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

fluids after about half of the ambulance journey. I was going to ask about cerebral oedema, but that question has been answered. Of course, this case also raises the problems about asthma and diving even if the asthma appears to be well controlled.

Question:

Why did you diagnose air-embolism?

Dr David Clinton-Baker

I think it was a diagnosis of assumption because he made a rapid ascent from 50 feet. There was no macroscopic evidence of pneumothorax. There was no emphysema. It was assumed that he had an air embolism. He never had any bronchospasm. There was a doctor on the boat, who auscultated his lungs and thought there were sounds on the left base but there was never any evidence of bronchospasm. He had inhaled salbutamol before diving. He had the salbutamol aerosol in his diving bag.

The bursting sensation inside his chest is the last thing that he can remember clearly. He thought that he ascended at a normal rate, but his buddies tried to attract his attention and they could not get any response from him. They both said that he ascended too quickly. But his memory of the accident is obviously quite blurry, and this feeling of an explosion in the chest may have happened after or during the last ascent. His memory is quite vague.

Question:

Should the absence of severe episodes of asthma for the previous three years indicate fitness to dive?

Dr David Clinton-Baker

I am sure that he had had virtually no trouble from his asthma, it had completely resolved by episodic inhalations of salbutamol for about four years.

But after this episode I would ask myself whether anyone who had a history of asthma requiring treatment is fit to dive.

Question:

There is no real evidence that he burst a bulla. Perhaps his asthma was co-incidental. Why did you diagnose an air embolism when he did not have a pneumothorax?

Dr Carl Edmonds

I am worried by the last question. Most cases of cerebral air embolism do NOT have pneumothorax or mediastinal emphysema. Why anyone should use the absence of pneumothorax as an argument against the diagnosis of cerebral air embolism is beyond me.

THE THEORETICAL BASIS OF THE US NAVY AIR DECOMPRESSION TABLES

Bruce Bassett

My topic is decompression. It is the area of physiology that I have spent most time with, starting with aerospace physiology and the problems of decompression of aviators and in later years, twenty years or so, getting involved with the diving side of it. I am going to discuss this and the theoretical basis of the US Navy tables and an analysis of their safety. In this first presentation, I will just cover the theoretical basis. My second talk is a proposed design for sport diver tables, and that is a natural offshoot from the safety analysis of the tables, so those will come together. The final presentation will be about the problems of flying after diving and diving at altitude.

I am going to limit my discussion to the US Navy tables. There is good reason for that, as the tables that we are using here in Madang, however they are laid out as sport diver tables, are all based on this standard. The US Navy tables are probably the most used tables in the world.

Your primary speaker next year is Brian Hills, who has another concept about decompression altogether. So I will be talking about the old historical stuff, while he will be talking about his theory and maybe the twain shall never meet.

US Navy diving tables are a masterpiece of design. They evolved over the years. They represent a cookbook for diving, with recipes for decompression. If you can read, you can follow these schedules. By design they have to be this way, they have to take care of the average layperson diver. You do not have to have a high school education or a university degree to understand them. If you follow the instructions they are pretty easy to use whether they are presented in the original format or in the sport diver format. The numbers are all the same. If you can follow instructions and if you can read, then you can follow the tables for decompression.

I like to go into the theoretical basis for the tables because it is somewhat obscure. Unless you can find somewhere the 1906 Journal of Industrial Hygiene from London, you do not find Haldane's original stuff. Haldane and Priestly published a textbook on respiration up until the 1930's, so you can find a little bit of this up to about then. The US Navy reports on the development of these tables, which was really just an offshoot of Haldane's stuff, are buried in the Experimental Diving Unit Reports, which are classified, and are not sitting around in most medical or other libraries. I will try to present in this paper a little about what Haldane found, why he developed what he did, and what the Navy did with that. In the next paper I will talk about the analysis of how good they are or how poor they are.

I do not like the term no-decompression, but that usage is very common (no-decompression is a dive that you never