Norfolk eleven weeks after his admission. His colostomy closed at a later date with completely satisfactory anal function.
Mr MT was a 58 year old gardener.

History

A fourteen day illness with abdominal pain, diarrhoea. For three days an E Coli urinary tract infection. He had been treated with erythromycin and tetracyclines.

Admission Findings

Septicaemic, distended tender abdomen, creptins over right iliac fossa. Scrotum and perineum oedematous and indurated.

Investigations

WCC 23,0OO
Normal Glucose and renal function
X-ray of the abdomen showed gas in the tissues of the right flank

Management

IV fluids, antibiotics

Day One

Operation

An extensive right ischiorectal lesion was found and widely excised. The abdomen was explored and showed necrotic muscle and fascia, from the perineum, over the abdomen to the lower right ribs. Relieving incisions only were performed.

Cultures from the wounds grew heavy mixed growth of E Coli. Bacteroides fragilis and anaerobic strep. Postoperatively he was continued on antibiotics, penicillin, Gentamycin and metronidazole. IV fluids and Cimetidine were given.

Day One to Four

Twice daily HBO treatments given to a total of six treatments.

Day Four

He was greatly improved being afebrile with a normal WCC. His treatment was continued.

Second Operation

Closure of the defect with Teflon mesh. Postoperatively HBO was resumed and a TPN commenced as well as oral feeding through a nasogastric tube.

Days Six to Ten

Seven further HBO treatments

Day Twenty-Eight

Split skin grafts applied to the teflon mesh which had become filled with granulation tissue. The perineal wound left to granulate with success.

Colostomy closed at a later date with completely satisfactory anal function.

CASE REPORTS

John McKee

I am a general surgeon in Bega on the far south coast of New South Wales and while the amateur divers do not bother me very often, quite a few times over the years I have been asked to give assistance in the treatment of abalone divers who have run into problems. There are forty abalone divers in my area and from time to time they get into real or imagined problems. This year I have dealt with two cases of possible decompression sickness.

The first was a 28 year old abalone diver. He had the usual story of severe pain in one shoulder and arm. He had been diving for seven years. He used decompression stops very rarely. He hardly ever used a depth gauge. He said that he spent the last half hour or so in shallower water than earlier in the day. That can mean anything.

On the day in question he had done three dives. The first to 90 feet for 45 minutes. He then came up and emptied his abalone shells into the boat. After a surface interval of five minutes he dived to 90 feet for 90 minutes. He then came up to the surface again and unloaded his bag. This time he extended his surface interval to 10 minutes before he went down to 60 feet for 30 minutes. When coming up, before he reached the surface, he experienced severe pain in his left arm and shoulder. When he arrived at the hospital I estimated that he had a limb bend of moderate severity. We gave him oxygen to breathe all night. At this stage I was contemplating whether or not to try to send him by helicopter to Sydney. His general condition was quite satisfactory. He had no other evidence of decompression sickness. By the next morning his pain was a lot better, so he was kept on oxygen for another 24 hours and the pain completely resolved. The X-rays of both shoulders showed no abnormality.

The second case was 10 years older and perhaps a little wiser. He had dived for quite a few years as an abalone diver. He also was brought to the hospital at night with suspected decompression sickness. He gave a history of repeated dives to no more than 70 feet that day. The first dive was to 70 feet for at least 90 minutes and the subsequent six to eight dives were at shallower depths but for similar lengths of time. When coming back to shore he felt discomfort in both legs, a feeling of numbness. By the time

he reached hospital he had developed rigors and a high temperature. We kept him under observation while giving him oxygen. Overnight he completely recovered from his symptoms and walked out of hospital the following day.

Then I felt that the correct diagnosis was a virus infection with a high temperature and discomfort with paraesthesia in both limbs.

The third case is one which I think could be included with Fred Bove's discussion on diving fitness. He was a school teacher, a diver, a diving instructor, who died last year at the age of 30. When aged 22 he had a subarachnoid haemorrhage from a congenital berry aneurism involving the anterior communicating artery. He survived this episode. The aneurism was clipped off in Melbourne. Eventually he resumed diving. I think this particular case was brought up either at a SPUMS meeting or one of Carl Edmond's courses some years ago, when we discussed whether or not this fit young man should be allowed to dive again. Here was a young man who had actually had an angiogram which had revealed an aneurism which had ruptured and been clipped. The rest of us were still diving and none of us had had an angiogram. (Remember that the most common age for ruptured berry aneurism is in the forties). It was felt that as he was symptomless and well he should be allowed to return to diving. He passed very adequately a medical examination undertaken by a SPUMS member. Unfortunately some months later, which was exactly four years from when the aneurism was clipped, he had his first grand mal seizure. Prior to this, he had been completely symptomless. After the grand mal episode he was started on Dilantin 100 mg three times daily, and was free of fits. He continued to dive for a further twelve months, then he had a further episode of grand mal. So the serum Dilantin was measured and it was found to be 22 millimoles per litre, the normal range being between 40 and 80. His Dilantin was then increased to 180 mg daily.

Over the next twelve months, until about twelve months ago, he had three or four further fits. During this time he did dive intermittently, but he did not instruct. Until about twelve months ago it seemed as if the grand mal seizures were under control. Early last year, he started to have what was thought to be petit mal. The episodes came on without warning and he would partly lose awareness of his surroundings and was often unable to retain the thread of a conversation. The attacks usually concluded within seconds but often they would come on so quickly, that he himself was aware that he looked vague.

Late last year he was seen by the neurologist in Melbourne, who had been involved with his management from the time of the sub-arachnoid haemorrhage onwards, and he was given prominal in an increasing dose in addition to the Dilantin. Unfortunately within a few weeks of this he was brought to the hospital one afternoon, unconscious. He had been by himself in his dive shop. He was thought to have been eating his lunch as he was found unconscious, with food in his mouth. It was uncertain whether he had asphyxiated through food inhalation or through some other factor. He was immediately placed on a respirator. A lumbar puncture was done and blood-stained fluid obtained. He was then transferred the same day to Canberra for further management, as we felt the outlook was rather poor. He died the following day.

At post mortem he was found to have had a further subarachnoid haemorrhage. It was impossible to determine the site of the leakage. I have spoken to numerous neurosurgeons and I understand that the anterior communicating artery aneurism has the worst prognosis of all, because it is the most difficult to reach, and technically the most difficult to get adequate occlusion of the aneurism.

Initially it was felt that for some years he was doing well, that he was fit to dive on most base lines that we would draw. But as time went by, presumably through the effects of the original ischaemic episode he developed grand mal seizures. As we all know, a seizure of any sort in the water is very likely to lead to drowning. In this case fortunately the final episode was on land and no-one else was involved. It is an unusual case that I would draw to your attention, for all of us to remember in the future.

Dr Victor Brand: Chairman

How did you administer oxygen to the first two cases?

Dr John McKee

For giving oxygen at sea, or immediately they have landed, I have an adaptor that takes a regulator that fits an oxygen cylinder. Others have been given oxygen by mask by the ambulance man while coming to hospital, and the flow rate there could be almost anything. At hospital we have usually given them something between six and ten litres a minute.

Dr David Brownbill

A couple of comments about your case of subarachnoid haemorrhage. He was not likely to re-bleed from the clipped aneurism. The most likely thing is that he developed another haemorrhage from a different site. It is well recorded and I think it is the most likely cause of death.

I would like to remind you that following surgery he had a 10% chance, right from the start, of developing epilepsy. The fact that he did not have anything for four years, one quarter of the people do not have epilepsy for four years, did not alter his chances.

There is no such thing as a well controlled epileptic. They are always liable, under the right (or perhaps wrong) set of circumstances, to develop a further fit. In someone who has bad intracranial surgery, if one admits to a 10% risk of him having a fit, diving is a matter requiring consideration by all concerned. But once he has shown that he has a predilection to fitting by having had one, diving is absolutely contraindicated.

Dr Fred Bove

I do not think that you would expect to find any changes in the X-rays of the shoulders of a person with decompression sickness. We think that somehow there is a vascular involvement of the long bones, although that is not an explanation of the pain. A bone scan would be a much more useful piece of information because it would tell you whether there was any alteration of the blood flow in the region of the pain. The X-ray obviously would not change for many months. So if you are concerned you might do a bone scan to see whether there are ischaemic areas.

Dr John McKee

The X-rays were taken 300 miles from a centre with scan equipment, but more to reassure the diver than me and partly to help decide whether to spend about $100,000 transferring him to Sydney for treatment. Very frequently one gets these divers with various symptoms, and the transfer cost, by helicopter or by Hercules, is now pretty tremendous.

Dr Fred Bove

Just one more comment. When I was at Philadelphia, we treated a case of limb bends in a commercial diver working in the Atlantic Ocean who, by the time he got to the chamber had lost all his symptoms. We still treated him in the chamber. I would suggest that where there is a chamber available you should still treat the symptom free patient with perhaps a table 5. There is a remote chance that you are preventing long term changes in the bone.

HOW COMMON IS DYSBARIC OSTEONECROSIS?

John Knight

Every diver has heard of dysbaric osteonecrosis. Very few are aware that the risk of developing dysbaric osteonecrosis is low if one follows normal air diving procedures.

Surveys of naval divers in the UK (1) and the USA (2) have shown a very low incidence of dysbaric osteonecrosis. This contrasts with earlier surveys of compressed air workers. The major difference between the two groups was that compressed air workers spent a working shift under pressure while the divers had much shorter periods under water. Many of the naval divers with bone changes had been involved in experimental diving such as testing new decompression tables and deep diving.

JAPANESE EXPERIENCES

In 1976 there were two papers from Japan published in English. Ohta and Matsunage (3) reported a three year survey (1966-1968) of the men of a village (Ohura) on the shore of the Ariake Sea off Northern Kyushu where there were about 400 divers (active or retired). The men dived to collect the expensive shell fish Atruria Japonica from November to March (winter). Except during bad weather they dived in 10m to 30m, while there was light, with a lunch break of an hour. This gave two four-hour dives each day. During the rest of the year they collected other shell fish from 30m to 60m, worked on salvage jobs from 20m to 70m or more, and did construction jobs in 10m to 30m. To quote Ohta and Matsumaga they “had used no modern technique of decompression”. This probably explains the fact that three to five men died each year from accidents or decompression sickness. Decompression sickness was very common, so common as to be considered unavoidable. Their treatment was baths and booze.

Of the 301 divers who were X-rayed, 152 (50.51) had bone lesions. Of these, 44 men (29%) had juxta-articular lesions. Between them they had 54 juxta-articular lesions. The group had a high incidence of previous decompression sickness, but there was no significant relationship between the site of the bends pain and the lesions. The incidence of bone changes was higher in those who had been diving longer and in those who had gone deeper. The results of the first year’s X-rays had been published in Japan in 1966.

The figures were quite horrifying. In the group with up to 4 years diving experience, 22% had bone changes, none of which were juxta-articular. The 5 to 9 year group had an incidence of 46%. The 10 to 14 year group had an incidence of 71%. After 15 years of diving the incidence was 74% and after 20 years it settled at 82%. The deepest depth figures were just as depressing. No lesions in those who had not exceeded 9m, but there were only 8 of them. The 10m to 19m group had a 20% incidence, luckily for them without juxta-articular lesions. Those who dived between 20m and 29m had a 46% incidence, while 30m to 39m was associated with an incidence of 53%. The 40m

TABLE 1

CLASSIFICATION OF DYSBARIC OSTEONECROSIS

JUXTA-ARTICULAR LESIONS

A1	Dense areas with intact articular cortex
A2	Spherical segmental opacities
A3	Linear opacity
A4	Structural failures
a)	Translucent subcortical band
b)	Collapse of articular cortex
c)	Sequestration of cortex
A5	Secondary degenerative arthritis (osteo-arthritis)