In reading this book think sometimes of the men who worked from open bells in cold, dark waters and their successors who depended on unreliable air pumps for their next breath, whose helmets might either fall off or fill with water if they stumbled. Our recreational (and commercial) diving is an elegant (and safe) descendant of such rude ancestry and represents the fruit of the interaction between innovative engineers (some of whom dived in their own creations) and the humble people whose bodies served as the test-beds for their bright ideas. Never forget that this process continues to the present day, though modern divers are no longer so uncomplaining nor so accepting of risks and problems. One has only to consider the constant production of "improved and safer" dive tables to understand that the process, of divers acting as the subjects for the trials of new ideas, continues. This procedure has been made much safer by the practice of seeking feed-back of information to engineers and inventors from the users (divers) and the medical person involved in the management of the results of erroneous practices. It is on this triad of interests that improvements in diving safety ultimately depend.

Naturally there is a drawback to this book. It is the cost. And the Editor might have exercised a tighter control over the enthusiasm of some of his authors for their subjects. One can even have too many pictures of those marvelous copper and brass helmets. This is a book to grace any library. It is intended as a compliment to the authors to say that the reader may be left with a desire to learn more about the men and equipment whose troubled history has brought us to our present informed position on the major problems of present day diving.

Douglas Walker

SPUMS ANNUAL SCIENTIFIC MEETING 1990

PSYCHIATRIC ASPECTS OF DECOMPRESSION SICKNESS

Chris Acott

Introduction

Over the past 10 years our understanding of and the treatment regimes for decompression sickness (DCS) have been gradually changing. Perhaps perfluorocarbons, SPEC scanning and the use of helium will be common place in the therapy of decompression sickness (DCS) in the 1990's.

There is increasing evidence that DCS primarily involves the central nervous system (CNS) and that pure Type 1 lesions are rare. Figures for involvement of the CNS range from 10 - 30% in the 1970's to 78% in a series from the school of Underwater Medicine at HMAS Penguin in 1985-86. Indeed, in another series up to 50% of Type 1 DCS were shown to have abnormal EEG's which took up to a month to normalize.

Recently, an article in the Lancet[1] showed that all Type II DCS cases had cerebral perfusion deficits shown by SPEC scanning. Therefore, it may appear that the diagnosed involvement of the CNS is dependent upon the thoroughness of the CNS examination. If one does not look for it one does not see it.

Other recent publications allude to the "punch drunk" diver, and the late neuropsychological changes occurring with diving and DCS. This has received a lot of attention in the literature. However, at present I feel that all these can be summed up by Hayward in "The Long Term Neuropsychological Effects of Compressed Air Diving". "The issue of whether DCS or various indices of dive stress are associated with decrements in neuro-psychological function still remains unresolved. It seems likely if there are effects on neuro-psychological functioning due to exposure over time to risks taken when diving they are probably not large (at least in the short term) and probably only affect a subset of individuals"

There is no data available on the acute psychiatric, or psychological manifestations of DCS, except perhaps, the mention of personality changes noted with cerebral DCS. There are numerous anecdotal reports, particularly those associated with the abalone divers, that acute psychiatric changes occur. I have talked to some of the shellers who remain in the boat while the diver is below. Their reports indicate that acute personality changes occur and these vary with the particular dive profile. The shellers could differentiate the depth of the dive on the mood and personality change seen in the abalone diver upon surfacing. These stories were so constant that one would have to assume that they are true. Acute confusional states were seen with deeper dives while aggressive, abusive behaviour was seen with the longer shallower dive.

There is often considerable delay between the onset of the symptoms of DCS and the actual time the diver presents himself for treatment. This delay is frequently attributed to:

1 Ignorance of the meaning of the symptoms both in the diver and the dive leader.
2 Over indulgence in alcohol.
3 The symptomatology being blamed on a previous injury.
4 Denial. The dive was well within the limits of the tables so the diver could not possibly be bent.
5 Guilt. DCS is regarded in some circles as shameful as an STD.

Failure to recognize that there is something wrong may, in fact, be a manifestation of the disease. Unrealistic or perhaps, in some instances, a paranoid reaction to the symptoms may in part be part of the disease itself. This is sort of a "Catch 22" situation. To recognize that one has DCS one must recognize the symptoms, but one of the symptoms of DCS is that one does not recognize that one has got it.

This brings me to my topic, which, could be called The Post DCS Blues and DCS Psychosis.

Since joining the Hyperbaric Unit at the Royal Adelaide Hospital (RAH) I have made the clinical observation that most post treatment patients go through a depressive phase, and that divers diagnosed as having cerebral DCS have a degree of psychosis, manifesting itself mainly by paranoid behaviour. The following case histories will demonstrate this.

Case Histories

Case 1

This 32 year old diving instructor had been working in the Maldives. He presented at our department on the 27th of September 1989. He had been flown down from Darwin Hospital by the Royal Flying Doctor Service in a pressurized aircraft.

He was agitated, irritable, slow and vague. He had poor short term memory. He was extremely unstable on his feet with a sharpened Romberg of 2 seconds. When doing serial 7's he made 4 mistakes in 120 seconds, while serial 5's took 60 seconds. He had worked in a statistics department before going to the Maldives).

CT scan, EEG and chest X-ray were all normal, but auditory evoked potentials were abnormal.

His history prior to admission was extremely interesting and unfortunate. But it does illustrate some important factors.

He began working in the Maldives in April 1989. He usually dived twice a day, 6 days a week. The first dive in the morning was to 20 or 30 m for 50 to 60 minutes. The second dive was about 3 hours later and could be to 20 m. He used a Beuchat dive computer.

Occasionally he had the afternoon off. He drank heavily. He played hard. The staff at the resort noticed a slow personality change. He became aggressive and abusive, not only towards the staff but at times to the guests. He was told to stop drinking which he did, at least one month prior to his eviction from the island. His last recorded dive was on the morning of the 14th of September. He told me later that he does not know how he finished the dive. On ascent he became extremely confused. He remembered falling over in the boat. He was put on the first available flight back to Australia which was that day. He described the flight as being terrible. His confusion worsened. He disembarked at Singapore and stayed there for a week. His Darwin girlfriend reported receiving several distressing phone calls. She said his conversation was bizarre, vague and extremely slow. He finally arrived in Australia on the 22nd of September. He was unable to fill in his customs declaration form. He was strip searched by Customs as they suspected drugs. His girlfriend took him to Darwin Hospital. There he was admitted to the psychiatric ward. They were unable to diagnose the problem as his behaviour was unlike anything they had seen. Then finally somebody found out that he had been a diving instructor. They contacted us and he was flown to Adelaide that day.

It is of an interest that his employer sent all his possessions back to Australia except his dive log.

He received 10 hyperbaric treatments. He improved after each treatment, but even the optimists could never say that he was quite right. His auditory evoked potentials returned to normal.

He was reviewed on the 10th of October 1989. It was at this time that he had an acute paranoid psychotic reaction. He was hospitalized in a major psychiatric hospital. He underwent a series of investigations and psychometric assessments. Finally after rejecting our initial diagnosis the psychiatrists concluded that he had a "neuropsychological profile that one would expect from a person suffering from hypoxia, probable cause DCS". His presentation and what was subsequently found on psychometric assessment was not described in the text books.

Follow up revealed a pleasant fellow, off all medications and able to return to work. He still had a moderate degree of memory deficit. Assessment had also revealed frontal lobe impairment. However it is expected that he will make some spontaneous improvement.

Case 2

This 31 year old male.first dived 2 years ago, one of the dives was to 20 metres and involved a "slow" rapid

ascent. When he surfaced his symptoms were consistent with having suffered from a cerebral arterial gas embolism (CAGE) or cerebral DCS. He was told never to dive again. He waited for 2 years, then did four dives over 2 days. None were deeper than 15 metres. The bottom times were conservative and there were no instances of a rapid ascent. He surfaced from his last dive (to 10 m) with similar symptoms to his episode of 2 years earlier. He went home having decided to sweat it out. He slept that night with his bed on blocks to keep his head down. In the morning he was no better, in fact his symptoms were a little worse. Finally he contacted us. The diagnosis was made of cerebral DCS, with him probably having a right.to left shunt. (His daughter was diagnosed 2 days after his admission as having an ASD).

On admission he was extremely aggressive and abusive with paranoid overtones. He made remarks like "What are the police divers doing here? Are they after me?". He later said that he could not stop these odd feelings. He had a frontal headache, complained of paraesthesia and was very unsteady. There was short term memory loss.

His treatment was successful, to a degree!

Follow up revealed a different person to the one who had been admitted. He was a gentle caring father. He admitted to having had paranoid feelings, and also to having been extremely depressed. He said these feelings took about a month to disappear. He is at present undergoing psychometric assessment. He still complains of short term memory loss and of being "slower" than he was before this episode.

He will not be diving again.

Case 3

This Swedish tourist was treated at Townsville. She was reviewed at the RAH. She had been diagnosed as having had cerebral DCS. Although successfully treated clinically, she admitted to not feeling her normal self.

She was still slightly depressed, and said that this had gradually got better over the past couple of months. The interview also revealed that she had had extreme paranoid feelings during treatment and just after. At that time she did not like being alone because space creatures were going to get her. These creatures had been in a book that she had been reading when she went diving.

Discussion

Organic causes for depression and psychosis are numerous, although depression may just be a simple reaction to an environmental circumstance. Depression has some hypochondriacal associations. Some of the symptoms commonly seen after therapy, which disappear during hyperbaric treatment, may just be a manifestation of this depressive phase. Common symptoms frequently seen are poorly localized joint pain, insomnia, fleeting nondescript pains, fatigue and generally feeling unwell. Perhaps we should be using antidepressive medication in some of these patients, especially if the symptoms are different before and after treatment.

Encephalopathies following organic illnesses are numerous, e.g. delayed hypoxic encephalopathy is characterized by apathy, confusion, irritability, agitation and mania. Endocrine encephalopathies are characterized by confusion, agitation, hallucinations, delusions, anxiety and depression. But perhaps the one clinically closest to what I have described here is puerperal psychosis. This is a brief psychological disturbance seen in the puerperium. Typically it is one of depression, however, there are other degrees of confusion and thought disorders. Recovery takes time.

The study by Adkisson et al[1] showed that the cerebral perfusion deficits in divers suffering from Type II DCS were mainly in the frontal and parietal lobes. The clinical significance of this was unclear. Perhaps it may manifest itself as depression and/or other psychiatric or psychological problems. Psychometric assessment of one of these cases showed frontal lobe involvement. Adkisson et al. described that their divers had disorders of mentation, but what these disorders were was not described.

Conclusions

I have presented reflections based on clinical observations. To be bent and not recognize it may in fact be an important diagnostic tool for cerebral DCS.

The symptomatology of DCS, which is obvious to us may not be of importance to the diver because the various pathological processes invovled alter perception. Reluctance to seek treatment may actually be part of the disease process due to specific CNS deficits. Indeed aggression, abnormal and paranoid behaviour indicate significant CNS involvement.

A good clinical guide to a diver's well-being after DCS would be the diver's immediate family's assessment of when the diver is back to "normal" again. Unfortunately this may never occur.

Reference

1 Adkisson GH, MacLeod, Hodgson M et al. Cerebral perfusion deficits in dysbaric illness. *Lancet* 1989; ii: 119-122

This is an editorially revised transcript of a lecture given at the 1990 Annual Scientific Meeting. Full references

are available from Dr Acott.

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A SERIES OF DIVING ACCIDENTS FROM NORTHLAND, NEW ZEALAND, 1984 - 1989

Peter Chapman-Smith

Introduction

This group of 80 cases was collected in a region of northern New Zealand, where snorkel and scuba diving is a prevalent leisure and food gathering activity. Most cases presented in general practice, by referral from dive shops or from the local Base Hospital in Whangarei.

An overview is provided, followed by consideration of several specific cases of inner ear barotrauma and then some unusual cases.

Clinical Material

80 cases presented over this 5 year period.

BAROTRAUMA

Ear	
External	1
Middle	17
Ruptured tympanic membrane	8
Alternobaric vertigo	2
Round window rupture	11
Inner	3
Sinuses	

Although it is my impression that this is the most common problem only 1 case was recorded.

PULMONARY	
Arterial gas embolism	
Cerebral (CAGE)	9
Mesenteric AGE	1
Pneumothorax	1
Other	2

DECOMPRESSION SICKNESS (DCS)

Of the 8 cases none had neurological DCS.

Skin	1
Elbow	2
Knee	1
Shoulder	3
Gastro-intestinal	1

TRAUMA

This group was quite varied. The most dramatic was the case of traumatic pneumothorax. The others were:

Neuropraxia of ulnar nerve	1
Head injury	1
Foreign bodies in the skin	2
Subconjunctival haemorrhage	1

MISCELLANEOUS

Shallow water blackout	2
Dermatitis due to jellyfish contact	4
Near drowning	2
Retinal haemorrhage	1
Hypoglycaemia in an insulin dependant diabetic.	1

Inner Ear Barotrauma

The cases of inner ear barotrauma were of interest. To differentiate between the round and oval window ruptures, pure cochlear or vestibular damage, or inner ear membrane rupture can be difficult. And there is always the possibility of inner ear haemorrhage. Even at tympanotomy a demonstrable fistula is often not apparent.

PRESENTATION

Of the 14 cases, 2 were snorkelling, the remainder were scuba divers. There were equal numbers of left and right round window ruptures.

Seven divers had considerable dive experience Nine had had difficulty during the dive performing an adequate Valsalva manoeuvre. Nine developed symptoms while in the water Symptoms were usually of sudden onset. Eleven divers complained of deafness. Giddiness or vertigo was noticed by five. Ten had tinnitus and it was often still present when they were seen. Four had nausea which was often marked, while four vomited. Other symptoms reported were epistaxis, full or blocked or watery sensation in the ear, hyperacusis, and pain. Hyperacusis, especially acute hearing, was an unexpected finding. Edmonds[1] mentions that some patients with a patulous Eustachian tube, following barotrauma, may have excessive awareneess of their respiration or of their own speech (causing them to speak softly) or reverberations of sounds such as footsteps. My patients did not complain of these problems.

Three divers had been using treatment for allergic rhinitis. The divers were frequently late in presenting, appearing days or weeks after the day of the dive accident. They had often seen an after hours GP, casualty officer or