CVP.
2. You have got to have a person who is skilled in resuscitation, so obviously you need an anaesthetist.
3. You need full resuscitation equipment.

The other major thing is that the barbiturates will alter all neurological signs and so you need a careful clinical assessment beforehand, using the Glasgow coma scale and various clinical evaluations of what is going on.

That sort of thing does not really present much of a problem to us in South Australia, because all our retrievals are done by anaesthetists, and we actually do these things in the field without any problems.

Advantages that may be seen for this treatment are:

1. If a diver is convulsing then obviously these convulsions are repressed.
2. If you consider the cerebral compliance curve the stimuli response in intracranial pressure is reduced to a minimum because you had him barbiturized and still and transport him like that.
3. It is thought that using hyperbaric oxygen with the patient barbiturized you will increase his threshold to having cerebral oxygen toxicity.

Question: Dr Bill Hurst
What advantage does barbiturate have over Decadron?

Dr Chris Acott
A lot of people believe that steroids have got no place in the treatment of acute head injuries. They have only got a place in the treatment of cerebral rumours. Patients coming to an operation for removal of a cerebral rumour are often only kept alive by their dose of steroids. It is a debatable point. If I had a head injury I would like everything given to me including steroids if there was any chance that they might do me some good. There are cerebral compliance curve studies where steroids have shown a great decrease in intracranial pressure. It has been similar to that with hyperventilation, barbiturates and mannitol. So if you did not want to use barbiturates in a patient who is convulsing, I feel that you could begin with a dose of steroids. I do not think that you are going to do him any harm at all.

Question: Dr Janene Mannerheim
When you are measuring his neurological signs by examining him, how are you going to do that when he is unconscious and you are transporting him?

Dr Chris Acott

You would use thiopentone initially and then transport him. Theoretically, if you use up to 4 or 5 mg of thiopentone per kg and the transportation getting him to a chamber takes two hours, the patient can be aroused and examined at the end of the journey. It takes a while for the barbiturates, especially this barbiturate, to be redistributed however I

think you could examine him. 4 mg per kg is the standard dose for induction of anaesthesia.

Question: Dr Janene Mannerheim
You do not think you might overdose the patient trying to get a flat EEG?

Dr Chris Acott

That is when you have got them on an EEG in theatre and an EEG in the ICU. What I meant by the isoelectric EEG is the fact that once you have got the EEG isoelectric you do not have to go to any greater dose of thiopentone. I think any dose of thiopentone is worthwhile because it will reduce the cerebral metabolic rate and so reduce the oxygen requirements. If you have got a diver from Mt Gambier who is also cold, you are also adding the advantage of a guy who is hypothermic as well as having barbiturates on board. In South Australia we are the only place in the world at the moment that still uses hypothermia to remove intracerebral rumours.

Question: Dr Tony Slark

You gave us your starting dose, if I have got you right, as 3 to 15 mg per kg. That is an extraordinarily wide range of dosage to start. How do you choose when you are going to give 3 mg or 15 mg ?

Dr Chris Acott

Just on the haemodynamic responses if you give 3 mg per kg and you do not get much of a response you can increase it if you want to. People advocate using up to 15 mg in some cases. Obviously if you hit them with 5 mg or 6 mg and then, as you often do, you get a great decrease in blood pressure and a bit of tachycardia, you will be worried about cerebral perfusion pressure. If you want to get the EEG isoelectric you have to watch the blood pressure as well.

Question: Dr Darrell Wallner
I am just a little bit concerned; how would you ensure that you were also not depressing respiration?

Dr Chris Acott

In South Australia, those who go out to collect patients are all anaesthetists. If necessary we can put down an endotracheal tube and ventilate them which we would do if the respiration was depressed.

Question: Dr Chris Lourey

I can see the rationale of using barbiturates for controlling fitting and the rationale of having the expertise on site. However, I cannot see the rationale of large doses of barbiturates. If you can have an anaesthetist on site who has the skill to place the endotracheal tube, why not completely paralyse the patient, ventilate him with at least 60% oxygen, possibly give steroids, and then when he reaches a hospital with recompression facilities, de-curarise?

Dr Chris Acott

I discussed what we use in head injuries at Flinders Medical Centre and what they are trying to do. I would not like to use relaxants to stop him fitting, as just because somebody is paralysed and cannot move, does not mean that the EEG stops having fits. I agree with you paralyse and ventilate him and take him away. I think that is the way to do it. If you are going to use barbiturates, you are going to have to ventilate the patient. We know that we can reverse all the muscle relaxants once we get him into the chamber. At least you have a secure airway and you are reducing the secondary damage that is going to happen.

Dr John Miller

Oxygen toxicity in this situation with oxygen enrichment during transport, particularly with a barbiturate load on board is unlikely to be a problem. There are situations where repeated oxygen treatments may be given in the chamber where oxygen toxicity will probably ensue. I am pretty simple minded about some of these things. For the most part, pulmonary oxygen toxicity is a reversible lesion, at least functionally, if not pathologically. Pathologically there are significant changes in the lung. The gas exchange returns to normal, particularly at rest. What does it matter, if you impair his exercise capabilities to some extent in order to save somebody's life?

A couple of comments about the use of barbiturates in these cases and a little bit about the differences between cerebral air embolism and central nervous system decompression sickness. Central nervous system decompression sickness is due primarily to obstruction of the venous outflow from the spinal cord. Cerebral decompression sickness is very uncommon by itself. Central nervous system decompression sickness is a global cord lesion. It may be quite high in the cord. In cerebral air embolism you may have one or a series of focal lesions that are arterially mediated throughout the brain and the brain stem. The nature of both is to produce an endothelial injury and that results in oedema either of the cord or of the brain.

For some time now we have also thought about giving patients barbiturates, particularly the ones that have obviously very severe injuries. We have not done so on the following grounds. Recovery and rousability following a single induction dose of thiopentone is rapid. However, although thiopentone is said to be short-acting, it is only short-acting by virtue of the fact that the drug redistributes in the body tissues, and comes therefore to a lower level. So when you give another dose of thiopentone that will also produce a peak with unconsciousness and subsequently redistribution. In order to have a maintenance level at a nice elementary point, the patient may not be rousable. We have avoided using thiopentone because of the fact that we cannot predict beforehand just how rousable a patient

will be after a particular series of doses of thiopentone when we are faced with the situation where the co-operation of the patient may be vital. Theoretically, I am very much in favour of doing it in serious cases of cerebral air embolism, where we have documented a major injury and have made a full clinical evaluation beforehand. Essentially, you are cooling the brain down, reducing cerebral oxygen requirements, reducing the perfusion pressure and at the same time going on with oxygen, steroids and all the other things.

Dr Jimmy How

If you have a diver with a cerebral air embolism, there is obviously going to be tissue damage within the lungs, with air being carried to the brain. He may also have a pneumothorax. I share Janene's worry. I think such a high dosage of thiopentone to a patient could mask useful signs.

We have used very small doses of Valium such as 5 mg and our patients go off to sleep. This introduces two problems when you reduce pressure. With a very drowsy patient who is breathing slowly during the ascent from 165' to 60', you have to keep on rousing the patient, as there is the chance of lung damage from over-expansion if he is breathing very slowly. How serious this is, I am not sure, but we always keep our patients awake. Secondly, if he is drowsy the signs and symptoms of a small pneumothorax getting bigger may be masked. The patient may not be awake enough to complain of pain or difficulty in breathing.

Question: Dr Chris Lourey

Could we have some comments in regard to transport delays such as getting the patient to the RAN chamber in Sydney?

Dr Chris Acott

The time lag is one thing that I am worried about. That is why I was advocating the use of barbiturates, etc. In the extreme case of a person convulsing, to give him a haemodynamically stable dose of thiopentone and put in an endotracheal tube. In South Australia we have intercostal drainage tubes and we know how to put them in. If we put them in, on both sides is necessary, if we have clinical indications.

Once you have given the initial dose of thiopentone you will decrease the cerebral metabolic rate. Then you can paralyse him and take him to Sydney. Those of us who have worked in ICU caring for patients with a Richmond screw intraventricular drainage will have seen the changes in intracranial pressure when somebody is moved and what thiopentone does to it. As an anaesthetic aside, bypass patients all obviously get the same air bubbles but very few have any clinical problems.

You have got everything going for you, you are moving him, he is still, he is paralysed, you are reducing his stimuli and you can get him to the chamber and there is no need for another dose of thiopentone within 2 or 3 hours. To get him moving again you give him atropine and some neostigmine. It all followed from my reading about cerebral oedema.

Dr John Miller

We have treated a number of patients with severe cerebral air embolism following some sort of medical catastrophe rather than diving. They have had significant delays of up to 54 hours between the incident and treatment in the chamber. In most patients where there has been a significant delay, we have found despite very aggressive compression therapy and very aggressive oxygenation, that there is consistently a latent period of about 18 to 30 hours between the onset of treatment and the beginning of an improvement. If one is going to use a barbiturate and recognising that there is a latent period, then why not take the advantage of the cerebral barbiturate right through into that period? Instead of turning off when you get to the chamber, use the chamber as the extra part of the treatment as well as the barbiturate. Then turn the barbiturate off some time just before you would expect to see some improvement, and the patient may well wake up.

Dr Mike Davis

You are in a unique position in Adelaide. You have a group of people who are extremely competent in the basic precepts of resuscitation and maintenance of patients during transfer. That is unique and there are very many areas of the Pacific Basin and America and Europe and a lot of other places where such skills are not available. I think there is a great risk of putting the cart before the horse. Whilst theoretically barbiturates in this situation sound attractive, there are certain riders that I would put on that. Firstly, that there is a great deal more basic education required in the management of airways, fluids, etc. during any form of evacuation. The second thing is the role of thiopentone, as far as I understand it, in the management of severe intracranial conditions, is in the reduction of intracranial pressure. There is no evidence as yet that the use of barbiturates in situations where intracranial pressure is not elevated improves survival. This includes Reye's syndrome, when the primary problem is a raised intracranial pressure which you are treating with thiopentone.

We do not know to what degree the intracranial pressure rises in air embolism. I would be somewhat dubious about the use of barbiturates in a situation where you are not monitoring intracranial pressure. I think that there would be potential risks to advocating that sort of treatment, particularly in the field, when not everybody may have your competence to manage the patient.

Dr Chris Acott

According to a paper in Critical Care Medicine thiopentone will reduce cerebral oedema. They

treated cardiac arrest patients who had 5 minutes to 22 minutes of cardiac standstill. How much cerebral oedema we get with cardiac arrest we do not know, but they used barbiturates. It is a debate that is going on on both sides of the North of the Atlantic. I am one who ardently believes.

Dr Jimmy How

Patients should be transported with a clear airway and adequate oxygenation.

Dr John Miller

Yes, and hyperoxygenation of the patient. Hyperoxygenation is a cerebral vasoconstrictor and a reducer of intracranial pressure.

Question: (unidentified voice)

Do you give the barbiturate all at once?

Dr Chris Acott

No, the slug dose is titrated. You do not draw up a gram and just give it. If you do not get the response you want with 3 mg per kg give more and see what happens.

Chairman (Dr John Knight)

Any more questions on barbiturates, cerebral oedema and so on? If not, I think we will ask Jimmy How to tell us about the diving habits of the people he treats, and why they get bent 4 days out from Singapore.

Dr Jimmy How

I will not talk too much about the fishermen divers because you can hear a great deal of this at the conference in Singapore. Professor Ong is looking after them in terms of the preventive side and Professor Bose of the orthopaedic department after we have finished with them and have sent the patient to the rehabilitation unit.

We see four categories of patients. Firstly, we have professional divers. They come to us because we have good communications in Singapore and we have the chamber here. The cases that come have already been treated and failed.

By and large these cases are due to failure to follow the tables. They are rushing. In an emergency they have to surface, and when they surface their problems start. They also like surface oxygen decompression. They come up to about 40 feet and then from 40 feet they shoot up to the surface. They rush into the chamber and try to be under pressure again within 5 minutes. Then they breathe oxygen just to cut short the decompression time. Many of them have problems at the stage when they leave the water, getting caught up with things as they rush into the chamber. When they reach the chamber some of them start to get the symptoms and signs coming on. They come to us from Brunei, from the Indonesian side and even from the east coast of Malaysia. All these come to Singapore for treatment.

In the second group are the fishermen divers. They are the people you will hear about in Singapore. Their equipment is primitive. They just do not know anything about diving. They do about three or four dives a day. Basically, they are of the older age group. One would think that because they do so many dives a day they would have a tolerance, but this is not really so. When they get a hit, it is just purely because of the type of diving that they are doing. The best way to get their fish is just to throw some dynamite in, “boom” it goes, and then they can take any hookah and go down and choose the size of fish they want. That is exactly what they do at 120 feet out in the South China Sea, about three days away from Singapore.

By and large those who come back to us have gone down to fish as soon as they anchor. They remain underwater for two or three hours and they come up. They have their lunch and down they go again. They repeat the same thing over again. They go on about three dives a day. The amazing thing is that they have been doing this for 10 years and no bends. But lo and behold in the tenth or eleventh year they become completely paralysed.

The sports divers are mostly visitors. They come up here and they get very engrossed in their diving. They put on a second tank and they go in, they put on a third tank and they go in. Some of them do not use the tables properly and decompress less than they should. They too come back to Singapore and give us some problems.

The fourth group we see are divers in the Navy and it is a very small group. They do not get decompression sickness. We have some experimental diving. We have to push them deep. We get some problems. So these are the sorts of cases we have seen in Singapore.

We will hear more in Singapore regarding the fishermen divers, when we will be showing you some slides about these people. The equipment they have is very primitive. They wear a World War II gas mask. Air comes in through the hose and bubbles out at the side of the mask. There is one chap on top controlling the compressor. As they lower the hookah they increase the pressure. Sometimes the compressor stops and no more air reaches the man. They ditch the mask and charge all the way up. Very, very primitive indeed. We are trying to educate these fishermen divers, they are beginning to understand decompression and lately we are receiving fewer cases.

Chairman (Dr John Knight)

Thank you Jimmy, that is a most illuminating description of what I would have called an almost suicidal method of diving. But it is not very different from what the abalone divers were doing in Victoria 10 to 12 years ago. The abalone divers had problems with decompression sickness. I am sure Jimmy can remember very clearly the two men who went out from Eden and did what turned out to be a suicidal series of dives. They involved other